



Information for teachers

Teachers often feel that they lack the knowledge or experience to educate students about cannabis and the range of issues surrounding its use. This document provides up-to-date and evidence-based background information to help teachers improve their own knowledge and understanding of cannabis, its use and potential harms.

Terminology: Cannabis or marijuana?

Cannabis is known by many names depending on where an individual lives and their culture group. Although there are many street terms used to describe the drug (such as grass, pot, weed, and dope) cannabis and marijuana are the most likely to be used in drug prevention. The term 'cannabis' is used internationally and is correct from a botanical point of view as the flowering plant is named *Cannabis sativa*. The term 'marijuana' is used mainly by the US as a result of its use in the country's documents of law, including the *Marihuana Tax Act of 1937*. The term 'marijuana' is often used to describe the dried leaves and flowers from the cannabis plant that are generally smoked. Although these terms can be used interchangeably, in this resource the term 'cannabis' is used.

For more detail on this topic, [click here](#).

Cannabis and the law

In Australia, it is illegal to possess, use, grow or sell cannabis under federal and state law. The penalties for breaching these laws differ between the states and territories. Some jurisdictions have 'decriminalised' cannabis meaning that, if the cannabis in question is deemed to be 'small' (1-2 plants, or up to 25-50 grams of plant material) and for personal use, the penalty for a cannabis offence can be reduced to a fine without a criminal charge (i.e., a 'civil penalty', typically \$50-\$200). South Australia, Western Australia, the Australian Capital Territory and the Northern Territory have decriminalised cannabis use. In the remaining states where cannabis is not decriminalised, a cannabis offence attracts a criminal charge, although minor offences are often diverted from the criminal justice system. For example, offenders caught with less than 50 grams of plant material in Queensland can agree to attend a drug assessment or brief intervention.

For up to date information regarding cannabis and the law and the different penalties that exist across the country, [click here](#).

Types of drug use

An individual's reasons for use and the context in which they use can assist in identifying any associated health risks. Fluctuation between these categories of use is not necessarily linear and will vary for each individual. Most young people will not experience cannabis dependence as their use is most often sporadic, occurs within a social context and is frequently determined by availability (1; 2). As is discussed below, each individual's experience from smoking cannabis can be partially explained by the mediating effects of the drug itself (in terms of dose, purity and mode of administration), the user (in terms of mood, attitudes, tolerance, susceptibility, prior experience and psychiatric symptoms), and the environment it is used in (in terms of social setting) (3; 4).

Table 1. Categories of drug use and their implications for young people

Type of use	Description	Issues to consider with cannabis
Experimental	A person tries a drug once or twice for the first time out of curiosity For example, having a puff of a joint at a party	The person is unaware of the response they will have and the dose they may respond to, thus there is an increase in the possibility of an adverse response. Use in this context might be unplanned and therefore take place in an environment that could be risky (e.g., a public park)
Recreational	A person is motivated to and chooses to use a drug. This type of use is typically less than weekly For example, using to enhance the enjoyment of watching a DVD with a group of friends	The person is likely to have had some experience with the drug but may be unaware of the potency, environmental and consequential effects
Situational	A person uses cannabis as a coping mechanism for certain situations For example, smoking cannabis to settle nerves before an exam or smoking at a party to ease social anxiety	The use may become directly associated with a specific experience depriving the person of finding other ways to manage the situation. Links have been made between young people who use cannabis to avoid/manage negative emotions (stress or distress) and higher likelihood of developing ongoing or problematic use
Intensive	Intensive or heavy use is typically defined as daily or near daily use. This kind of use can have detrimental effects on many aspects of personal functioning For example, smoking cannabis daily during school holidays	Accumulative cannabis toxicity increases the likelihood of an adverse response. Neuro-adaptation may occur leading to the risk of withdrawal symptoms upon ceasing use. The risk of many physical and/or mental impairments has been shown to increase with increasing dose
Dependent	A person becomes dependent on the substance after prolonged or heavy use over time. They feel a need to take the drug consistently to feel 'normal' and avoid unpleasant withdrawal symptoms. They may develop a need to consume more of the drug to attain the same effect or feel less benefit from their usual dose (tolerance) For example, prolonged daily use	The use of cannabis interferes with their day-to-day functioning and takes priority over other activities. The person may be constantly preoccupied with thoughts about smoking. Young people who use a lot of cannabis can experience a lack of motivation. Ceasing use is likely to result in uncomfortable withdrawal symptoms; typically experienced as trouble sleeping and increased irritability

Cannabis – the drug

Cannabis, referring to the plant *Cannabis sativa*, is a product of almost 500 compounds, including over 70 compounds that are unique to the plant, known as cannabinoids. The main psychoactive constituent of cannabis is the cannabinoid delta-9-tetrahydrocannabinol (THC). The effect of THC does not work in isolation and may be enhanced or reduced through interactions with other cannabinoids, particularly the cannabinoid 'cannabidiol' (CBD) (5; 6).

The flowering tops or 'buds' of the female cannabis plant contain higher levels of THC than the leaves and are the most commonly used part of the plant. These buds are typically smoked using a hand-rolled cigarette called a 'joint' (approximately 84% of cannabis users), and/or by using a water pipe (whereby the smoke passes through water before being inhaled) called a 'cone' or 'bong' (approximately 82% of cannabis users) (7). Dried cannabis resin, although infrequently used, also has a high concentration of THC and is commonly referred to as 'hash' or 'hashish'. Oil-based extracts from hashish, referred to as hash oil, are also infrequently used but are likely to have higher still THC content or 'potency'. Cannabis can also be added to food for consumption. Using cannabis in this way extends the time taken to achieve a 'high', meaning that controlling the dose effect can be difficult. In occasional users, a dose of 2-3mg is likely to deliver a 'high' when the cannabis is consumed (8).

Cannabis is often mixed or 'mulled up' with tobacco and as such the potency and associated effects from smoking can vary greatly. Importantly, smoking this mix is associated with a significant increase in the risk of cannabis use



disorder and psychosocial problems (9). Moreover, simultaneous cannabis and alcohol use is associated with an increased risk of substance-related legal, academic, and relationship problems among adolescents (10).

For more information regarding cannabis and its effects, [click here](#).

Potency

Cannabis potency is of particular interest as cannabis plants with different cannabinoid content are likely to have different adverse health effects (11; 12). Potency is highly variable and is mostly based on the type of plant and where it is grown, as well as the season, quality and freshness of the plant (13). International trends show that plant cultivation techniques tend to favour plants with high THC and low CBD (14). Notably, data from the US shows that the concentration of THC in the US has increased from an average of 3.4 per cent THC in 1993 to 8.8 per cent in 2008 (15). THC concentration has remained relatively stable during this period in European samples (12; 16). Unfortunately, cannabis potency has not been systematically tested in Australia. In a recent analysis of cannabis seized by NSW police, however high levels of THC (14.7%) and low levels of CBD (0.1%) were found, which did not appear to be affected by cultivation technique (indoor, often referred to as ‘hydro’ or ‘hydroponics’, or outdoor cultivation, ‘bush buds’) (13).

In conjunction with the complex chemical nature of cannabis, the variability of cannabis potency that is found across studies is compounded by the methods and sample sizes used in the analysis. For example, most studies have examined the potency of cannabis that has been seized by police (which may not be representative of all cannabis) and some studies do not identify which part of the plant has been analysed (5). In addition, studies which state an increase in potency may actually reflect differences in the quality of the plants which result from reductions to testing delays over time (17).

For further information on cannabis potency, [click here](#).

Effects/Intoxication

The effects from smoking cannabis (referred to as the ‘high’) typically begin to occur within a few minutes, and peak within approximately one to two hours (18; 19). In a systematic review of 30 studies regarding the effects of cannabis use, great variation in the reported effects was noted, although the most commonly reported subjective effect from smoking was a feeling of relaxation (20). Other common effects include changes in appetite, ability to concentrate, talkativeness and a feeling of happiness invoking laughter (20).

Overdose

Unfortunately, there is not enough reliable data to determine whether smoking cannabis poses a direct threat to mortality (21). Fortunately, a fatal dose of cannabis is beyond that which any user could realistically smoke or eat (the estimated acute lethal blood concentration is at 180-315 µg/L) (22). While an overdose is not possible, ‘greening out’ is a term that refers to the situation where people feel sick after smoking cannabis. They can go pale and sweaty, feel dizzy, nauseous and may even start vomiting.

Brain

Persistent use of cannabis during adolescence is a significant concern. A comprehensive prospective study of 1,037 individuals followed from birth to 38 years showed that cannabis use, when initiated in adolescence and continued into adulthood, was associated with significant and broad neuropsychological decline when controlling for demographic variables including education (23). Notably, initiation to cannabis use prior to the age of 20 years showed greater impairment to neuropsychological functioning than initiation after the age of 20 years. This significant detriment to health that is associated with initiating cannabis use in the teenage years has been consistently supported in the literature (24-26).

Withdrawal

A number of withdrawal symptoms have been observed in adults and adolescents seeking treatment for cannabis use (27; 28). The symptoms most commonly reported and intensely experienced include problems with sleep, increased irritability, and increased aggression, particularly among those with a previous history of aggression (29-32). Its time course appears to begin from about 24 hours, with symptoms peaking between one and two weeks from last use, and declining to baseline levels over three to four weeks (28; 29; 33; 34). Notably among young people,



withdrawal symptoms such as tiredness and trouble concentrating have been described to result in an inability to complete school work (28).

For more information regarding cannabis withdrawal, [click here](#).

The individual factors associated with initiating cannabis use

A large body of research has described many individual risk factors of initiation to cannabis use. These include psychological factors such as poor emotional regulation, low self-esteem and behavioural self-control, and physiological factors such as an intense neurological response that increases craving (35). The more consistent individual risk factors of initiation to cannabis use are described below:

Mental health

There is a well described comorbidity between mental health disorders and substance use disorders (36). In particular, depressive symptoms, behavioural problems and antisocial behaviours have been found to be significantly associated with both cigarette and cannabis use uptake (37; 38). Mental health disorders and other substance use disorders have been described to increase the risk of cannabis use onset by a mean of 1.2 to 3.3 times (39). Notably, females with poor mental health are at a greater risk of frequent cannabis use compared to males with poor mental health (2). Finally, a common motivation reported for cannabis use is to help alleviate stress (40). This is a dangerous combination as this pattern of use is associated with more entrenched use and poorer health outcomes (41; 42).

For more information regarding cannabis and mental health, [click here](#).

Motivations and expectations

An early study on cannabis motives found that among 634 recent high school graduates, over half of cannabis users would use cannabis for enjoyment or fun (52.1%) (43). That is, most users wish to achieve a so-called 'high' from smoking, typically described as entailing mild euphoria, relaxation and perceptual alterations (8; 20). Other common motives included social cohesion (42.8%) and experimentation (41.3%) (43). Using cannabis in a social context is often done to increase sociability and 'talkativeness' (44) and may be associated with lower levels of intoxication (45; 46). Using cannabis to help cope with certain situations (18.1%) or out of addiction or habit (1%) is less common (43). Using cannabis out of habit, or to help cope, is a strong predictor of frequent cannabis use. This finding is consistent with further research of adolescent cannabis use where coping motives predict greater frequency of use and more negative cannabis-related problems compared to social motives (41; 42). Finally, the extent to which we perceive the effects of cannabis would harm our health is likely to affect the risk of cannabis use onset. That is, low expectations of negative effects from cannabis use have been consistently shown to be associated with subsequent frequent use while expectation of negative effects is a predictor of non-use (47-49).

Impulsivity

Individuals with an impulsive nature are thought to be at greater risk of substance use uptake than those without (50-52). This is of particular relevance to young people who are in a stage of neurocognitive development which is associated with heightened levels of impulsivity (51). For example, neuroimaging studies have shown that compared to adult brains, adolescent brains show a heightened response to novel stimuli, suggesting a greater vulnerability to initiating substance use (26). Interestingly, impulsive decision-making is also more likely to occur during cannabis intoxication. This finding has been supported by a number of studies including self-report (53), neuroimaging (54; 55) as well as observation using measures of behavioural impulsivity (56-61). As such, smoking cannabis can progress to further frequent use via increases to impulsivity.

Genetics

Our individual genetics have a significant impact on our vulnerability to initiate cannabis use and to continue to problematic cannabis use (62). The influence of genetics in this respect appears to be stronger than the influence of environmental factors, although the individual's gender is an important factor (62). That is, in a review of 28 twin studies on cannabis use initiation and 24 studies on problematic cannabis use among males, genetic factors appear to predict use progression to a greater extent than environmental factors. Alternatively, environmental factors appear to be a better predictor of use onset (26, 63). As such, the influence of genetics is of obvious importance but it cannot be conceived in complete ignorance of the individual's environment (50; 63).



Environment and cannabis

Four large, separate longitudinal cohort studies in three different countries (totalling over 40,000 participants) have found that environmental factors can increase the likelihood of initiating cannabis use by a mean of between 2.5 and 3.9 times over a period of two to four years (39; 64-66). Across these four studies, the most important environmental influences included familial influences, social environment influences as well as the price and availability of cannabis. Each of these factors and the additional environmental influence of the Australian media are described in detail here.

Familial environmental influences

Familial and environmental influences are complex and include such factors as behavioural modelling, parental attitudes and parental monitoring (26; 67). In particular, parenting practices can shape a child's decision to maintain abstinence from drug use or to progress to substance use (26; 35; 38). Firm and consistent limit-setting, careful monitoring, nurturing and open communication patterns have been shown to act as protective factors for adolescent substance use (35). In contrast, permissive parental attitudes, parental substance use and low parental monitoring are predictors of substance use onset (26; 38). Similarly, family dysfunction, including parental divorce and abuse, is a predictor of substance use onset (which is commonly used to help cope with such stressors) (39; 40; 64).

Social environmental influences

These include such factors as peer influences and participation in social activities. A peer's disapproval of substance use has been consistently shown to reduce the risk of substance use onset although this relationship appears to be stronger among males compared to females (37; 38; 62; 65; 68; 69). There is some contention however, as to whether this peer influence is an artefact of choosing 'like-minded' peers with similar attitudes toward substance use, or whether the influence is a result of the intention to model behaviour from peers' own behaviours. A combination of the two theories is likely (26). In addition, young people who connect with non-users and engage in community activities, particularly religious attendance, have also been described to be less likely to engage in substance use (35; 39; 66).

Price and availability

The price of cannabis is also an important environmental factor which is significantly associated with the risk of onset to cannabis use (70; 71). Using data from the 1998 Australian National Drug Strategy Household Survey and the Australian Illicit Drug Report (supplied by police), one study has linked lower cannabis prices with an increased likelihood of early initiation into cannabis use in the teenage years (70). As would be expected, the more available that cannabis is to potential users the greater the risk of initiation into cannabis use and of continuing use (69; 72).

Media portrayal of cannabis

The media portrayal of licit and illicit substances has been found to influence adolescent attitudes, expectancies and perceived positive consequences of use (35; 73-75) and is one of the dominant forms by which substance-related information is communicated in Australia (76; 77). Among the illicit drugs, cannabis shares the greatest coverage in Australian news articles, and articles on the topic tend to focus on the legal problems associated with use (78). Despite this, the impact of media on public opinion regarding cannabis use has only recently been explored and has focussed on the media portrayal of synthetic cannabis (79) and rates of cannabis seizures (80). Importantly, these articles suggest that cannabis-related media in Australia is likely to be indirectly shaping an escalated sense of risk (81). That is, the sense of risk is likely to be escalated by exaggerating the prevalence of synthetic cannabis use and the size and frequency of cannabis seizures.

Negative health impacts of cannabis use

Both short-term (acute use) and long-term (chronic) cannabis use are associated with significant and negative health impacts (82). Acute cannabis use is most likely to impact on the risk of heart attack, as well as lead to increased risk of injury by motor vehicle accident. Chronic cannabis use however, is of greater concern due to its more significant implications to user health. Each of the more commonly cited negative health impacts of both acute and chronic cannabis use are described here.

Acute cannabis use

Risk of heart attack

Acute cannabis smoking is known to produce increases in heart rate which correspond to the dose consumed (83; 84). This increase in heart rate is likely to be experienced as mild stress in young people which decreases as tolerance develops (84-86). For those who are particularly vulnerable to heart complications, such as older adults with ischaemic heart disease or hypertension, the increase to heart rate may exacerbate symptoms as well as cause a greater risk of heart-related complications (84; 86-89). In one notable study of over 3,000 patients who had experienced a heart attack, of those who had smoked cannabis (3.2% of the sample) the risk of heart attack was calculated to have increased by 4.8 times within the first hour of smoking (90).

Accidental injury

Cannabis use has been consistently shown to affect cognitive performance and a close relationship is likely (19; 91). Cognitive and subjective dose-response effects of acute oral delta-9-tetrahydrocannabinol (THC). In regards to young people, those who use cannabis prior to 15 years frequently show reduced cognitive capabilities compared to non-users. The most commonly reported impairments to cognition include memory problems (92), failure to maintain attention (93), and a reduction to overall IQ (94). Whether or not such impairments to cognition may result in an increased risk of accidental injury is not as clear. The most researched form of injury thought to be associated with cannabis use is that which results from motor vehicle accidents. This is an area of continued debate and has spurred multiple systematic and meta-analytic reviews (95). In the most recent review of observational studies, case-control studies and culpability studies, the pooled relative risk of being involved in a motor vehicle accident was calculated to be between 1.23 and 6.33 times higher when driving under the influence of cannabis than when driving unimpaired (1.92 times higher on average among observational studies, 2.79 times among case-control studies and 2.10 times among culpability studies) (95). Overall, the literature suggests a near doubling of the risk of motor vehicle accident occurring when driving under the influence of cannabis.

Chronic cannabis use

The respiratory system

As with tobacco smoke, cannabis smoke contains harmful chemicals which can damage the lungs, and the respiratory system (96-98). For some people this damage is increased due to a prolonged and deeper inhalation when smoking cannabis compared with tobacco. This style of smoking can lead to a three-fold increase in the amount of tar and nearly five-fold increase in the carbon monoxide that is taken into the lung (99). The literature on the respiratory health effects of chronic cannabis smoking suggests a causal link with symptoms of bronchitis, such as coughing and sputum production (100-106), and damage to mucosa which may result in an impaired immune response to ingested toxins (107-112). As stated in the most recent literature review on these effects, the extent to which these symptoms result in a reduction to the size of the airway or the development of emphysema remains unclear without further research controlling for tobacco use and other confounding issues such as environmental exposure to other toxins (113). In addition, the possibility that cannabis smoking may lead to lung cancer, as has been shown with tobacco smoking, is a subject of some contention (114; 115). In the most recent longitudinal cohort study which controlled for other substance use, smoking cannabis more than fifty times was calculated to approximately double the risk of developing lung cancer over a period of 40 years among a sample of Swedish conscripts (114). Notably, less frequent use showed no significant association.

For more information about cannabis and tobacco use, [click here](#).

Reproduction and fertility

Although cannabis is the most commonly used illicit drug amongst women of reproductive age, or who are pregnant, research on the effects of smoking during pregnancy that controls for other substance use is scarce. While it is strongly recommended that pregnant women abstain from using illicit drugs, including cannabis, relatively little is known about the impact on children born to mothers who do use cannabis. In the most recent review of studies on prenatal cannabis exposure, foetal development (in terms of birth size and early growth) was found to be consistently affected, while negative impacts on infant behaviour and cognition were not as clear (116). Moreover, the majority of effects on behaviour or cognition which were significant at birth, and shortly after, were seen to disappear over



the first few years of the newborn's life. Unfortunately, only three longitudinal cohort studies have examined human prenatal cannabis exposure to date and more replication studies are needed (116). In contrast, a large body of research has consistently shown that male fertility is negatively affected by chronic cannabis use, typically via hormonal disruptions as well as by reduced sperm motility (117).

For information regarding cannabis use and pregnancy, [click here](#).

Mental health

Chronic cannabis use compared to infrequent use and non-use is thought to increase the risk of developing psychotic symptoms (118; 119). This dose-related risk has been quantified by two meta-analysis which state that cannabis users are likely to develop psychotic symptoms approximately two to three years prior to non-users (119) and are at approximately twice the risk of receiving a diagnosis (118). In addition, cannabis users are between 1.5 and 4.3 times more likely to develop schizophrenia compared to non-users (120-132). This increase in risk of psychosis is particularly important among cannabis users with a first degree relative who has symptoms of psychosis, as for these individuals the risk doubles from around 10 per cent to 20 per cent (133). Notably, the prevalence of cannabis users with schizophrenia is low given the already low prevalence of schizophrenia in the population (134).

Anxiety is one of the most commonly reported negative effects of cannabis use and is also a commonly cited withdrawal symptom when abstaining from use (135-137). In fact, the experience of anxiety as a withdrawal symptom is so intense for some individuals that it causes relapse to cannabis use (138). The association between cannabis use and anxiety is further explained by a 2009 review of the literature (139). First, inexperienced users are particularly at risk of symptoms of anxiety resembling panic attacks. Second, anxiety is frequently cited by regular cannabis users (8% to 33% of occasional non-dependent users, and 36 per cent to 83 per cent of cannabis dependent users). Third, there is a high prevalence of co-morbid panic and/or social anxiety disorder diagnoses among those with cannabis dependence (between 13% to 31% of dependent users, compared to approximately 5% of non-users) (139) and cannabis use is most likely to predate the onset of these diagnoses (140).

For more information regarding cannabis and mental health, [click here](#).

Cannabis use disorder

Approximately one in fifty people in the US and Australia report a cannabis use disorder (cannabis dependence or cannabis abuse) in national surveys (141; 142). When looking at the prevalence of cannabis use in the population, Hall and Pacula estimate that one in ten cannabis users are at risk of cannabis dependence (3). Cannabis use disorders are typically first experienced in adolescence, within 10 years of initiation, and are more commonly cited by males compared to females (65; 143-148). Other factors thought to increase the likelihood of developing a cannabis use disorder include having greater access to cannabis, a lower socio-economic status, a co-morbid substance use disorder, living in an urban centre, experiencing early parental death, and using cannabis more frequently and in greater quantity (149-151).

Cannabis use disorders are characterised by habitual use despite social, psychological, and physical impairments, relationship and family problems, guilt associated with use of the drug, and low life satisfaction (152; 153). In addition, those with a cannabis use disorder are also likely to experience withdrawal when abstaining from cannabis use. In a recent investigation of the prevalence and intensity of withdrawal symptoms, the most commonly reported and distressing symptoms included having trouble sleeping, increased aggression, imagining being stoned and having no appetite (33).

For more information on cannabis and dependence, [click here](#).

Cognition

It is clear from studies matching chronic cannabis users with non-users that cannabis use is associated with impairments to verbal learning, memory, and attention span (92; 93; 154; 155). In fact, these impairments can also be seen in the brain using functional imaging where areas responsible for memory and attention show reduced activity in cannabis users (156-158). In addition, a large New Zealand longitudinal study found that controlling for factors that may affect IQ; persistent cannabis use during adolescence explained a reduction of 8 IQ points compared to non-users (23). This reduction was not seen when cannabis onset began during young adulthood. Importantly, there is some evidence that these deficits may persist for several years following complete abstinence from use (91; 154; 159).

Compounding the negative health impacts of cannabis use

The negative health impacts associated with cannabis use are compounded through two main patterns of use – that is, early initiation and frequent and long-term use. When cannabis is smoked at an early age, the association with many negative health impacts is strengthened, including poorer long-term health outcomes (160-162), worse academic outcomes (163), social and legal problems (160), and a greater likelihood of developing dependence (70; 146; 160; 164). This is particularly concerning as national surveys from the US, the European Union and Australia suggest that the age of initiation to cannabis use has been declining over time (70). Further, the majority of health-related harms from cannabis use share a dose relationship with the amount and frequency to which cannabis is smoked (82; 165; 166). For example, longitudinal study suggests that those who use cannabis less than 100 times in their life are not likely to experience significantly worse health outcomes compared to non-users (165).

References

1. **Swift, W., Coffey, C., Carlin, J. B., Degenhardt, L., & Patton, G. C.** (2008). Adolescent cannabis users at 24 years: trajectories to regular weekly use and dependence in young adulthood. *Addiction* 103, 1361-1370.
2. **Tu, A. W., Ratner, P. A., & Johnson, J. L.** (2008). Gender differences in the correlates of adolescents' cannabis use. *Substance Use and Misuse* 43, 1438-1463.
3. **Hall, W. & Pacula, R. L.** (2003). *Cannabis use and dependence: Public health and public policy*. Cambridge: Cambridge University Press, 38-46.
4. **O'Brien, C. P.** (1996). *Drug addiction and drug abuse* (pp. 557-577). New York: McGraw-Hill, 557-577.
5. **McLaren, J., Swift, W., Dillon, P., & Allsop, S.** (2008). Cannabis potency and contamination: a review of the literature. *Addiction* 103, 1100-1109.
6. **Morgan, C. J. A., Schafer, G., Freeman, T. P., & Curran, H. V.** (2010). Impact of cannabidiol on the acute memory and psychotomimetic effects of smoked cannabis: naturalistic study. *British Journal of Psychiatry* 197, 285-290.
7. **Australian Institute of Health and Welfare (AIHW).** (2008). *2007 National Drug Strategy Household Survey: detailed findings*. Canberra: Australian Government Department of Health and Ageing.
8. **Hall, W. & Degenhardt, L.** (2009). Adverse health effects of non-medical cannabis use. *The Lancet* 374, 1383-1391.
9. **Peters, E. N., Budney, A. J., & Carroll, K. M.** (2012). Clinical correlates of co-occurring cannabis and tobacco use: a systematic review. *Addiction* 107, 1404-1417.
10. **Brière, F. N., Fallu, J. S., Descheneaux, A., & Janosz, M.** (2011). Predictors and consequences of simultaneous alcohol and cannabis use in adolescents. *Addictive Behaviors* 36, 785-788.
11. **Morgan, C. J. A., Freeman, T. P., Schafer, G. L., & Curran, H. V.** (2010). Cannabidiol attenuates the appetitive effects of [Delta]9-tetrahydrocannabinol in humans smoking their chosen cannabis. *Neuropsychopharmacology* 35, 1879-1885.
12. **United Nations Office on Drugs and Crime.** (2013). *World drug report 2012*. United Nations Publication.
13. **Swift, W., Wong, A., Kong, M., Arnold, J., & McGregor, I.** (2013). Analysis of cannabis seizures in NSW, Australia: Cannabis potency and cannabinoid profile. *PLoS ONE* 8, e70052.
14. **Arnold, J. C., Boucher, A. A., & Karl, T.** (2012). The yin and yang of cannabis-induced psychosis: the actions of Delta(9)-tetrahydrocannabinol and cannabidiol in rodent models of schizophrenia. *Current pharmaceutical design* 18, 5113-5130.
15. **Mehmedic, Z., Chandra, S., Slade, D., Denham, H., Foster, S., Patel, A. S., Ross, S. A., Khan, I. A., & ElSohly, M. A.** (2010). Potency trends of Delta9-THC and other cannabinoids in confiscated cannabis preparations from 1993 to 2008. *Journal of Forensic Sciences* 55, 1209-1217.
16. **Hardwick, S. & King, L.** (2008). *Home Office Cannabis Potency Study 2008*. St Albans: Home Office.
17. **Sevigny, E. L.** (2013). Is today's marijuana more potent simply because it's fresher? *Drug Testing and Analysis* 5, 62-67.

18. **D'Souza, D. C., Ranganathan, M., Braley, G., Gueorguieva, R., Zimolo, Z., Cooper, T., Perry, E., & Krystal, J.** (2008). Blunted psychotomimetic and amnesic effects of delta-9-tetrahydrocannabinol in frequent users of cannabis. *Neuropharmacology* 33, 2505-2516.
19. **Curran, H. V., Brignell, C., Fletcher, S., Middleton, P., & Henry, J.** (2002). Cognitive and subjective dose-response effects of acute oral delta-9-tetrahydrocannabinol (THC) in infrequent cannabis users. *Psychopharmacology* 164, 61-70.
20. **Green, B., Kavanagh, D., & Young, R.** (2003). Being stoned: a review of self-reported cannabis effects. *Drug and Alcohol Review* 22, 453-460.
21. **Calabria, B., Degenhardt, L., Hall, W., & Lynskey, M.** (2010). Does cannabis use increase the risk of death? Systematic review of epidemiological evidence on adverse effects of cannabis use. *Drug and Alcohol Review* 29, 318-330.
22. **Gable, R. S.** (2004). Comparison of acute lethal toxicity of commonly abused psychoactive substances. *Addiction* 99, 686-696.
23. **Meier, M. H., Caspi, A., Ambler, A., Harrington, H., Houts, R., Keefe, R. S. E., McDonald, K., Ward, A., Poulton, R., & Moffitt, T. E.** (2012). Persistent cannabis users show neuropsychological decline from childhood to midlife. *Proceedings of the National Academy of Sciences*.
24. **Solowij, N., Jones, K., Rozman, M., Davis, S., Ciarrochi, J., Heaven, P., Lubman, D., & Yücel, M.** (2011). Verbal learning and memory in adolescent cannabis users, alcohol users and non-users. *Psychopharmacology* 216, 131-144.
25. **Rubino, T., Zamberletti, E., & Parolaro, D.** (2012). Adolescent exposure to cannabis as a risk factor for psychiatric disorders. *Journal of Psychopharmacology* 26, 177-188.
26. **Thatcher, D. L. & Clark, D. B.** (2008). Adolescents at risk for substance use disorders: Role of psychological dysregulation, endophenotypes, and environmental influences. *Alcohol Research and Health* 31, 168-176.
27. **Vandrey, R., Budney, A. J., Kamon, J., & Stanger, C.** (2005). Cannabis withdrawal in adolescent treatment seekers. *Drug and Alcohol Dependence* 78, 205-210.
28. **Dawes, M. A., Liguori, A., & Dougherty, D. M.** (2006). Cannabis withdrawal among adolescent cannabis users in an outpatient research setting. *American Journal on Addictions* 15, 485-486.
29. **Hesse, M. & Thylstrup, B.** (2013). Time-course of the DSM-5 cannabis withdrawal symptoms in poly-substance abusers. *Biomed Central Psychiatry* 13, 258.
30. **Allsop, D. J., Copeland, J., Norberg, M. M., Fu, S., Molnar, A., Lewis, J., & Budney, A. J.** (2012). Quantifying the clinical significance of cannabis withdrawal. *PLoS ONE* 7, e44864.
31. **Gorelick, D. A., Levin, K. H., Copersino, M. L., Heishman, S. J., Liu, F., Boggs, D. L., & Kelly, D. L.** (2012). Diagnostic criteria for cannabis withdrawal syndrome. *Drug and Alcohol Dependence* 123, 141-147.
32. **Smith, P. H., Homish, G. G., Leonard, K. E., & Collins, R. L.** (2013). Marijuana withdrawal and aggression among a representative sample of U.S. marijuana users. *Drug and Alcohol Dependence* 132, 63-68.
33. **Allsop, D. J., Norberg, M. M., Copeland, J., Fu, S., & Budney, A. J.** (2011). The Cannabis Withdrawal Scale development: patterns and predictors of cannabis withdrawal and distress. *Drug and Alcohol dependence* 119, 123-129.
34. **Levin, K. H., Copersino, M. L., Heishman, S. J., Liu, F., Kelly, D. L., Boggs, D. L., & Gorelick, D. A.** (2010). Cannabis withdrawal symptoms in non-treatment-seeking adult cannabis smokers. *Drug and Alcohol Dependence* 111, 120-127.
35. **Griffin, K. W. & Botvin, G. J.** (2010). Evidence-based interventions for preventing substance use disorders in adolescents. *Child and Adolescent Psychiatric Clinics North America* 19, 505-526.
36. **Lopez-Quintero, C., Cobos, J. P. d. I., Hasin, D. S., Okuda, M., Wang, S., Grant, B. F., & Blanco, C.** (2011). Probability and predictors of transition from first use to dependence on nicotine, alcohol, cannabis, and cocaine: Results of the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC). *Drug and Alcohol Dependence* 115, 120-130.

37. **Creemers, H. E., Harakeh, Z., Dick, D. M., Meyers, J., Vollebergh, W. A. M., Ormel, J., Verhulst, F. C., & Huizink, A. C.** (2011). DRD2 and DRD4 in relation to regular alcohol and cannabis use among adolescents: Does parenting modify the impact of genetic vulnerability? The TRAILS study. *Drug and Alcohol Dependence* 115, 35-42.
38. **Connell, C. M., Gilreath, T. D., Aklin, W. M., & Brex, R. A.** (2010). Social-Ecological Influences on Patterns of Substance Use Among Non-Metropolitan High School Students. *American Journal of Community Psychology* 45, 36-48.
39. **Agrawal, A. & Lynskey, M. T.** (2009). Correlates of later-onset cannabis use in the National Epidemiological Survey on Alcohol and Related Conditions (NESARC). *Drug and Alcohol Dependence* 105, 71-75.
40. **Hyman, S. M. & Sinha, R.** (2009). Stress-related factors in cannabis use and misuse: Implications for prevention and treatment. *Journal of Substance Abuse Treatment* 36, 400-413.
41. **Brodbeck, J., Matter, M., Page, J., & Moggi, F.** (2007). Motives for cannabis use as a moderator variable of distress among young adults. *Addictive Behaviors* 32, 1537-1545.
42. **Fox, C. L., Towe, S. L., Stephens, R. S., Walker, D. D., & Roffman, R. A.** (2011). Motives for cannabis use in high-risk adolescent users. *Psychology of Addictive Behaviors* 25, 492-500.
43. **Lee, C. M., Neighbors, C., & Woods, B. A.** (2007). Marijuana motives: Young adults' reasons for using marijuana. *Addictive Behaviors* 32, 1384-1394.
44. **Hall, W.** (2009). The adverse health effects of cannabis use: What are they, and what are their implications for policy? *International Journal of Drug Policy* 20, 458-466.
45. **Tart, C. T.** (1971). *On being stoned: a psychological study of marijuana intoxication*. Palo Alto: Science and Behavior Books,
46. **Babor, T. F., Rossi, A. M., Sagotsky, G., & Meyer, R. E.** (1974). Group behavior: verbal interaction. In **J. H. Mendelson, A. M. Rossi & R. E. Meyer** (Eds.), *The use of marijuana: a psychological and physiological inquiry* (pp. 61-72). New York: Plenum Press, 61-72.
47. **Schafer, J. & Brown, S. A.** (1991). Marijuana and cocaine effect expectancies and drug use patterns. *Journal of consulting and clinical psychology* 59, 558-565.
48. **Galen, L. W. & Henderson, M. J.** (1999). Validation of cocaine and marijuana effect expectancies in a treatment setting. *Addictive behaviors* 24, 719-724.
49. **Aarons, G. A., Brown, S. A., Stice, E., & Coe, M. T.** (2001). Psychometric evaluation of the marijuana and stimulant effect expectancy questionnaires for adolescents. *Addictive behaviors* 26, 219-236.
50. **Creemers, H. E., Dijkstra, J. K., Vollebergh, W. A. M., Ormel, J., Verhulst, F. C., & Huizink, A. C.** (2010). Predicting life-time and regular cannabis use during adolescence; the roles of temperament and peer substance use: the TRAILS study. *Addiction* 105, 699-708.
51. **Gullo, M. J. & Dawe, S.** (2008). Impulsivity and adolescent substance use: Rashly dismissed as "all-bad"? *Neuroscience and Biobehavioral Reviews* 32, 1507-1518.
52. **von Diemen, L., Bassani, D. G., Fuchs, S. C., Szobot, C. M., & Pechansky, F.** (2008). Impulsivity, age of first alcohol use and substance use disorders among male adolescents: a population based case-control study. *Addiction* 103, 1198-1205.
53. **Pedersen, W.** (1991). Mental health, sensation seeking and drug use patterns: a longitudinal study. *British Journal of Addiction* 86, 195-204.
54. **Gruber, S. A. & Yurgelun-Todd, D. A.** (2005). Neuroimaging of marijuana smokers during inhibitory processing: a pilot investigation. *Cognitive Brain Research* 23, 107-118.
55. **Tapert, S. F., Schweinsburg, A. D., Drummond, S. P., Paulus, M. P., Brown, S. A., Yang, T. T., & Frank, L. R.** (2007). Functional MRI of inhibitory processing in abstinent adolescent marijuana users. *Psychopharmacology (Berlin)* 194, 173-183.
56. **Clark, L., Roiser, J., Robbins, T., & Sahakian, B.** (2009). Disrupted 'reflection' impulsivity in cannabis users but not current or former ecstasy users. *Journal of Psychopharmacology* 23, 14-22.

57. **Solowij, N., Jones, K. A., Rozman, M. E., Davis, S. M., Ciarrochi, J., Heaven, P. C., Pesa, N., Lubman, D. I., & Yücel, M.** (2012). Reflection impulsivity in adolescent cannabis users: a comparison with alcohol-using and non-substance-using adolescents. *Psychopharmacology (Berlin)* 219, 575-586.
58. **Ramaekers, J. G., Kauert, G., Theunissen, E. L., Toennes, S. W., & Moeller, M. R.** (2009). Neurocognitive performance during acute THC intoxication in heavy and occasional cannabis users. *Journal of Psychopharmacology* 23, 266-277.
59. **Ramaekers, J. G., Moeller, M. R., van Ruitenbeek, P., Theunissen, E. L., Schneider, E., & Kauert, G. F.** (2006). Cognition and motor control as a function of delta-9-tetrahydrocannabinol concentration in serum and oral fluid: limits of impairment. *Drug and Alcohol Dependence* 85, 114-122.
60. **McDonald, J., Schleifer, L., Richards, J. B., & Wit, H. d.** (2003). Effects of THC on behavioral measures of impulsivity in humans. *Neuropsychopharmacology* 28, 1356-1365.
61. **Moreno, M., Estevez, A. F., Zaldivar, F., Montes, J. M. G., Gutiérrez-Ferre, V. E., Esteban, L., Sánchez-Santed, F., & Flores, P.** (2012). Impulsivity differences in recreational cannabis users and binge drinkers in a university population. *Drug and Alcohol Dependence* 124, 355-362.
62. **Verweij, K. J. H., Zietsch, B. P., Lynskey, M. T., Medland, S. E., Neale, M. C., Martin, N. G., Boomsma, D. I., & Vink, J. M.** (2010). Genetic and environmental influences on cannabis use initiation and problematic use: a meta-analysis of twin studies. *Addiction* 105, 417-430.
63. **Meyers, J. L. & Dick, D. M.** (2010). Genetic and environmental risk factors for adolescent-onset substance use disorders. *Child and Adolescent Psychiatric Clinics of North America* 19, 465-477.
64. **Hammer, T. & Vaglum, P. E. R.** (1990). Initiation, continuation or discontinuation of cannabis use in the general population. *British Journal of Addiction* 85, 899-909.
65. **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, 1679-1690.
66. **Silins, E., Hutchinson, D., Swift, W., Slade, T., Toson, B., & Rodgers, B.** Factors associated with variability and stability of cannabis use in young adulthood. *Drug and Alcohol Dependence*.
67. **Yule, A. & Wilens, T.** (2011). Familial Influences on Adolescent Substance Use. *Psychiatric Times* 28, 42-43.
68. **Clark, A. E. & Loh'eaç, Y.** (2007). "It wasn't me, it was them!" Social influence in risky behavior by adolescents. *Journal of Health Economics* 26 763-784.
69. **von Sydow, K., Lieb, R., Pfister, H., Höfler, M., & Wittchen, H. U.** (2002). What predicts incident use of cannabis and progression to abuse and dependence?: A 4-year prospective examination of risk factors in a community sample of adolescents and young adults. *Drug and Alcohol Dependence* 68, 49-64.
70. **Van Ours, J. C. & Williams, J.** (2007). Cannabis prices and dynamics of cannabis use. *Journal of Health Economics* 26, 578-596.
71. **Williams, J.** (2004). The effects of price and policy on marijuana use: what can be learned from the Australian experience? *Health Economics* 13, 123-137.
72. **Gillespie, N. A., Neale, M. C., & Kendler, K. S.** (2009). Pathways to cannabis abuse: a multi-stage model from cannabis availability, cannabis initiation and progression to abuse. *Addiction* 104, 430-438.
73. **Orcutt, J. D. & Turner, J. B.** (1993). Shocking numbers and graphic accounts: quantified images of drug problems in the print media. *Social Problems* 40, 190-206.
74. **Blood, R. W., Williams, J., & McCallum, K.** (2003). Representations of public risk: illegal drugs in the Australian press. *Media International Australia* 108, 82-100.
75. **Forsyth, A. J. M.** (2001). Distorted? A quantitative exploration of drug fatality reports in the popular press. *International Journal of Drug Policy* 12, 435-453.
76. **Blendon, R. J. & Young, J. T.** (1998). The public and the war on illicit drugs. *Journal of the American Medical Association* 279, 827-832.

77. **Bright, S. J., Marsh, A., Smith, L. M., & Bishop, B.** (2008). What can we say about substance use? Dominant discourses and narratives emergent from Australian media. *Addiction Research and Theory* 16, 135-148.
78. **Hughes, C. E., Lancaster, K., & Spicer, B.** (2011). How do Australian news media depict illicit drug issues? An analysis of print media reporting across and between illicit drugs, 2003–2008. *International Journal of Drug Policy* 22, 285-291.
79. **Bright, S. J., Bishop, B., Kane, R., Marsh, A., & Barratt, M. J.** (2013). Kronic hysteria: Exploring the intersection between Australian synthetic cannabis legislation, the media, and drug-related harm. *International Journal of Drug Policy* 24, 231-237.
80. **Matthew-Simmons, F., Shanahan, M., & Ritter, A.** (2011). Reported value of cannabis seizures in Australian newspapers: Are they accurate? *Drug and Alcohol Review* 30, 21-25.
81. **Lancaster, K., Hughes, C. E., Spicer, B., Matthew-Simmons, F., & Dillon, P.** (2011). Illicit drugs and the media: Models of media effects for use in drug policy research. *Drug and Alcohol Review* 30, 397-402.
82. **Hall, W. & Degenhardt, L.** (2013). The adverse health effects of chronic cannabis use. *Drug Testing and Analysis*.
83. **Chesher, G. & Hall, W. D.** (1999). Effects of cannabis on the cardiovascular and gastrointestinal systems. In **H. Kalant, W. Corrigall, W. D. Hall & R. Smart** (Eds.), *The health effects of cannabis* (pp. 435-458). Toronto: Centre for Addiction and Mental Health, 435-458.
84. **Jones, R. T.** (2002). Cardiovascular system effects of marijuana. *Journal of Clinical Pharmacology* 42, 58s-63s.
85. **Institute of Medicine.** (1999). *Marijuana and medicine: Assessing the science base*. Washington, DC: National Academy Press,
86. **Sidney, S.** (2002). Cardiovascular consequences of marijuana use. *Journal of Clinical Pharmacology* 42, 64s-70s.
87. **Aronow, W. & Cassidy, J.** (1974). Effect of marihuana and placebo marihuana smoking on angina pectoris. *New England Journal of Medicine* 291, 65-67.
88. **Aronow, W. & Cassidy, J.** (1975). Effect of smoking marijuana and of a high nicotine cigarette on angina pectoris. *Clinical Pharmacology and Therapeutics* 17, 549-554.
89. **Gottschalk, L., Aronow, W., & Prakash, R.** (1977). Effect of marijuana and placebo marijuana smoking on psychological state and on psychophysiological and cardiovascular functioning in angina patients. *Biological Psychiatry* 12, 255-266.
90. **Mittleman, M. A., Lewis, R. A., Maclure, M., Sherwood, J. B., & Muller, J. E.** (2001). Triggering myocardial infarction by marijuana. *Circulation* 103, 2805-2809.
91. **Solowij, N.** (1998). *Cannabis and cognitive functioning*. Cambridge, UK: Cambridge University Press,
92. **Solowij, N. & Battisti, R.** (2008). The chronic effects of cannabis on memory in humans: A review. *Current Drug Abuse Reviews* 1, 81-98.
93. **Fontes, M. A., Bolla, K. I., Cunha, P. J., Almeida, P. P., Jungerman, F., Laranjeira, R. R., Bressan, R. A., & Lacerda, A. L. T.** (2011). Cannabis use before age 15 and subsequent executive functioning. *British Journal of Psychiatry* 198, 442-447.
94. **Fried, P. A., Watkinson, B., & Gray, R.** (2005). Neurocognitive consequences of marihuana—a comparison with pre-drug performance. *Neurotoxicology and Teratology* 27, 231-239.
95. **Asbridge, M., Hayden, J. A., & Cartwright, J. L.** (2012). Acute cannabis consumption and motor vehicle collision risk: systematic review of observational studies and meta-analysis. *British Medical Journal* 344, e536
96. **Moir, D., Rickert, W. S., Levasseur, G., Larose, Y., Maertens, R., White, P., & Desjardins, S.** (2008). A comparison of mainstream and sidestream marijuana and tobacco cigarette smoke produced under two machine smoking conditions. *Chemical Research in Toxicology* 21, 494-502.
97. **Novotný, M., Merli, F., Wiesler, D., Fencl, M., & Saeed, T.** (1982). Fractionation and capillary gas chromatographic—mass spectrometric characterization of the neutral components in marijuana and tobacco smoke condensates. *Journal of Chromatography A* 238, 141-150.

98. **Hoffmann, D., Brunnean, D. K., Gori, G. B., & Wynder, E. L.** (1975). On the carcinogenicity of marijuana smoke. *Recent Advances in Phytochemistry* 9, 63-81.
99. **Wu, T. C., Tashkin, D. P., Djahed, B., & Rose, J. E.** (1988). Pulmonary hazards of smoking marijuana as compared with tobacco. *New England Journal of Medicine* 318, 347-351.
100. **Aldington, S., Williams, M., Nowitz, M., Weatherall, M., Pritchard, A., McNaughton, A., Robinson, G., & Beasley, R.** (2007). Effects of cannabis on pulmonary structure, function and symptoms. *Thorax* 62, 1058-1063.
101. **Sherrill, D. L., Krzyzanowski, M., Bloom, J. W., & Lebowitz, M. D.** (1991). Respiratory effects of non-tobacco cigarettes: a longitudinal study in general population. *International Journal of Epidemiology* 20, 132-137.
102. **Moore, B. A., Augustson, E. M., Moser, R. P., & Budney, A. J.** (2005). Respiratory effects of marijuana and tobacco use in a U.S. sample. *Journal of General Internal Medicine* 20, 33-37.
103. **Reid, P. T., McKenzie, J., Copeland, L., Elton, R., Macleod, J., & Robertson, J. R.** (2011). P263 Cannabis use, respiratory symptoms and lung function, in a North Edinburgh primary care population. *Thorax* 66, A174-A175.
104. **Tashkin, D. P., Coulson, A. H., Clark, V. A., Simmons, M., Bourque, L. B., Duann, S., Spivey, G. H., & Gong, H.** (1987). Respiratory symptoms and lung function in habitual heavy smokers of marijuana alone, smokers of marijuana and tobacco, smokers of tobacco alone, and nonsmokers. *The American review of respiratory disease* 135, 209-216.
105. **Bloom, J. W., Kaltenborn, W. T., Paoletti, P., Camilli, A., & Lebowitz, M. D.** (1987). Respiratory effects of non-tobacco cigarettes. *British Medical Journal (Clinical research ed.)* 295, 1516-1518.
106. **Taylor, D. R., Poulton, R., Moffitt, T. E., Ramankutty, P., & Sears, M. R.** (2000). The respiratory effects of cannabis dependence in young adults. *Addiction* 95, 1669-1677.
107. **Beshay, M., Kaiser, H., Niedhart, D., Reymond, M. A., & Schmid, R. A.** (2007). Emphysema and secondary pneumothorax in young adults smoking cannabis. *European Journal of Cardio-thoracic Surgery* 32, 834-838.
108. **Barsky, S. H., Roth, M. D., Kleerup, E. C., Simmons, M., & Tashkin, D. P.** (1998). Histopathologic and molecular alterations in bronchial epithelium in habitual smokers of marijuana, cocaine, and/or tobacco. *Journal of the National Cancer Institute* 90, 1198-1205.
109. **Sarafian, T. A., Magallanes, J. A., Shau, H., Tashkin, D., & Roth, M. D.** (1999). Oxidative stress produced by marijuana smoke. An adverse effect enhanced by cannabinoids. *American journal of respiratory cell and molecular biology* 20, 1286-1293.
110. **Fligiel, S. E. G., Roth, M. D., Kleerup, E. C., Barsky, S. H., Simmons, M. S., & Tashkin, D. P.** (1997). Tracheobronchial histopathology in habitual smokers of cocaine, marijuana, and/or tobacco. *CHEST Journal* 112, 319-326.
111. **Barbers, R. G., Gong, H., Jr., Tashkin, D. P., Oishi, J., & Wallace, J. M.** (1987). Differential examination of bronchoalveolar lavage cells in tobacco cigarette and marijuana smokers. *The American review of respiratory disease* 135, 1271-1275.
112. **Beals, T. F., Fligiel, S. E. G., Stuth, S., & Tashkin, D. P.** (1989). Morphological alterations of alveolar macrophages from marijuana smokers. *The American review of respiratory disease* 139 (part 2), A336.
113. **Tashkin, D. P.** (2013). Effects of marijuana smoking on the lung. *Annals of the American Thoracic Society* 10, 239-247.
114. **Callaghan, R. C., Allebeck, P., & Sidorchuk, A.** (2013). Marijuana use and risk of lung cancer: a 40-year cohort study. *Cancer Causes Control* 24, 1811-1820.
115. **ornfield, J., Haenszel, W., Hammond, E. C., Lilienfeld, A. M., Shimkin, M. B., & Wynder, E. L.** (2009). Smoking and lung cancer: recent evidence and a discussion of some questions. *International Journal of Epidemiology* 38, 1175-1191.
116. **Huizink, A. C.** (2013). Prenatal cannabis exposure and infant outcomes: Overview of studies. *Progress in Neuro-Psychopharmacology and Biological Psychiatry In Press*.
117. **Fronczak, C. M., Kim, E. D., & Barqawi, A. B.** (2012). The Insults of Illicit Drug Use on Male Fertility. *Journal of Andrology* 33, 515-528.

118. **Moore, T. H. M., Zammit, S., Lingford-Hughes, A., Barnes, T. R. E., Jones, P. B., Burke, M., & Lewis, G.** (2007). Cannabis use and risk of psychotic or affective mental health outcomes: a systematic review. *The Lancet* 370, 319-328.
119. **Large, M., Sharma, S., Compton, M. T., Slade, T., & Nielssen, O.** (2011). Cannabis use and earlier onset of psychosis: a systematic meta-analysis. *Archives of General Psychiatry* 68, 555-561.
120. **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, 473-480.
121. **Andreasson, S., Allebeck, P., Engstrom, A., & Rydberg, U.** (1987). Cannabis and schizophrenia. A longitudinal study of Swedish conscripts. *The Lancet* 2, 1483-1486.
122. **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, 1199.
123. **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, 319-327.
124. **Weiser, M., Knobler, H. Y., Noy, S., & Kaplan, Z.** (2002). Clinical characteristics of adolescents later hospitalized for schizophrenia. *American Journal of Medical Genetics* 114, 949-955.
125. **Fergusson, D. M., Horwood, L. J., & Swain-Campbell, N. R.** (2003). Cannabis dependence and psychotic symptoms in young people. *Psychological Medicine* 33, 15-21.
126. **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, 1212-1213.
127. **Ferdinand, R. F., Sondeijker, F., van der, E. J., Selten, J. P., Huizink, A., & Verhulst, F. C.** (2005). Cannabis use predicts future psychotic symptoms, and vice versa. *Addiction* 100, 612-618.
128. **Henquet, C., Krabbendam, L., Spauwen, J., Kaplan, C., Lieb, R., Wittchen, H.-U., & van Os, J.** (2005). Prospective cohort study of cannabis use, predisposition for psychosis, and psychotic symptoms in young people. *British Medical Journal* 330.
129. **Wiles, N. J., Zammit, S., Bebbington, P., Singleton, N., Meltzer, H., & Lewis, G.** (2006). Self-reported psychotic symptoms in the general population: results from the longitudinal study of the British National Psychiatric Morbidity Survey. *British Journal of Psychiatry* 188, 519-526.
130. **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, 1333-1341.
131. **deBrey, H.** (1983). A cross-national validation of the client satisfaction questionnaire: The Dutch experience. *Evaluation and Program Planning* 6, 395-400.
132. **Manrique-Garcia, E., Zammita, S., Dalmana, C., Hemmingsson, T., Andreasson, S., & Allebeck, P.** (2012). Cannabis, schizophrenia and other non-affective psychoses: 35 years of follow-up of a population-based cohort. *Psychological Medicine* 42, 1321-1328.
133. **Gottesman, I. I.** (1991). *Schizophrenia Genesis: The Origins of Madness*. New York: Henry Holt and Company, 82-102.
134. **Hickman, M., Vickerman, P., Macleod, J., Lewis, G., Zammit, S., Kirkbride, J., & Jones, P.** (2009). If cannabis caused schizophrenia—how many cannabis users may need to be prevented in order to prevent one case of schizophrenia? England and Wales calculations. *Addiction* 104, 1856-1861.
135. **Thomas, H.** (1996). A community survey of adverse effects of cannabis use. *Drug and Alcohol Dependence* 42, 201-207.
136. **Bonn-Miller, M., Zvolensky, M., & Bernstein, A.** (2007). Marijuana use motives: concurrent relations to frequency of past 30-day use and anxiety sensitivity among young adult marijuana smokers. *Addictive Behaviors* 32, 49-62.
137. Budney, A. (2011). American Psychiatric Association DSM-5 Development: R 25 Cannabis Withdrawal. 21.04.2010, from <http://www.dsm5.org/ProposedRevisions/Pages/proposedrevision.aspx?rid=430#>
138. **Bonn-Miller, M. O. & Moos, R. H.** (2009). Marijuana discontinuation, anxiety symptoms, and relapse to marijuana. *Addictive Behaviors* 34, 782-785.

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139. **Crippa, J. A., Zuardi, A. W., Martín-Santos, R., Bhattacharyya, S., Atakan, Z., McGuire, P., & Fusar-Poli, P.** (2009). Cannabis and anxiety: a critical review of the evidence. *Human Psychopharmacology: Clinical and Experimental* 24, 515-523.
 140. **Buckner, J. D., Heimberg, R. G., Schneier, F. R., Liu, S.-M., Wang, S., & Blanco, C.** (2012). The relationship between cannabis use disorders and social anxiety disorder in the National Epidemiological Study of Alcohol and Related Conditions (NESARC). *Drug and Alcohol Dependence* 124, 128-134.
 141. **Substance Abuse and Mental Health Services Administration.** (2009). *Results from the 2008 National Survey on Drug Use and Health: National Findings*. Rockville, MD: Office of Applied Studies, NSDUH Series H-36, HHS Publication No. SMA 09-4434.
 142. **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, 737-748.
 143. **Compton, W. M., Grant, B. F., Colliver, J. D., Glantz, M. D., & Stinson, F. S.** (2004). Prevalence of marijuana use disorders in the United States; 1991–1992 and 2001–2002. *Journal of the American Medical Association* 291, 2114-2121.
 144. **Stinson, F. S., Ruan, W. J., Pickering, R., & Grant, B. F.** (2006). Cannabis use disorders in the USA: Prevalence, correlates and co-morbidity. *Psychological Medicine* 36, 1447-1460.
 145. **Swift, W., Hall, W., & Teesson, M.** (2001). Cannabis use disorders among Australian adults: Results from the National Survey of Mental Health and Well-Being. *Addiction* 96, 737-748.
 146. **Swift, W., Coffey, C., Carlin, J. B., Degenhardt, L., & Patton, G.** (2008). Adolescent cannabis users at 24 years: Trajectories of ongoing and problematic use. *Addiction* 103, 1361-1370.
 147. **Von Sydow, K., Lieb, R., Pfister, H., Höfler, M., Sonntag, H., & Wittchen, H. U.** (2001). The natural course of cannabis use, abuse and dependence over four years: A longitudinal community study of adolescents and young adults. *Drug and Alcohol Dependence* 64, 347-361.
 148. **Anthony, J.** (2006). The epidemiology of cannabis dependence. In **R. A. Roffman & R. S. Stephens** (Eds.), *Cannabis dependence: Its nature, consequences, and treatment* (pp. 58-95). Cambridge: Cambridge University Press, 58-95.
 149. **Grant, B. F. & Pickering, R.** (1999). The relationship between cannabis use and DSM-IV cannabis abuse and dependence: results from the National Longitudinal Alcohol Epidemiologic Survey. *Journal of Substance Abuse* 10, 255-264.
 150. **Von Sydow, K., Lieb, R., Pfister, H., Höfler, M., & Wittchen, H. U.** (2002). What predicts incident use of cannabis and progression to abuse and dependence? A 4-year prospective examination of risk factors in a community sample of adolescents and young adults. *Drug and Alcohol Dependence* 68, 49-64.
 151. **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, 187-194.
 152. **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.
 153. **Stephens, R. S., Babor, T. F., Kadden, R., Miller, M., & Marijuana Treatment Project Research Group.** (2002). The Marijuana Treatment Project: Rationale, design, and participant characteristics. *Addiction* 97, 109-124.
 154. **Solowij, N. & Pesa, N.** (2011). Cannabis and cognition: short- and long-term effects. In **D. Castle, R. Murray & C. D. D'Souza** (Eds.), *Marijuana and Madness* (pp. 91-102). Cambridge: Cambridge University Press, 91-102.
 155. **Solowij, N., Stephens, R. S., Roffman, R. A., Babor, T., Kadden, R., Miller, M., Christiansen, K., McRee, B., & Vendetti, J.** (2002). Cognitive functioning of long-term heavy cannabis users seeking treatment. *Journal of the American Medical Association* 287, 1123-1131.
 156. **Block, R. I., O'Leary, D. S., Hichwa, R. D., Augustinack, J. C., Boles Ponto, L. L., Ghoneim, M. M., Arndt, S., Hurtig, R. R., Watkins, G. L., Hall, J. A., Nathan, P. E., & Andreasen, N. C.** (2002). Effects of frequent marijuana use on memory-related regional cerebral blood flow. *Pharmacology, Biochemistry and Behavior* 72, 237-250.
 157. **Quickfall, J. & Crockford, D.** (2006). Brain neuroimaging in cannabis use: a review. *The Journal of neuropsychiatry and clinical neurosciences* 18, 318-332.



158. **Yucel, M., Solowij, N., Respondek, C., Whittle, S., Fornito, A., Pantelis, C., & Lubman, D. I.** (2008). Regional brain abnormalities associated with long-term heavy cannabis use. *Archives of General Psychiatry* 65, 694-701.
159. **Bolla, K. I., Brown, K., Eldreth, D., Tate, K., & Cadet, J. L.** (2002). Dose-related neurocognitive effects of marijuana use. *Neurology* 59, 1337-1343.
160. **Griffin, K. W., Bang, H., & Botvin, G. J.** (2010). Age of alcohol and marijuana use onset predicts weekly substance use and related psychosocial problems during young adulthood. *Journal of Substance Use* 15, 174-183.
161. **Schubart, C. D., van Gastel, W. A., Breetvelt, E. J., Beetz, S. L., Ophoff, R. A., Sommer, I. E. C., Kahn, R. S., & Boks, M. P. M.** (2011). Cannabis use at a young age is associated with psychotic experiences. *Psychological Medicine* 41, 1301-1310.
162. **Van Ours, J. C. & Williams, J.** (2009). Why parents worry: Initiation into cannabis use by youth and their educational attainment. *Journal of Health Economics* 28, 132-142.
163. **Horwood, L. J., Fergusson, D. M., Hayatbakhsh, M. R., Najman, J. M., Coffey, C., Patton, G. C., Silins, E., & Hutchinson, D. M.** (2010). Cannabis use and educational achievement: Findings from three Australasian cohort studies. *Drug and Alcohol Dependence* 110, 247-253.
164. **Swift, W., Coffey, C., Carlin, J. B., Degenhardt, L., Calabria, B., & Patton, G. C.** (2009). Are adolescents who moderate their cannabis use at lower risk of later regular and dependent cannabis use? *Addiction* 104, 806-814.
165. **Fergusson, D. M. & Boden, J. M.** (2008). Cannabis use and later life outcomes. *Addiction* 103, 969-976.
166. **Degenhardt, L., Coffey, C., Carlin, J. B., Swift, W., Moore, E., & Patton, G. C.** (2010). Outcomes of occasional cannabis use in adolescence: 10-year follow-up study in Victoria, Australia *The British Journal of Psychiatry* 196, 290-295.