Cannabis Use and Mental Health: A Review of Recent Epidemiological Research

T.H. Richardson
Mental Health Research and Development Unit, University of Bath, UK

Abstract: Cannabis is the most commonly used drug in the world. This review examines recent epidemiological research on the relationships between cannabis use and mental health problems. Relationships with depression, anxiety disorders, mania and psychosis are examined, with relevant issues such as the effect of confounding variables, temporal directions and causality being discussed. Factors which influence the relationship such as dose-response effects, age of first cannabis use and risk of mental health problems are also examined. Causality is often difficult to establish, as cannabis is often used by those with mental illness for self-medication. However, there is substantial evidence to suggest that cannabis may induce or exacerbate a number of mental health problems.

Key words: Cannabis, marijuana, epidemiology, mental health

INTRODUCTION

Cannabis is the most commonly used drug in the world, with an estimated 162 million current users, representing 3.9% of the worldwide population (United Nations Office on Drugs and Crime, 2006). Cannabis use is more common in young people and in countries such as the US, Europe, Australia and New Zealand, though there is evidence to suggest that use is increasing globally (Hall and Degenhardt, 2007, 2009; Naisi et al., 2009; United Nations Office on Drugs and Crime, 2006). In recent years evidence has emerged that such use is associated with a number of adverse effects on health (Hall and Degenhardt, 2009; Ahadi and Basharpour, 2010). This includes relationships with a number of mental health problems (Hall and Degenhardt, 2007, 2009; United Nations Office on Drugs and Crime, 2006). The aim of this review is to provide a review of epidemiological research conducted within the past decade which examines the relationship between cannabis use and mental health problems, focusing on the four mental health problems of depression, anxiety disorders, mania and psychosis.

CANNABIS AND DEPRESSION

A number of studies suggest that cannabis use may increase depressive symptoms and the risk of diagnosed depression. In a study of more than fourteen thousand adults from the general population, Cheung et al. (2010) found that diagnosed depression was more common in heavy cannabis users compared to non-users. A longitudinal study by Hayatbakhsh et al. (2007) found that frequent cannabis use in adolescence predicted an increased risk of depressive disorders in young adulthood. Similarly, Van Laar et al. (2007) found that in those with no baseline affective disorders, cannabis use predicted major depression 3 years later. Bovasso (2001) found that cannabis abuse at baseline increased the risk of depression by four fold 14 years later. In particular those who abused cannabis were more likely to experience suicidal ideation and anhedonia (Bovasso, 2001). Rey et al. (2002) similarly reported that adolescents who use cannabis are more likely to have somatic complaints accompanying their depressive symptoms. Brook et al. (2002) found that cannabis use in childhood, adolescence or young adulthood increased the risk of experiencing a major depressive disorder at age 27. Finally, Chen et al. (2002) found that 24% of cannabis users had experienced an episode of major depression by age 40, compared to just 15% of non users. Recent research has also begun to explore the potential causal mechanisms behind these relationships, for example Medina et al. (2007) used functional magnetic resonance imaging to suggest that increased depressive symptoms in adolescent cannabis users are mediated by smaller white matter volume in the brain. Similarly, Gray et al. (2005) showed that maternal cannabis use during pregnancy predicts depressive symptoms in offspring and they suggest that this may be due to damage to neural mechanisms during prenatal development.

Risk factors: Recent research has highlighted that certain factors may place individuals at an elevated risk of experiencing depression after cannabis use. For example Durdle et al. (2008) found that cannabis use increased the risk of depression only in women and Wilcox and Anthony (2004) found that cannabis use increased the risk of suicide attempt and suicidal
Identification related to depression, but only in women. Other research suggests that relationships are still present with men, but are considerably stronger in women (Patton et al., 2002). Research has also found that cannabis is more likely to lead to depression in those of an older age (De Graaf et al., 2010).

**Dose response effects:** Research suggests that more frequent or heavy use of cannabis is associated with a greater risk of depression. Cheung et al. (2010) found that depression diagnoses were more common in those with more frequent use. Lee et al. (2008) found that depression was more common in heavy cannabis users than those who used less heavily and Patton et al. (2002) found that more frequent use was related to more severe depression. Similarly, Chen et al. (2002) found that the number of times cannabis had been used predicted an increased risk of depression and meeting criteria for dependence further increased this risk. There is even evidence that occasional cannabis use is not linked to depression; Poulin et al. (2005) found that depression was increased only in those who used cannabis weekly or daily. However, other research has failed to find evidence of dose-response relationships for cannabis use and depression specifically (Van Laar et al., 2007).

**Age of first use:** As well as frequency of use, the age of first cannabis use may also be related to subsequent depression. Hayatbakhsh et al. (2007) found that the relationship between adolescent cannabis use and depression in early adulthood was strongest in those who began using before the age of 15. A survey of more than eighty-five thousand participants in 17 countries found that early onset cannabis use increased the risk of later depression (De Graaf et al., 2010). This suggests there may be a high risk period where the developing adolescent brain is more vulnerable to the adverse effects of cannabis use. However, other research has found that early use only increases the risk in females (Wilcox and Anthony, 2004), or that it predicts factors such as suicide risk, but not depression specifically (Lynskey et al., 2004).

**Self-medication:** As is often the case with epidemiological research, it is very hard to demonstrate causality in this type of research. It may be, as the evidence above suggests, that cannabis use exacerbates or independently induces depressive symptoms. However there is also evidence to suggest that the reverse temporal direction might occur- it might be that depression precedes cannabis use and cannabis may be used as an attempt to self-medicate depressive symptoms. For example Wittchen et al. (2007) found that a diagnosis of major depressive disorder predicted later cannabis use in adolescents. There is also evidence that individuals use cannabis directly to cope with symptoms related to depression; Green and Ritter (2000) found that those who used cannabis as a way to cope with their problems were more depressed than those who use cannabis for other reasons. Similarly, Buckner et al. (2007) found that relationships between depression and increased cannabis use were moderated by distress tolerance; those who were less able to tolerate negative emotional states were more likely to use cannabis. Beck et al. (2009) found further evidence for self medication as those who used cannabis to help with emotional pain had higher levels of depressive symptoms. However, other research suggests that depressive symptoms do not predict later cannabis use (Bovasso, 2001; Chabrol et al., 2005; Patton et al., 2002) or that this is only the case in men (Repetto et al., 2008). Those with depression also appear to use cannabis for the same reasons as without depression and intoxication often leads to exacerbation rather than relief of symptoms (Arendt et al., 2007). This suggests that self medication of depressive symptoms is rare.

**Confounding variables:** It has been suggested that cannabis does not induce depression, but rather epidemiological research finds relationships between the two because certain factors increase the risk of both depression and cannabis independently of one another. Thus the two appear to be causally related, but correlations are accounted for by these confounding variables. For example a study by Degenhardt et al. (2001b) found a relationship between cannabis and depression, however once demographic variables, neuroticism and other drug use were controlled for, this relationship was no longer statistically significant. Harder et al. (2006) studied this issue in more than 12 thousand people and found that there was no relationship after adjusting for baseline risk factors for cannabis and depression. De Graaf et al. (2010) found that relationships between cannabis use in adolescence and adult depression were mediated by child behaviour and conduct problems and Green and Ritter (2000) found that relationships were mediated by marital and employment status, education and other drug use. Lynskey et al. (2004) found that cannabis increased the risk of major depression in dizygotic, but not monozygotic twins, suggesting that shared genetic and environmental risk factors contribute to both outcomes. Denson and Earleywine (2006) found that those using cannabis for medical reasons had higher levels of depression, thus it may be that research fails to take into account the effects.
of illness and disability. Mariani et al. (2009) found that the research setting influences relationships, with a stronger association between cannabis use and depression in clinical trials compared to research where treatment was not offered. This suggests that help seeking, rather than cannabis useful itself, is related to depressive symptoms. However, many studies have controlled for potential confounding variables such as gender, age, ethnicity, marital status, education level, socioeconomic status, alcohol, tobacco and other drug use as well as baseline mental health and have found that cannabis use is still associated with an increased risk of depression (Bovasso, 2001; Brook et al., 2002; Chen et al., 2002).

No relationship: Finally, it is worth reviewing a small body of recent literature which has failed to find a relationship between cannabis use and depression. Zvolensky et al. (2006) found that cannabis use, abuse or dependence did not predict the incidence of major depression. A 10 year follow up study by Degenhardt et al. (2010) found that occasional cannabis use in adolescence did not predict depression in adulthood. Monschauer et al. (2006) demonstrated a link with externalising problems such as aggression, but not internalising problems such as depression. Finally, one study reported that regular cannabis users in fact showed lower levels of depression, with fewer somatic complaints and increased positive affect (Denson and Earlywine, 2006).

CANNABIS AND ANXIETY DISORDERS

Recent research has begun to examine the complex relationship between cannabis use and anxiety disorders. Bonn-Miller et al. (2005) found that cannabis use was related to increased anxiety and frequency of use predicted severity of anxiety. A later study by Bonn-Miller et al. (2008) found that those who used cannabis frequently had the highest levels of anxiety. Cheung et al. (2010) sampled more than fourteen thousand participants from the general population and found that anxiety disorders were present in 18% of heavy cannabis users compared to 8.7% of non users and more frequent risk further increased the risk. A longitudinal study by Hayatbakhsh et al. (2007) found that frequent cannabis use in adolescence was related to an elevated likelihood of anxiety disorders in young adulthood. This relationship was particularly strong for those who began using cannabis before the age of 15 (Hayatbakhsh et al., 2007). Similarly Patton et al. (2002) found that weekly use in adolescence increased the risk of later anxiety by two fold and daily use increased the risk five fold. Finally, laboratory evidence suggests that cannabis may elevate anxiety in the short term during intoxication; Bhattacharyya et al. (2009) found that administering the active component of cannabis; Δ9-Tetrahydrocannabinol (THC) increased acute anxiety in healthy subjects.

Confounding variables: There is however evidence to suggest that a number of these relationships are mediated or moderated by confounding variables such as other substance use and shared socio-demographic variables. For example Degenhardt et al. (2001a) found that relationships between cannabis and anxiety were no longer statistically significant once neuroticism, demographic variables and other drug use were controlled for. Similarly a 3 year longitudinal study of more than three thousand participants by Van Laar et al. (2007) found that relationships no longer remained after confounders such as age, gender, employment, urbanicity, marital status, childhood trauma and abuse and baseline mental health and drug use were taken into account. There is also evidence to suggest that the relationship between cannabis and anxiety is different for different cultural and ethnic groups (Gilde et al., 2006). The effect of other substance use appears to be particularly important; research has found that alcohol and tobacco use are related to anxiety, but cannabis is not (Cranford et al., 2009; Low et al., 2008). Bonn-Miller et al. (2010) further found that cannabis increased anxiety only when combined with tobacco use. However other studies have controlled for additional substance use and found that relationships between cannabis use and anxiety remain statistically significant (Bonn-Miller et al., 2005; Patton et al., 2002).

Self-medication: As with work on cannabis and depression, research suggests that anxiety may increase the risk of cannabis use, perhaps due to self-medication. For example Wittchen et al. (2007) found that in adolescents, anxiety disorders predicted later cannabis use. This was particularly strong for panic, but also for separation anxiety (Wittchen et al., 2007). Self-medication is suggested by research which demonstrates higher levels of anxiety in those who reported that they use cannabis to cope with problems (Bonn-Miller et al., 2008) and that such coping motives for cannabis use mediate the relationship between cannabis and anxiety (Johnson et al., 2009). It has also been demonstrated that anxiety sensitivity- how much one is afraid of symptoms of anxiety- relates to reasons for cannabis use (Comeau et al., 2001) and that this moderates the relationship between cannabis use and physiological symptoms of anxiety (Buckner et al., 2009).
individuals who find the physiological sensations which accompany anxiety particularly unpleasant may use cannabis as a way to cope with these. Other work however has found that anxiety does not predict later cannabis use (Chabrol et al., 2005; Patton et al., 2002; Tournier et al., 2003). In addition evidence previously cited suggests that cannabis intoxication may increase acute anxiety symptoms (Bhattacharyya et al., 2009), thus cannabis may not always be used for self-medication.

**Cannabis and panic attacks:** Recent research has shown that panic disorder is particularly strongly related to cannabis use (Wittchen et al., 2007). Dannon et al. (2004) found that many patients with panic disorder had their first panic attack within 24 h of using cannabis, suggesting that cannabis may induce panic attacks. Similarly, Zvolensky et al. (2006) found that a lifetime history of cannabis dependence increased the risk of panic attacks. Furthermore, the age of first panic attack was lower for those with cannabis dependence and onset of panic occurred at the same time as cannabis use (Zvolensky et al., 2006), further suggesting that cannabis may lead to panic attacks. However, other research has found that such a relationship no longer remains significant once tobacco use is controlled for (Zvolensky et al., 2008, 2010).

**Cannabis and social anxiety disorder:** Another anxiety disorder which has been shown to commonly have comorbid cannabis use disorders is Social Anxiety Disorder (SAD). Indeed, some research has found that SAD is the only anxiety disorder related to cannabis use (Buckner et al., 2006, 2008; Buckner and Schmidt, 2009). However, the evidence seems to suggest that SAD increases the risk of using cannabis, rather than cannabis inducing or exacerbating symptoms; Buckner et al. (2008) found that SAD increased the risk of cannabis dependence 14 years later. Research has shown that such a relationship is moderated by stress reactivity- the perceived ability to cope with unpredictable events and stimuli (Buckner et al., 2006). Thus those with SAD do not feel able to cope and use cannabis to try to ease tension relating to their anxiety. The relationship is also mediated by the anticipated effects of intoxication, with those with SAD expecting more cognitive and behavioural impairment (Buckner and Schmidt, 2009; Buckner et al., 2009). Those with SAD are also more likely to use cannabis to cope and to conform with others (Buckner et al., 2007), suggesting that those who are socially anxious want to cope with their anxiety and fit in with their peers and often use cannabis to do so.

**Withdrawal symptoms:** Finally, it is important to note that a small body of research suggests that those who use cannabis may experience elevated anxiety when they stop use. Born-Miller and Moos (2009) found that in those receiving treatment for cannabis addiction, stopping cannabis use increased anxiety. In addition, more heavy users experienced the most anxiety and this predicted relapse to cannabis abuse (Born-Miller and Moos, 2009). Born-Miller et al. (2007) showed that those with higher anxiety sensitivity are more likely to experience withdrawal symptoms from stopping cannabis use. Thus it is possible relationships between cannabis use and anxiety occur as a result of withdrawal symptoms.

**CANNABIS AND MANIA/HYPMONANIA**

**Cannabis use in bipolar disorder:** Cannabis is commonly abused in patients with bipolar disorder (Bauer et al., 2005) and use can have an impact on the symptomatology of bipolar patients, with increased duration and severity of manic phases (Strakowski et al., 2000, 2007; Van Rossum et al., 2009). Research with bipolar patients has found that the majority begin to use cannabis before their first episode (Frank et al., 2007) and that cannabis use predicts future hypomanic or manic symptoms (Baethge et al., 2008). This suggests that cannabis use can exacerbate manic symptoms, though whether it induces it in its own right is a matter of controversy. Diagnostic manuals such as the DSM-IV-TR and ICD-10 hold that manic or hypomanic episodes cannot be directly due to the effects of drugs such as cannabis, but evidence has been presented to argue that a diagnosis of ‘cannabis-induced mania’ should be introduced (Bertolin-Guillen et al., 2007).

**Cannabis use and mania in the general population:** Recent epidemiological research has begun to demonstrate possible relationships between cannabis use and hypomanic/manic symptoms in the general population. van Laar et al. (2007) studied more than 3 thousand people from the general population over 3 years and found that in those with no baseline affective disorders, cannabis use predicted an increased risk of a bipolar disorder diagnosis 3 year later. Richardson and Garavan (2010) found that, in a national sample of students, those currently using cannabis had higher levels of hypomania. Those who used more heavily and began using at an earlier age had higher scores still (Richardson and Garavan, 2010). Henquet et al. (2006) conducted a longitudinal study in the Netherlands, finding that cannabis use predicted later manic symptoms. There was also evidence for a dose-response effect with
more severe manic symptoms in heavier users and the results suggested that increased manic symptoms were the result of prolonged exposure rather than the acute effects of intoxication (Henquet et al., 2006). Thus there is evidence to suggest that cannabis may induce manic symptoms, though few heavy cannabis users actually meet the diagnostic criteria for bipolar disorder (Arendt and Munk-Jorgensen, 2004).

Self-medication: There is also evidence that manic or hypomanic symptoms increase the risk of future cannabis use. Wittchen et al. (2007) found that, in adolescents, hypomania/mania was related to later cannabis use disorders. A 20 year longitudinal study by Merikangas et al. (2008) found that those with manic symptoms were more likely to go on to develop cannabis abuse or dependence. Kwapi et al. (2000) also found that those who scored highly on a measure of hypompanic personality were more likely to have cannabis abuse or dependence 13 years later. Thus there is evidence to suggest that even mild manic symptoms increase the risk of future cannabis use. However, other studies have found that manic symptoms do not predict future cannabis use (Henquet et al., 2006).

CANNABIS AND PSYCHOSIS

Cannabis and schizophrenia: Work has documented the impact of cannabis use on schizophrenia and psychotic disorders. Degenhardt et al. (2007) found that in those with a diagnosis of schizophrenia, cannabis use increased the severity of psychosis. Similarly, Katz et al. (2010) found increased hallucinations and grandiosity in patients using cannabis. Half of all first episode psychosis patients have used cannabis (Barnett et al., 2007) and the majority begin using cannabis before the emergence of their psychotic symptoms (Sevy et al., 2010), suggesting that cannabis may induce or exacerbate psychosis. The number of patients with cannabis use before psychosis is in fact increasing over time (Boydell et al., 2006). Cannabis use may also lead to a poorer long term prognosis, as stopping cannabis use is associated with better functioning and fewer negative symptoms (Gonzalez-Pinto et al., 2009).

Cannabis and psychotic symptoms in the general population: There is also evidence that cannabis use increases the risk of psychotic symptoms in the general population. Arseneault et al. (2002) found that those who used cannabis age 18 had more psychotic symptoms at age 26, after accounting for baseline psychotic symptoms. This relationship was stronger than for any other drug (Arseneault et al., 2002). Fergusson et al. (2005) found that daily users had higher levels of psychosis and cannabis appeared to lead to psychosis not vice versa. Van Os et al. (2002) found that cannabis use predicted psychosis 3 years later, including severe symptoms requiring care. The authors estimated that half of all diagnosed psychotic disorder is related to cannabis use (Van Os et al., 2002). Degenhardt et al. (2001b) estimated that those with cannabis dependence have an eleven fold likelihood of psychosis compared to non users. Some work suggests that any lifetime use is related to increased psychosis risk (Miettunen et al., 2008), whereas other work suggests this effect is only seen in those currently using cannabis (Richardson et al., 2010). A number of additional studies have further documented a relationship between cannabis use and more severe psychosis and elevated hallucinations and delusions specifically (Ferdinand et al., 2005a; Fergusson et al., 2003; Henquet et al., 2005; Hides et al., 2009; McGrath et al., 2010; Richardson et al., 2010; Rossler et al., 2007). However it is important to note that some research has failed to find such a relationship (Cranford et al., 2009) and epidemiological research has found that trends of increasing cannabis use do not correspond with an increased prevalence of psychotic illness in the general population (Degenhardt et al., 2003; Frish et al., 2009).

Confounding variables: There is limited evidence to suggest that relationships between cannabis and psychosis may be mediated by confounding variables. Van Dam et al. (2008) found that relationships were accounted for by cannabis users being more likely to use other drugs in particular stimulants and ecstasy. However research has controlled for confounding variables such as child behaviour problems and abuse, early psychotic symptoms, other drug use, socio-economic status and family history of mental health and still found strong relationships (Ferdinand et al., 2005a; Fergusson et al., 2003; Henquet et al., 2005).

Dose response effects: As with other mental health problems, work suggests that more heavy use is associated with a greater risk of psychosis suggesting a dose-response effect (Miettunen et al., 2008). Richardson et al. (2010) found that those who used more frequently scored higher on a measure of hallucinatory experiences and there was a correlation with increased level of consumption being related to more psychotic symptoms including hallucinations and delusions. Other work suggests that more frequent use increases the severity of positive and negative psychotic symptoms (Skinner et al., 2010; Korver et al., 2010) and moving from

moderate to daily use increases the risk of psychosis (Compton and Ramsay, 2009). Work suggests a dose response relationship between extent of cannabis use and risk of a diagnosis of schizophrenia (Zammit et al., 2002), with long term use and the use of strong cannabis increasing the risk of psychotic illness (Di Forti et al., 2009). Other work however showed that those using cannabis more frequently have lower levels of psychotic symptoms than those who use less frequently (Hides et al., 2009). Richardson and Gallagher (2010) suggest that this may be because those who use frequently have an increased tolerance to the acute effects of cannabis.

**Age of onset:** There is evidence that age of first cannabis use and age of onset of psychosis are related. Arsenault et al. (2002) found that, at age 26, those who began using cannabis early on in adolescence had more severe psychotic symptoms. Additional research supports this finding that early use predicts more severe psychosis later on in life (McGrath et al., 2010; Skinner et al., 2010; Stefanis et al., 2005). Work also suggests that those who use cannabis have a lower age of onset of psychosis (Arendt et al., 2005; Veen et al., 2004), in particular for those with heavy use (Sugranyes et al., 2009). Earlier first cannabis use has also been related to an earlier first psychotic episode (Barnett et al., 2007; Barrigon et al., 2010). Some research even suggests that cannabis only increases the risk of psychosis if it is used early on in adolescence (Konings et al., 2008), though other research has failed to find an effect of age of first cannabis use (Richardson et al., 2010).

**Psychosis risk:** There is evidence that cannabis is more likely to lead to psychosis in those who are already at risk of psychotic illness (Henquet et al., 2005; Verdoux et al., 2003). For example, a study of more than 2 million people in Denmark found that those who had a mother with schizophrenia were more likely to experience psychosis from cannabis use (Arendt et al., 2008). Additional evidence suggests that a genetic vulnerability to psychosis increases the risk of experiencing psychotic symptoms from cannabis use (Henquet et al., 2006) and that specific susceptibility genes may moderate the relationship (Caspi et al., 2005; Henquet et al., 2009). Those with high levels of schizotypal personality are also more likely to experience psychotic like symptoms from cannabis use (Barlow and Lewis, 2008; Mason et al., 2009; Stirling et al., 2008). Cannabis use may combine with other environmental risk factors in an additive fashion to increase risk: Harley et al. (2009) found that both childhood trauma and cannabis use independently Gallagher the risk of psychosis and having both elevates the risk further still. Shevlin et al. (2009) found that cannabis use only increased the risk of psychotic symptoms in those with a history of sexual abuse in childhood.

**Acute effects of intoxication:** As discussed above, research has demonstrated that cannabis increases the risk of psychosis in the long term. However there is also evidence to suggest that users may experience acute psychotic like symptoms during intoxication. D’Souza et al. (2004) gave THC to healthy participants and found it produced a number of positive symptoms, altered perception and cognitive deficits seen in psychosis such as poor working memory. Bhattacharyya et al. (2009) found a similar effect of THC on psychotic symptoms and using Functional Magnetic Resonance Imaging found evidence to suggest that impaired ventriculotial functioning during intoxication may underlie the effects of cannabis on psychosis. Hammersley and Leon (2006) found that a non-clinical sample of students often reported a number of effects under cannabis intoxication which would be seen as psychotic symptoms if they occurred in everyday life, such as seeing things not there and being paranoid. There is evidence that the acute effects of cannabis intoxication on psychotic experiences are mediated by vulnerability to psychosis (Verdoux et al., 2003). Such acute psychotic symptoms during intoxication are more common in those with a schizotypal personality (Mason et al., 2009), or in those with specific susceptibility genes (Henquet et al., 2009). Corcoran et al. (2008) found that those who were high risk for psychosis experienced more perceptual disturbances during periods of cannabis use and Peters et al. (2009) found that high risk individuals reported increased suspiciousness after cannabis use. There is evidence that those with a psychotic illness are more likely to experience acute symptoms during intoxication such as increased auditory and visual hallucinations (Henquet et al., 2010; Peters et al., 2009). Thus cannabis may exacerbate psychotic symptoms in the short term in those with psychosis.

**A bi-directional relationship?** As previously reviewed, there is evidence to suggest that cannabis increases the risk of later psychotic symptoms. Most evidence suggests that psychosis does not predict later cannabis use (e.g., Henquet et al., 2005). However there is evidence that both causal pathways may be relevant: Ferdinard et al. (2005b) conducted a longitudinal study.
of adolescents finding that cannabis use predicted future psychotic symptoms, but psychotic symptoms also predicted future cannabis use. The authors argue that this suggests a bi-directional relationship, with cannabis inducing psychotic symptoms as well as being used for the self-medication of psychosis. Other authors have hypothesised that those with early onset sub-clinical psychosis may be more likely to use cannabis later on, perhaps to try to cope with symptoms and this use may in turn trigger a full psychotic episode (Richardson, 2010).

Is there a specific cannabis psychosis?: There is some evidence that cannabis induced psychosis may differ from psychosis which is not related to cannabis use. Half of those who experience cannabis induced psychosis go on to be diagnosed with schizophrenia, with this being more likely in young men (Arendt et al., 2005). Some research has found few differences in symptomatology and no differences in family history in those with schizophrenia compared to cannabis induced psychosis, suggesting that there may not be a distinct cannabis psychosis (Boyedell et al., 2007). However, other work has found that cannabis induced psychosis differs from other psychotic illness, with increased visual hallucinations and sensory disturbances as well as a greater likelihood of expansive mood and depersonalisation (Nunez and Gurpegui, 2002). Thus some have concluded that cannabis induced psychosis may represent an early warning sign of later schizophrenia, rather than a diagnosis in itself (Arendt et al., 2008).

CONCLUSIONS

Recent epidemiological work has linked cannabis use with a number of mental health problems. There is evidence to suggest that cannabis may be used to cope with the symptoms of depression, but also that cannabis use increases the risk of later depression. This relationship is stronger with those who use heavily and begin use early on and it may also be stronger in women. However, relationships between cannabis and depression may often occur as a result of shared sociodemographic variables. Recent research has shown that cannabis use is frequently related to anxiety, though this is often the result of shared variables in particular tobacco use and this may represent withdrawal symptoms from the drug. Cannabis use for self-medication of anxiety is common in particular in those with social anxiety disorder. The strongest evidence for a causal role of cannabis in anxiety is that it may induce panic attacks. Cannabis is frequently used in bipolar disorder and has been shown to predict manic and hypomanic symptoms in the general population. However, those with such manic symptoms are at increased risk of later cannabis, thus relationships are complex. The largest body of literature concerns cannabis and psychosis where epidemiological research has shown that cannabis use considerably increases the risk of psychotic symptoms and diagnoses such as schizophrenia. There is little evidence that self-medication accounts for this relationship, though some have suggested the relationship may be bi-directional. More frequent use entails an elevated risk and early use increases the likelihood of psychosis and an earlier onset of psychotic symptoms. The relationship is much stronger in those already at risk of psychosis. Cannabis can induce acute psychotic like experiences during intoxication, as well as increase more chronic psychotic symptoms, though whether a specific cannabis psychosis exists is controversial.

The past decade has seen a considerable body of epidemiological research documenting a relationship between cannabis use and mental health problems. Though causality is often unclear there is substantial evidence to suggest that cannabis may induce or exacerbate the symptoms of certain mental illnesses. This is important for clinicians to take into account when treating those with mental illness or cannabis use disorders. Future research using longitudinal designs which control for confounding variables will help explain the causal mechanisms underlying these complex relationships.

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