

EXPLORING THE RELATIONSHIP BETWEEN CANNABIS AND PANIC

Thesis submitted to the University of Leicester in partial fulfilment of the Doctorate
in Clinical Psychology.

Submitted February 2010

By

David Ward

Declaration

This thesis submitted for the degree of Doctorate in clinical psychology entitled 'Exploring the relationship between cannabis and panic', is based on work conducted by the author whilst residing in the Department of Clinical Psychology at the University of Leicester between September 2005 and February 2010. All of the work recorded in this thesis is original unless otherwise acknowledged in the text or by references.

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STRUCTURE AND WORD COUNT

	Word count without references	Word count with references
SECTION A: Thesis Abstract	300	300
SECTION B: Literature Review	8,301	9,630
SECTION C: Research Report	10,644	12,863
SECTION D: Critical Appraisal	4,701	4,792
SECTION E: Appendices		

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Section A

1. Thesis Abstract

- 1.1 Background:** Despite Cannabis being the most widely used recreational drug in the western world (Earlywine, 2002) little is known about its potential association with anxiety and panic pathology.
- 1.2 Literature Review:** A systematic literature review was conducted with twenty-nine studies critically reviewed. Papers suggested contradictory and equivocal results across all research designs and anxiety disorders. Whilst a significant number of studies have observed an association between cannabis and anxiety/anxiety disorders, the nature and direction of that association is still a point of contention.
- 1.3 Research Report:** No known British empirical research has focused on exploring relationships between cannabis and panic attacks. Also no known research has investigated the differential effects of consuming different types of cannabis on panic pathology. Inspired somewhat on established research (e.g. Zolvensky *et al.*, 2006a) a cross-sectional study was undertaken to explore the potential relationship between cannabis and anxiety. A self-selecting opportunity sample of 306 students drawn from both of Leicester's universities completed a battery of questionnaires concerning cannabis use, tobacco use, panic attack history, alcohol use, poly-substance use and various psychometrics. Significant levels of both cannabis use and panic attack history were reported among the sample. Survival analysis revealed cannabis users were of significant increased risk (OR 2.01) of experiencing a panic attack compared to non-users. Mann-Whitney analysis found cannabis users who use mainly high potency 'sensimillia' experienced significantly more lifetime panic attacks than those who used other types. Limitations are explored.
- 1.4 Implications:** The research report concludes that cannabis use is a risk factor in experiencing panic attacks and experiencing more lifetime attacks. High potency cannabis further increases this risk. Education for substance misuse and mental health professionals is recommended along with cannabis use forming part of assessment for panic attacks/disorder.
- 1.5 Critical Appraisal:** Reflective appraisal of the research process is presented alongside key learning points.

Section B

Exploring the Association between Cannabis and Anxiety:

A Critical Analysis

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1. Abstract

1.1 Introduction. Cannabis is the most prevalent recreational drug used in the UK (Earlywine, 2002). Its link with psychosis has received much research and media attention. Whilst a recent review (Degenhardt, Hall & Lynskey, 2002) has examined potential links with depression, there is a paucity of research focused on any association with anxiety and a lack of systematic review. This paper has been written to collate and analyse the current state of research regarding cannabis and anxiety.

1.2 Method A systematic search of the literature was undertaken to identify papers examining the potential association between cannabis use, anxiety and anxiety disorders.

1.3 Results Twenty-nine studies were selected for critical review. Papers suggested contradictory and equivocal results across all research designs and anxiety disorders. The current literature is fraught with difficulties in finding a consistently replicable direction of association between cannabis and anxiety. Findings from studies reviewed are grouped into recognisable categories, ‘Cannabis and Panic’, ‘Cannabis and Subjective Experience’, ‘Cannabis and Social Anxiety’ and ‘Cannabis, Anxiety and Anxiety Disorders’

1.4 Discussion A broad critique of the literature is given, and the difficulties associated with conducting research with drug-using populations are noted. A significant number of studies have observed an association between cannabis and anxiety/anxiety disorders, however the nature and direction of that association is still a point of contention.

2. Introduction

Cannabis is one of the most widely used recreational drugs in the world, with between 200 and 300 million people worldwide who report smoking marijuana (Earlywine, 2002). It is the most commonly used illicit substance in Canada (Russell, Newman & Bland, 1994) as well as in the USA. A majority of young people in the UK, USA, New Zealand and Australia now use cannabis recreationally (Patton, Coffey, Carlin Degenhardt, Lynskey & Hall, 2002) and there are indications that its use is rising. Among various physical and psychosocial concerns pertaining to cannabis use, its effects upon mental health have received increasing attention within the health professions.

Whilst cannabis use and possible contribution towards psychosis aetiology has received much contemporary research interest, the effects upon other mental health problems has received considerably less attention (Degenhardt, Hall & Lynskey 2002). Nevertheless, an extensive literature review into the association between cannabis use and depression was conducted by Degenhardt *et al.* (2002) which concluded that whilst the idea of a causal relationship remained ambiguous, heavy cannabis use was associated with depression and may increase depressive symptoms in some users. Whilst cannabis effects upon depression have received some research attention, there has been a paucity of research exploring the association between cannabis and anxiety disorders (Buckner, Schmidt, Bobadilla & Taylor, 2006). The present paper will critically examine the research that has investigated the effects of cannabis upon anxiety and the various anxiety disorders. To begin, a rationale for conducting the present review will be discussed followed by key questions to be addressed by the current paper. This will then be followed by a detailed search strategy explanation. Relevant research in the area will then be discussed along with the most pertinent methodological limitations. A more detailed and comprehensive methodological analysis of caveats in the present area will then be presented. Finally, the implications of the findings in relation to Clinical Psychology will be offered along with considerations for future research.

2.1 Why expect cannabis to have an association with anxiety states and disorders?

Before exploring the potential associations between anxiety, anxiety disorders and cannabis use, it would be pertinent to consider briefly some of the potential reasons as to why such an association may exist. These theoretical possibilities range from the neurological to the psychosocial.

Over the last fifteen years an increasing amount of research has examined the effect of cannabinoids in animal samples. The major psychoactive ingredient in cannabis is tetrahydrocannabinol (THC); however, this is only one of 61 different cannabinoids present in cannabis (Szuster, Pontius & Campos, 1988). A series of studies have shown that administration of high doses of THC produces intense reactions in nearly all subjects (Tassinari, Ambrosetto, Peraita & Gestaut, 1976). Research has shown that THC given in a pure form has an anxiogenic effect in healthy volunteers (Zuardi, Shirakawa, Finkelfarb, & Karniol, 1982). Indeed more recent research has shown THC to have a greater effect on euphoria and anxiety symptoms (Williamson & Evans, 2000), whereas cannabidiol (CBD), another important cannabinoid, has been found to reduce the anxiety reaction caused by THC in both human and animal studies (Carlini & Santos, 1970; Karniol, 1974). Research into cannabis potency shows that the amount of THC in cannabis has increased over the last ten years (Potter, Clark & Brown, 2008; Smith, 2005; Hardwick & King, 2008) whilst cannabidiol has been falling.

Much of the data from research with animals provide evidence of dose-dependent bidirectional modulation of anxiety by the cannabinoid system in that both anxiogenic and anxiolytic effects have been observed (Viveros, Marco & File, 2005). Brain regions such as the hippocampus, cortex and amygdala which are directly involved in the regulation of mood and emotional behaviour have been found to have high concentrations of CB1 receptors (Witkin, Tzavara & Nomikos, 2005). THC has been found to be a cannabinoid receptor agonist and thus may elicit a physiological anxiety response through this mechanism. Therefore, while the neurological explanations are complex they do provide a good basis for examining the effects of cannabis on anxiety in human beings.

Other possibilities involve a more psychological basis for the association. For example some individuals may have a greater sensitivity to the psychoactive effects of cannabis and perceive them as negative and unwanted. This may in turn create anxiety, as once ingested it would take some time to wear off. Indeed Zvolensky, Bonn-Miller, Bernstein, McLeish, Feldner, and Leen-Feldner, (2006b) have suggested underlying anxiety sensitivity, catastrophic cognitions and negative affect as potential precursors for any relationship between cannabis and anxiety reactions. Some individuals may naturally dislike the loss of control associated with the ingestion of psychoactive drugs, and thus may experience anxiety or even panic as a result. Zvolensky, Bernstein, Sachs-Ericsson, Schmidt, Buckner and Bonn-Miller (2006a) suggest that panic-related learning whilst under the influence of cannabis may be more likely due to repeated affect-learning with aversive interoceptive cues being a risk mechanism. As paranoia has been observed as one of the negative side-effects of cannabis use, it could be that paranoid thinking may lead to catastrophic interpretation of alterations in CNS activity, leading to increased anxiety and the possibility of panic attacks and other anxiety disorders such as GAD and social phobia. In addition it may be that people with anxiety or stress disorders use cannabis to self-medicate their symptoms, rather than cannabis having any sort of aetiological role.

Lastly, another possible explanation is that any association between cannabis and anxiety or anxiety disorders is in some way demographically or socially mediated. There could be common social or demographic causal factors that increase the likelihood of both cannabis use and anxiety. This argument has been raised vehemently in the literature (see Macleod, Oakes, Copello, Crome, Egger & Hickman 2004). Alternatively, anxiety or cannabis use could lead to life events, environmental influences or social circumstances that make the other more likely to occur. This brief examination of potential explanations leads naturally to a number of questions that the present review will attempt to address.

2.2 Key questions

1. Does cannabis use cause an increase or decrease in anxiety symptoms among users?
2. Do people who experience anxiety and anxiety-type disorders use cannabis to self-medicate or relieve anxiety symptoms?
3. Can cannabis use trigger anxiety disorders in vulnerable people?
4. If cannabis does have an anxiogenic effect, is there a dose-related relationship?
5. Is there an association between cannabis use and anxiety disorders; if so what is the direction of the association and what can explain such association?

3. Method

3.1 Search Strategy

Search terms were decided upon through examining other literature reviews and meta-analysis pertaining to cannabis use (Degenhardt, *et al.*, 2003; Macleod *et al.*, 2004; Grant, Gonzalez, Carey, Natarajan & Wolfson, 2003). Databases ‘PsychINFO’ (including PsychARTICLES), ‘Web of Science’ and ‘Ovid MEDLINE(R)’ yielded 667, 736 and 818 abstracts respectively when utilised in April 2007 with the following search criteria:

Marijuana OR marihuana OR tetra-hydrocannabinol OR THC OR cannabis OR cannabinoid OR hashish

AND

anxiety OR panic OR anxiolytic OR anxiogenic OR GAD OR OCD OR phobia OR social phobia OR agoraphobia OR agoraphobic OR compulsive OR obsessional OR hypochondriasis OR social anxiety

This resulted in more papers than was feasible for one researcher to review, and therefore there may be some relevant evidence that is not included in the present review. However this state of affairs is not uncommon in systematic reviews as Macleod *et al.* (2004) allude to. Refworks was used to remove duplicate papers from the three database searches. All abstracts were examined for relevance to the key questions posed and then rank ordered for applicability to the current review. Further papers were identified in the references of key articles, although again this was not a fully comprehensive search due to the aforementioned constraints.

The rationale behind the aims and content of the present review being so wide ranging was due to the limited number of relevant papers found after examining all abstracts. As such, all relevant papers relating to the key questions of the current review were included for critique.

3.2 Exclusion criteria

Studies were excluded that did not address or report a finding relating specifically to cannabis use and anxiety type problems. Papers pre 1970 were excluded from the review as were non-human studies or papers published in languages other than English that were not translated.

Despite the WHO (1997) report into cannabis commenting on how research pre 1997 will have been with a less potent type of cannabis and therefore not necessarily relevant to the effects of cannabis of post 1997 users; pre-1997 research was included in the study for the following reasons. Firstly, whilst pre-1997 research may have been conducted at a time with lower THC potency cannabis in general use, this does not mean that the results are not meaningful as THC content in cannabis varies from country to country (McLaren, Swift, Dillon & Allsop, 2008). As the research informing the present literature review is derived from research populations from around the world, excluding pre-1997 research would have been overly cautious. Secondly McLaren *et al.*'s review showed that post-1997 cannabis samples, whilst showing a general trend towards high THC levels in cannabis, still varied considerably by country in question.

3.3 Extraction template

An extraction template was used to organise the information gained from each paper reviewed (see Appendix B). This helped assess quality across studies along with guidance from Petticrew and Gilbody (2004).

4. Results

Analysis of the literature searches for the present review found a rather piecemeal picture of research into cannabis and anxiety. Nevertheless there are recognisable categories, ‘Cannabis and Panic’, ‘Cannabis and Subjective Experience’, ‘Cannabis and Social Anxiety’ and ‘Cannabis, Anxiety and Anxiety Disorders’. Without doubt some of the studies mentioned will overlap these categories; however the segregation of research into categories assists in the understanding of the myriad of findings. In addition, due to the obvious repetition that would occur if a detailed methodological critique was conducted on each study, a separate section on methodological issues is presented later. Appendix C provides a table with detailed summary of the studies included in the present paper.

4.1 Cannabis and Panic

4.1.1 Case reports

Panic attacks have been observed as an occasional side-effect of acute intoxication with cannabis; although this was mainly reported in naïve users (Earlywine, 2002). There are a number of case reports in the literature suggesting a link between cannabis use and panic attacks/disorder. These will be described in turn.

Strohle, Muller and Rupprecht (1998) reported on the case of a sixteen year old male, with no previous psychiatric history, who experienced his first panic attack during his second episode of marijuana intoxication. Despite discontinuing from any further use he continued to experience regular panic attacks and accompanied agoraphobia. Strohle *et al.* (1998) argued that marijuana use precipitated a panic disorder and that this may have been the case in other vulnerable individuals.

Deas, Gerding, and Hazy (2000) reported on a similar case of a fifteen year old male with oppositional defiant disorder who smoked marijuana weekly. After some time he began to experience acute anxiety and panic reactions whilst testing positive for cannabis in urine samples.

As in the previous case his symptoms persisted despite discontinuation of marijuana use. Again it was argued that marijuana precipitated the observed panic reactions.

Similar panic reactions to marijuana were reported by Langs, Fabisch, Fabisch and Zapotoczky (1997). Three cases were described where individuals of varying ages and gender experienced panic attacks during marijuana intoxication. In two of the three cases who had previous diagnoses of depression, panic attacks continued despite cessation of marijuana use. Once again it was postulated that cannabis use could trigger recurrent panic attacks in vulnerable individuals.

Adverse reactions to marijuana consumption were found in five cases presented by Ganz and Volkmar (1976). These five were all college students who came to the attention of psychiatric services. All five cases reported an anxiety reaction upon consuming cannabis, with three of the five having experienced a panic reaction. Some of the cases also reported extreme paranoia and depersonalisation whilst intoxicated. Four of the five cases reported a recurrence of anxiety reactions when intoxicated on other occasions. Interestingly, only one case continued to experience anxiety and panic after completely discontinuing use.

There are a number of pertinent caveats to be considered when looking at the above research. Firstly, there was little consideration of any poly-substance use in the cases described above, which could have independently triggered a panic reaction. Secondly, these were individuals who presented at treatment services and therefore the many cannabis users who do not experience panic reactions are obviously not reported. Thirdly, other potential psychosocial factors were not considered as a possible cause or mediating factor. For the authors to argue an aetiological link between marijuana use and panic attacks is rather bold at best. However, whilst case reports are arguably the least scientific form of evidence (Petticrew & Gilbody, 2004), they do provide some indication that in some, potentially vulnerable individuals, marijuana may precipitate both panic reactions and panic disorder.

4.1.2 Cross-sectional, co-morbidity and survey based research

Whilst there is a dearth of research into cannabis and its potential association with panic attacks/disorders, there are a number of interesting studies worth describing with increasing levels of methodological sophistication. These shall now be explored.

In 1988, Szuster *et al.* conducted a study looking at the relationship between marijuana smoking and panic anxiety with adult panic-disorder patients, depressed patients and non-disorder controls. Marijuana use was not significantly different across the three groups. Patients diagnosed with panic disorder (with and without agoraphobia) reported significantly more anxiety reactions to smoking marijuana than either depressed or non-patient controls. The majority of panic patients ceased use of marijuana due to experiencing anxiety reactions. This research supports the idea that marijuana use can exacerbate symptoms in people diagnosed with a panic disorder. However, the results found should be viewed tentatively as the sample size was rather small per group (<23); there was no matching of participants between groups; the contexts in which adverse reactions to marijuana occurred were not explored for alternative explanations and there was no control for poly-substance use.

Further support for a cannabis-induced panic reaction comes from Thomas (1996). He conducted a large community survey looking into the adverse effects of cannabis use in an adult New Zealand population. A sample of 528 adults aged between 18 and 35 (mean 27) took part with 199 confirmed cannabis users. Female cannabis users reported statistically significantly more panic attacks than male users. Ex-users reported significantly more panic attacks over the last week than current users. No significant differences were found with panic attacks for dependent versus non-dependent users. At first glance it seems that current cannabis use makes one less likely to experience panic attacks. However, Thomas (1996) argued that the ex-users reporting more panic attacks was probably due to those who experienced a panic attack as a result of cannabis consumption, discontinuing use in an attempt to avoid a recurrence of more attacks. The main

problem with this research is that without a non-using control group to compare prevalence, it does little to add useful data to the debate. In addition, there was no control for any poly-substance use in the sample. However, a significant gender bias in panic attack prevalence is worthy of note, although it's not clear if this bias is over and above the natural higher prevalence of panic attacks among women.

A secondary finding in Dannon, Lowengrub, Amiaz, Grunthaus and Kotler's (2003) work looking at treatment of panic disorder with and without prior cannabis use, is relevant to the present review. They found that 24 of the 66 patients with panic disorder in their sample had experienced their first panic attack within 48 hours of cannabis use and had gone on to develop panic disorder. Dannon *et al.* (2003) suggested that cannabis use in some vulnerable individuals caused panic attacks that may lead to panic disorder. Whilst the results of this study suggest a potential link between initial use of cannabis and the precipitation of panic disorder as a result, this must be viewed tentatively. As this was a co-morbidity sample the results cannot be extrapolated to the general population. Additionally there was no mention of individuals' histories and whether any poly-substance use played a part.

In more methodologically robust research, Zvolensky *et al.* (2006a) evaluated lifetime associations between cannabis use, abuse, dependence and panic attacks in a large cross-sectional survey based design. A total of 4745 randomly selected participants took part and data were collected through interviews conducted in participants' homes. After controlling for poly-substance use and demographic variables, lifetime history of cannabis dependence, but not use or abuse, (as defined by DSM-III diagnostic criteria) was significantly associated with an increased risk of panic attacks. In addition, the age of onset amongst those reporting a lifetime history of cannabis use and panic attacks was on average 8.6 years earlier than those individuals who had a lifetime history of panic attacks but no cannabis use.

These results suggest an association between dependant cannabis use and increased occurrence of panic attacks. Zvolensky *et al.* (2006a) argued that the relationship may have been due to dependent users being more likely to use cannabis to cope with panic-related discomfort following heavy cannabis use, and thus a cyclical relationship ensues. However due to the cross-sectional design it is impossible to untangle this relationship and infer any causality. In addition, one main problem with the study is that rather surprisingly tobacco use was not controlled for. This is especially surprising as Zvolensky, Felener, Leen-Felder and McLeish (2005) have reported significant associations between tobacco use and panic anxiety.

In a recent cross-sectional study Zvolensky (2006b) investigated whether anxiety sensitivity interacted with marijuana use in relation to the prediction of panic-relevant variables among young adult tobacco smokers. Anxiety sensitivity was defined as the fear of anxiety and anxiety-related sensations (McNally, 2002). Among tobacco smokers, marijuana users with high, but not low, anxiety sensitivity were at increased risk for anxiety symptoms and catastrophic interpretation of bodily events. This finding was observed after controlling for amount of tobacco use, negative affectivity and alcohol use. Zvolensky *et al.* (2006b) argued that anxiety sensitivity was a pertinent factor in better understanding the relation between marijuana use and panic-related processes in young adult cigarette smokers. Another interesting finding from this research suggested that the amount of marijuana consumed does not interact with anxiety sensitivity to increase the risk of catastrophic thinking. Therefore there does not appear to be a significant dose-dependent relationship. This research had a number of methodological strengths in that it controlled for relevant empirically-based panic related variables (tobacco, alcohol use and negative affectivity). However one significant drawback was the lack of control for other substance use and as with all other studies in this area the questionable reliability of self-report methods.

4.1.3 Summary

With the exception of the last two studies described, the majority of the research looking at associations between cannabis and panic was flawed methodologically. Any conclusions drawn from the research at present should be tentative at least. There does appear to be some kind of relationship between cannabis use and the occurrence of panic attacks, with the possibility that cannabis use could trigger a panic reaction in some individuals. It also appeared that heavy users were more likely to experience panic attacks earlier in life than non-users, and in greater frequency, and that those with higher levels of anxiety sensitivity were more vulnerable. There is of course the possibility that people who experience panic attacks self-medicate with cannabis to help cope with the aftermath of a distressing panic episode. However, as no prospective longitudinal research has, to the author's knowledge, been conducted in this area such causal associations remain uncertain.

4.2 Cannabis and Social Anxiety

Research pertaining to cannabis use and other anxiety disorders is extremely scarce. Nevertheless, what was discovered will be presented below.

Oyefeso (1991) examined the personality differences among 253 male undergraduate cannabis users categorised into five levels of use. Cannabis use was not related to trait anxiety, need for autonomy or self-esteem. Cannabis use was related to higher social anxiety where daily users reported higher social anxiety than other users. However, there was no control for other drug use in the sample and as heavy cannabis users have been shown often to use other substances, this could have significantly affected the results.

Similar results were found in a longitudinal genetic twin-based study conducted by Lynskey, Heath, Nelson, Bucholz, Madden, Slutske *et al.* (2002). Whilst the main purpose of the study was to look at genetic and environmental influences on cannabis dependence, a relationship with social anxiety was found. Social anxiety correlated with cannabis dependence and was found to be a risk

factor for cannabis dependence. However, the direction of this observed relationship cannot be ascertained by this study.

One recent study (Buckner *et al.*, 2006) attempted to explore the relationship found in earlier research between cannabis use and social anxiety, whilst controlling for alcohol use disorders. They examined potential moderators in this relationship and found that only perceived coping to unpredictable stimuli moderated the relationship between social anxiety disorder and cannabis use disorder. Buckner *et al.* (2006) argued the results were consistent with tension-reduction models of addiction in that perceived capacity to cope with stressors played an important role in adverse outcomes among anxious individuals. They also postulated that in the absence of cannabis as a coping strategy, those with dependence lack some of the coping skills that non-cannabis using individuals with social anxiety disorder may have employed. However the conclusions drawn by the authors are questionable due to two main methodological flaws, the lack of control for poly-substance use and the possibility of the acute effects of cannabis intoxication skewing the results.

4.2.1 Summary

It appears from the little available research examining the relationship between cannabis use and social anxiety that an association does appear to be evident. This seems to be more the case among heavy, dependent users than occasional users. It also appears that poor perceived coping moderates the relationship between social anxiety disorder and cannabis dependence, in that those socially anxious individuals with low perceived coping are more likely to display symptoms of cannabis dependence. This finding may be explained through the self-medication hypothesis.

4.3 Cannabis and subjective experience

A few studies have enquired into the perceived costs and benefits of cannabis use among different populations of users. What is presented below are the findings relevant to the current review.

Smart and Adalf (1982) conducted a large cross-sectional questionnaire survey into the adverse reactions of cannabis use across varying frequency of use among high school students in Ontario, Canada. Around 45% of all users and 60% of daily users reported adverse effects of anxiety and confusion. Twenty-five percent of all users and 56.5% of daily users reported recurrences of the experience. Predictors of anxiety and confusion were daily use and being female. The results of this study should be viewed with some caution as it was looking specifically for adverse reactions among users. Also the degree of anxiety experienced was not measured, and it was only the acute effects surveyed rather than any residual impact on anxiety levels.

The work of Reilly, Didcott, Swift and Hall (1998) challenged some of the previous study's findings. They investigated characteristics and patterns of cannabis and other drug use among a sample of 268 Australian long-term users using structured interviews. The most frequently cited reasons for using cannabis were to relieve tension and achieve relaxation (61%). The most commonly reported negative effects were anxiety, depression or paranoia (21%). The majority of users (72%) believed that the positive effects of cannabis use outweighed the negative. However this is unsurprising as generally speaking people do not continue with behaviours they find noxious and contain no positive reinforcers.

Hathaway's (2003) research findings are more in line with those of Reilly *et al.*'s (1998) study. One hundred and four experienced cannabis users residing in Toronto, Canada were interviewed regarding their reasons for use, among other questions. The results found that the most frequent reason for use was for enhancement of recreation and coping with stress and anxiety (95%). However around 50% of users reported the main negative effect of cannabis use was anxiety.

4.3.1 Summary

It appears from the studies described above that a rather dichotomous relationship exists among heavy long-term users of cannabis. On the one hand it appears that a significant number of users report anxiety as an adverse effect of using, yet at the same time report relaxation and tension reduction as a primary effect or reason for using. This paradox may be accounted for by considering the importance of the setting in which the substance is consumed. It may be that, as for other substances, cannabis merely enhances the current mood that exists within the consumer at any one point in time. On the other hand it could also be explained by biases in memory and the impact of this on self-report data. The results found above could also be explained through the varying concentrations of THC and CBD found in cannabis resulting in a bi-directional impact on anxiety.

4.4 Cannabis, Anxiety and Anxiety Disorders

The research concerning the effect of cannabis on non-acute anxiety and its potential associations with the prevalence and aetiology of anxiety disorders, paints a rather confusing picture. The stark inconsistencies of findings in this area are presented below.

4.4.1 Cross-sectional and qualitative studies

Evidence for an anxiolytic effect of cannabis use comes from the work of Sethi (1986). Part of their work took 50 male Indian chronic cannabis users and compared general anxiety to 50 male matched non-user controls using Taylor's Manifest Anxiety Scale. Participants were excluded if evidence of poly-drug use or previous physical or psychiatric history was present. The results found that the chronic cannabis users scored significantly lower on anxiety than the non-user controls. However the sample size in this study was relatively small and whilst the exclusion criteria may help extrapolate the findings to similar populations, at the same time users who may have developed an anxiety disorder from their use would not have been included.

Sethi's (1986) work is supported in some elements by the work of Stewart, Karp, Phil and Peterson (1997). Stewart *et al.*'s (1997) research investigated the relationship between anxiety sensitivity, drug use and reasons for drug use among university psychology students in Canada. Their results showed that marijuana and hashish users scored significantly lower in the anxiety sensitivity index than non-users. It was argued that marijuana may have an anti-anxiety effect, or that perhaps those with high anxiety sensitivity avoided marijuana for fear of its psychoactive effects. However a major methodological criticism of this study was that poly-drug use was not controlled for.

In other research, Tournier, Sorbara, Gindre, Swendsen, and Verdoux (2003) conducted a study investigating the association between cannabis use and anxiety in daily life in a non-clinical sample using the Experience Sampling Method (ESM). This method allows for multiple measures of anxiety to be recorded per day alongside any concurrent substance use. A total of 79 participants were categorised into high and low levels of cannabis use out of a total of 685 undergraduate students. The results found no significant association between level of anxiety (in either direction) and cannabis use in daily life. However a diagnosis of anxiety disorder was associated with an increased likelihood to use cannabis, independent of current levels of state anxiety, thus the association does not appear to be motivated by a self-medication to reduce current anxiety symptoms. There was also no association between panic disorder and cannabis use. However whilst a relatively well-designed study, there was no control for the influences of poly-drug use or environmental information during anxiety sampling times.

With contrasting results, Troisi, Pasini, Saracco, and Spalletta (1998) investigated psychiatric symptoms in 133 male Italian army draftee cannabis users who were free from cannabis and poly-substance use. All drug use was assessed through urine tests during routine medicals and participants were interviewed after 2-5 days of abstinence. The results found that cannabis dependence was associated with a higher prevalence of co-morbid psychiatric disorders, than either

abuse or use (DSM-III definitions). Significant differences were found in anxiety between users, abusers and dependent cannabis use. The severity of both state and trait anxiety increased with frequency of cannabis use, suggesting a dose-relevant effect. Although controlling for current poly-substance use, this study failed to consider the influence of prior poly-drug use on anxiety levels. Also these results are not likely to generalise to the greater cannabis-using population as only around 30-40% of cannabis users use the substance exclusively.

Similar dose-anxiety level associations were found in the qualitative cluster analysis work of Clough, d'Abbs, Cairney, Gray, Maruff, Parker and O'Reilly (2005). Clough *et al.* (2005) conducted a mixed design study looking into the mental health effects of cannabis use in indigenous communities in Australia. Interviews were conducted with 103 current cannabis users with a cluster analysis performed grouping symptoms into main categories. The results found that after controlling for age, gender and alcohol use, the 'anxiety-dependency' cluster was significantly positively associated with the number of 'cones' (a cannabis cigarette) smoked per week. There was an incremental effect observed whereby the more 'cones' smoked per week the more 'anxiety-dependency' symptoms observed.

Further dose-related associations were found in the work of Bonn-Miller, Zvolensky, Leen-Feldner, Feldner, and Yartz, (2005). They investigated the incremental validity of regular cannabis use and the relationship with anxiety and depression symptoms among young adult smokers residing in Vermont, USA. This research was useful in that it controlled for the effects of smoking tobacco upon anxiety states and therefore assisted any unique cannabis effect to come to light. The results found that after controlling for tobacco use, alcohol use and anxiety sensitivity, marijuana use and frequency of use were related to anxiety symptoms. Regular cannabis users reported more anxiety symptoms than both occasional and non-users, suggesting a dose dependent effect.

An inconsistency in the dose-related findings comes from the work of Degenhardt, Hall and Lynskey (2001). Their research took data from the Australian National Survey of Mental Health and Well being, with over 10,000 participants, and looked for associations between alcohol, cannabis and mental health problems in the general population. They found that whilst both tobacco and cannabis use were both associated with anxiety disorders, after controlling for confounding variables, cannabis use at any level (use, abuse or dependence) was not associated with anxiety or affective disorders. This is an interesting finding as tobacco use remained associated with anxiety disorders. As cannabis is often smoked with tobacco it seems surprising that tobacco use alone is associated. The main methodological caveat with this study was the fact that both substance use and mental health problems were only investigated for the period of the prior 12 months.

4.4.2 Summary

The results of cross-sectional studies in the present category are markedly inconsistent. It appears that whilst some studies have found that cannabis users experience less anxiety than non-users, in other studies no relationship was found at all. Also a number of other research studies have consistently found an incremental cannabis dose-related effect upon anxiety. This finding has been replicated across different populations and continents.

4.4.3 Longitudinal research

In a well-designed and robust study, Fergusson and Horwood (1997) examined data from a New Zealand birth cohort study of 1265 individuals born between 1972 and 1973 and followed for 18 years. They were investigating the relationship between early onset cannabis use and later psychosocial adjustment. The results did find an association between frequency of cannabis use and anxiety disorders, where those with an anxiety disorder were more likely to have used cannabis. However when other causal factors were taken into account no significant association was found. Ferguson and Horwood (1997) argued that the associations found between cannabis use and mental health arose due to those individuals choosing to use cannabis being part of a higher risk population for developing mental health disorders.

The work of Brook, Cohen and Brook (1998) supports the previous studies' findings. Their research took randomly selected children aged between one and ten years old and followed them for over 20 years, in order to examine temporal priority in the relationship between psychiatric disorders and drug use. The results found that at no point in time was there a reliable correlation between level of substance use and rates of anxiety disorder. There was no evidence that anxiety disorders in late adolescence have an influence on later drug use when controlling for earlier drug use.

Consistent with other longitudinal research in this area, McGee, Williams, Poulton and Moffitt (2000) found similar results to those already observed. This study examined cannabis use and mental health among other factors in over a thousand 15 year old New Zealand adolescents until they reached age 21. The cross-sectional results found that at age 15 cannabis use was significantly (four times) higher amongst those with anxiety/depressive disorders, when compared to those with no disorder. However longitudinal analysis found that at ages 18 and 21 cannabis use was not significantly higher amongst those with anxiety or depressive disorders. Cannabis use at age 15 did not predict anxiety or depressive disorders at age 18. Cannabis use at 18 also did not predict anxiety

or depressive disorder at age 21. The authors argued that the primary prediction direction of risk is from mental disorder to cannabis use, rather than the opposite. However they rejected the self-medication hypothesis and argued that the aforementioned finding is more a result of a differential drift towards substance use among adolescents who showed acting out behaviours which were non-conventional in the first place.

However an inconsistency in the studies in this area comes from the work of Patton, Coffey, Carlin, Degenhardt, Lynskey and Hall (2002). They conducted a study aiming to determine whether cannabis use in adolescence predisposes individuals to greater levels of anxiety and depression in young adulthood. This was a 'seven wave' cohort study following 1601 students aged 14-15 over six years into early adulthood. The results found that daily cannabis use in young adult women was associated with a five times increase in the odds of depression and anxiety after adjustment for concurrent substance use. However, in young adult men there was no significant increase. Weekly or more frequent (one to four times a week) cannabis use in female adolescents predicted an approximately twofold increase for depression and anxiety in early adulthood, after adjustment for concurrent substance use. Again, no significant increase for male adolescents was observed. In addition it was found that adolescent depression and anxiety did not predict later daily or weekly cannabis use in young adulthood in either gender. Thus the self-medication hypothesis was not supported.

Patton *et al.* (2002) postulate that the observed increase may be due to the psychosocial consequences of frequent cannabis use, such as educational failure, unemployment, crime and drop-out. They also argued that due to the largest increases being found in the daily users, there may also be a direct pharmacological effect of cannabis on cannabinoid receptors which are found in great abundance in areas of the brain which regulate mood. However such explanations may not account for the significant gender-specific effect observed.

4.4.4 Summary

With the exception of Patton *et al.*'s (2002) work, the majority of longitudinal studies fail to find associations between cannabis use and anxiety disorders. Where an association is found this is rendered non-significant once other pertinent environmental and social factors are accounted for. Therefore the longitudinal evidence, arguably the most valid and robust form of aetiological evidence, shows no consistent reliable association between cannabis use and anxiety disorders.

5. Methodological limitations in the research

Research within the field of substance use and abuse is fraught with methodological difficulties (Day & Robles, 1989). Cannabis research is by no means an exception to the rule, in fact it has arguably even more methodological difficulties than other substance-related research, due mainly (although not entirely) to the problem that it is often mixed with other substances (such as tobacco) when consumed. What follows is a detailed exploration of the methodological caveats that pertain to this area of research. Where certain studies have controlled for these difficulties they will be duly mentioned.

Firstly, none of the studies reviewed appear to have considered or controlled for the effects of maternal cannabis use on the individual participants' mental health. Whilst some studies (e.g. Brook *et al.*, 1998; Ferguson *et al.*, 1997) have considered parental drug use as a demographic variable for association, it does not seem to have been included as a potentially confounding variable in those participants with anxiety problems. Some studies have found a predisposition towards both depression and anxiety in people whose mothers used cannabis whilst pregnant (Leech, Larkby, Day & Day, 2006; Grey, Day, Leech & Richardson 2005). It is possible therefore those associations found in samples between cannabis use and anxiety may be the result of maternal exposure to cannabis, and may have impacted on the results of the majority of research in this area.

Secondly, another confounding effect that links with the above is that maternal cannabis use has been associated with children's cannabis use (Ferguson *et al.*, 1997) and can even predict offspring cannabis use, with those children exposed to cannabis in-utero being more likely to use cannabis themselves in later life (Day *et al.*, 2006). Therefore, those mothers whose children were exposed to cannabis during pregnancy and may have a predisposition to anxiety/depression are also more likely to engage in cannabis use in later life. Thus some of the cannabis users recruited for research may be a sub-group of people who firstly may be more likely to smoke cannabis due to their mother's use and also therefore more likely to be predisposed to an anxiety-based disorder in later life (than

non-cannabis users). This relationship could therefore lead to spurious associations in the literature between current cannabis users and anxiety problems.

Thirdly, the extremely varied use of measures in this area of research makes both comparisons between studies and conclusions based upon their findings difficult. When measuring cannabis use some studies have looked at use overall (e.g. Degenhardt *et al.*, 2001) others have spilt usage into various incremental categories (e.g. Ferguson *et al.*, 1997; Patton *et al.*, 2002) whilst others (e.g. Buckner *et al.*, 2006; Troisi *et al.*, 1998) have classified use into ‘use disorders’ as outlined in diagnostic manuals such as DSM-IV. It would seem important to split users into both frequency of use and dose categories as any relationships found may be dose and frequency related.

In addition, the methods for measuring anxiety and anxiety disorders have varied somewhat across this research area. Some studies have classified anxiety into DSM-III and IV disorders (e.g. Tournier *et al.*, 2003, Degenhardt *et al.*, 2001) which are not particularly useful as they tell us nothing about quantity or frequency of use. Others have used a variety of trait and state anxiety measures (e.g. Oyefeso, 1991; Troisi *et al.*, 1998) whilst others have looked at anxiety sensitivity (e.g. Bonn-Miller *et al.*, 2005; Zvolensky *et al.*, 2006a). It is likely that some of the discrepant findings in the literature may reflect these differences in measurement of both cannabis and anxiety.

Fourthly, some studies (e.g. Buckner *et al.*, 2006) did not adequately control for the acute effects of anxiety verses the non-acute effects when administering anxiety tests to the various categories of users and non-users. The main psychoactive ingredient in cannabis is THC which can remain in the plasma for six days following consumption and may remain in the fatty tissues in the brain for up to 30 days, due to its fat-soluble nature. Therefore any associations found may have been the result of residual anxiety present from acute intoxication or cannabis still being present in the system, rather than the individual’s ‘normal’ levels of anxiety. However, Tournier *et al.*’s (2003) findings may challenge this idea, and this may not be a problem for much of the panic research looking at the acute effects of cannabis upon anxiety.

Fifthly, a lot of studies have not controlled for poly-substance (both licit and illicit) use among cannabis users and the possible contamination effects that this could create in the data. This is quite surprising considering the wealth of literature concerning tobacco (Isensee, Wittchen, Stien, Hofler & Leib, 2003; Zvolensky *et al.*, 2005), alcohol (McGee *et al.*, 2000; Kushner *et al.*, 2000), cocaine (Paine, Jackman & Olmstead 2002) and ecstasy (Milani, Parrott, & Turner, 2004) all having associations with both acute and residual anxiety. Whilst some researchers did control for some poly-substance use (Clough *et al.*, 2005; Bonn-Miller, 2005) very few (e.g. Patton *et al.*, 2002) controlled for a comprehensive range of substances. This general lack of methodological rigour may put in question the results of the majority of research in the present area.

The issue of both type and strength of cannabis consumed by users has not been taken into account by any of the research reviewed. Both Smart and Adalf (1982) and the present author believe that this variable could have an impact on anxiety responses among cannabis users. As some authors (King, Carpenter & Griffiths 2005) have argued that the strength of cannabis has increased over the years, differences observed between conclusions of older studies and more recent ones may be due to potency (Langs *et al.*, 1997; McKim, 1997). As THC administered alone appears to cause anxious reactions (Zuardi *et al.*, 1982) it would follow that users consuming preparations high in THC may be more likely to experience anxiety and/or anxiety disorders.

Most studies have not controlled for the effects of tobacco on anxiety. As Reilly *et al.*'s (1998) study found that 48% of long-term cannabis users smoked cannabis mixed with tobacco it would seem pertinent to recognise this (it should be mentioned that these data were from Australian users and therefore may not extrapolate to other cultures). Isensee *et al.* (2003) and Zvolensky *et al.* (2005) have found a relationship between tobacco use alone and anxiety. The results observed in studies finding a link between cannabis consumption and anxiety may be attributing the effect spuriously to cannabis rather than tobacco. Also the vast majority of authors do not consider the interaction effects of the hundreds of chemicals in tobacco and over 600 separate chemicals present

in cannabis. Therefore it would seem pertinent to consider different methods of consumption when looking at cannabis users (e.g., smoking with tobacco, without tobacco, eating/baking). A desirable study would separate cannabis users in terms of method of consumption.

All but one study (Troisi *et al.*, 1998) used only self-report of both cannabis and other drug use in their research. There are obvious difficulties in accepting the reliability of self-report information as many factors can affect the accuracy of information, especially with regards to illicit drug use (e.g. exaggeration of use, minimisation of use in dependent users). Some research used telephone interviews to gather usage data (e.g. Patton *et al.*, 2002) or had interviews take place in participants' homes (Zvolensky *et al.*, 2006b), which may have influenced the results as many of the participants lived with parents. Zvolensky *et al.* (2006a) argued that any self-report from cannabis users may be more unreliable than non-users due to potential cannabis-related recall biases and memory distortions.

Also measures used to assess anxiety relied heavily on self-report. Thomas (1996) postulated that drug users may understate the problems of drug use in order to justify continued use and this may have affected the results in studies including dependent users.

There is undoubtedly some publication bias in this area as with many areas (Petticrew *et al.*, 2004), with the tendency for positive relationships to be published along with journal editors actively discouraging the submission of negative research. Unfortunately the possibility of a funnel plot exercise (see Petticrew *et al.*, 2004 for further information) to assess bias in this area was not within the scope of the present review.

Cultural homogeneity in published research in the present area of inquiry is also worthy of note. The majority of research has been conducted in Westernised societies which makes extrapolating findings to other cultures difficult. In addition, the media in western societies is amok with warnings about the dangers of illicit drugs and users of cannabis may have been influenced by this in terms of drug effect expectancies.

6. Discussion of findings

6.1 Does cannabis use cause an increase or decrease in anxiety symptoms among users?

In terms of the research reviewed in the present paper it seems that cannabis can both increase and decrease anxiety in users. Data from long-term users suggests that users experience more anti-anxiety effects than increased anxiety effects (e.g. Sethi *et al.*, 1986; Stewart *et al.*, 1997). Other research suggests there is neither an increase nor a decrease in anxiety (Tournier *et al.*, 2003). Other data suggest that anxiety is a common side-effect of cannabis consumption (Smart & Adalf, 1982).

6.2 Do people who experience anxiety and anxiety type disorders use cannabis to self-medicate to relieve anxiety symptoms?

Some researchers have argued that with regards to social anxiety (Buckner *et al.*, 2006) and panic anxiety (Zolvensky *et al.*, 2006a) cannabis may be used for purposes of self medication. However, others (McGee *et al.*, 2000; Patton *et al.*, 2002) argued against a self-medication explanation for observed research findings. Instead they either argued for a causal effect of cannabis upon anxiety disorders, or that findings are better explained by shared environmental risk factors that increase both risk of cannabis use and anxiety disorders (Fergusson & Horwood, 1997).

6.3 Does cannabis use trigger anxiety disorders in vulnerable people?

Much of the research into cannabis and panic attacks and panic disorders does point towards the potential for cannabis to trigger panic reactions that can lead to a panic disorder (e.g. Dannon *et al.*, 2004; Szuster *et al.*, 1988). Many instances of the onset of panic disorder in the research seemed to have occurred in the presence of cannabis intoxication. Whilst a pure causal relationship is yet to be established, the weight of evidence seems to suggest a pertinent role for cannabis in vulnerable individuals, with more recent research suggesting a frequency of use effect (Zvolensky *et al.*,

2006a). However such a conclusion should be viewed tentatively due to the mass of methodological caveats in this area.

In contrast when we turn to social anxiety the opposite relationship seems more likely, wherein those individuals with social anxiety seem more at risk of cannabis use as a coping mechanism.

6.4 If cannabis does have an anxiogenic effect, is there a dose-related relationship?

Within the research that found cannabis users to have higher levels of anxiety than non-users, there does appear to be an incremental dose effect (e.g. Troisi *et al.*, 1998; Bonn-Miller *et al.*, 2005). While more research is needed in this area, the available evidence does suggest a dose-related effect upon anxiety.

6.5 Is there an association between cannabis use and anxiety disorders, if so what is the direction of the association and what can explain such association?

Whilst some studies have shown an association between cannabis use and anxiety disorders (e.g. Patton *et al.*, 2002; Troisi *et al.*, 1998), the weight of the more robust longitudinal research indicates a lack of association. Once other potential mediating environmental and social factors are entered into the analysis, any association seems to be non-significant (e.g. McGee *et al.*, 2000; Ferguson *et al.*, 1997). Whilst this area could benefit from more longitudinal studies, the current state of research suggests a lack of association. However these findings seem in direct opposition to the research exploring the association between cannabis use and panic disorder and other cross-sectional research.

7. Implications for Clinical Psychology

Clinicians working in the area of substance misuse may not currently be aware of the impact of cannabis upon anxiety states. It may be pertinent for clinicians to educate cannabis users of the possible impact of cannabis on anxiety and its potential role in the development of anxiety disorders. Also when treating individuals for anxiety disorders it may be pertinent to assess the impact of any current cannabis use upon the success of such treatment.

As the research looking into the relationships between cannabis dependence and panic attacks has found significant associations (Zvolensky *et al.*, 2006) it may be possible in the future to use this information to develop targeted prevention and treatment programmes for high-risk people. There may be a role for clinical psychology to disseminate the potential risks of cannabis use to young adults, before the likely age at which cannabis is first experimented with.

8. Conclusions and Future Research

The present review has attempted to explore the relationship between cannabis use and anxiety. The conclusions stated in the previous discussion section need not be repeated here. However what needs to be said is that this area of research is lacking somewhat in both quantity and quality of available studies. Therefore, any conclusions reached thus far should be viewed with caution.

Future research in this area would benefit from controlling for, as best possible, the numerous methodological caveats outlined earlier. Most of the longitudinal and cross-sectional research has been conducted on adolescents or young adults. This may seem pertinent in that those populations are the most prevalent users of cannabis. However it would be useful for future research to examine the relationship between cannabis use and anxiety across the age range.

More research needs to consider and investigate the possibility of common or co-related genetic predispositions to use cannabis or develop an anxiety disorder to explain the co-morbidity between cannabis use and anxiety.

From the literature reviewed there does occasionally appear to be a gender bias in the data, with females seeming more adversely effected by cannabis-induced anxiety. The potential explanations for this warrant further research, and a relevant piece of research could examine the hypothesis that due to differences in size of the parts of the brain that regulate emotion (such as the hippocampus) between the genders, the effects of acute intoxication upon the cannabinoid receptors in this area may be a causal factor.

Lastly with regards to cannabis and the observed association with panic attacks, future research could investigate whether different potencies of the drug have a mediating effect. It may be that cannabis high in THC (shown to increase physiological anxiety) is more likely to cause a panic reaction due to the catastrophic interpretation of anxiety responses in some users. It would also be

interesting to see what role, if any, paranoia plays in this relationship. In addition the environmental context in which cannabis use and panic reaction occur would be worthy of exploration.

To conclude, this area is lacking in the breadth and depth of research needed to draw firm conclusions from the available data. However, there are a number of interesting, further research possibilities that should excite and inspire future investigation.

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Note – presence of asterisk ‘*’ indicates reference was a main focus of the present review.

Section C

**EXPLORING THE RELATIONSHIP BETWEEN
CANNABIS AND PANIC**

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1. Abstract

Introduction: Previous research has indicated potential associations between cannabis use and panic attacks. Current theories propose both chemical and psychological factors in associations observed.

Objectives: In the first known study in Britain, the present study aimed to explore the relationship between cannabis use and panic attacks. A secondary aim explored the effect of different types of cannabis.

Method: Inspired somewhat on established research (e.g. Zolvensky, Bernstein, Sachs-Ericsson, Schmidt, Buckner, & Bonn-Miller, 2006a) a cross-sectional study was undertaken to explore the potential relationship between cannabis and anxiety. A self-selecting opportunity sample of 306 students draw from both of Leicester's universities completed a battery of questionnaires concerning cannabis use, tobacco use, panic attack history, alcohol use, poly-substance use and various psychometrics including panic disorder (the PDSR: Newman, Holmes, Zuellig, Kachin, & Behar, 2006); locus of control (the MDLC: Levenson, 1972); anxiety sensitivity (the ASI: Reiss, Peterson, Gursky, & McNally, 1986) and catastrophic cognitions (the CCQ: Khawaja, Oei, & Baglioni, 1992).

Results: Significant levels of both cannabis use and panic attack history were reported among the sample. Survival analysis revealed cannabis users were of significant increased risk (OR 2.01) of experiencing a panic attack compared to non-users. Mann-Whitney analysis found cannabis users who use mainly high potency 'sensimillia' experienced significantly more lifetime panic attacks than those who used other types.

Conclusions: Whilst further research is needed the results of this study suggest cannabis a risk factor on panic attacks. The consumption of higher potency 'sensimillia' represents a higher risk

than other types of cannabis. Education for professionals and screening for cannabis use in panic disorder is recommended.

2. Introduction

It is estimated that cannabis is consumed by between 200 and 300 million people worldwide (Woody & MacFadden, 1995). It is the most commonly used recreational drug in the developed world including the UK, USA, Australia and New Zealand (WHO, 1997; NSDUH, 2005; EMCDDA, 2005). Across the western world a majority of young people use Cannabis regularly (Fergusson & Horwood, 2005) and there are indications that its use is rising (Patton, Coffey, Carlin, Degenhardt, Lynskey, & Hall, 2002). Research into prevalence among university students in the UK indicates that 60% had some experience with cannabis and 20% were engaging in regular use (Webb, Ashton, Kelly, & Kamali, 1996).

The use of Cannabis and its association with psychosis has secured much research attention over the last ten years (see Moore, Zammit, Lingford-Hughes, Barnes, Jones, and Burke, 2005 for a contemporary review). However, potential associations between Cannabis and affective states have received considerably less interest (Degenhardt *et al.*, 2002). Whilst potential associations with depression have received some interest (see Degenhardt *et al.*, 2002 for a review) there has been a paucity of research investigating the association between cannabis and anxiety disorders (Buckner Schmidt, Bobadilla, & Taylor, 2006).

A panic attack is a very distressing and debilitating experience. It is a unique fear state as the person often believes catastrophic consequences are about to occur (Clark, 1986). Prevalence studies have shown that between 9.5% (Wittchen, Nocon, Beesdo, Pine, Höfler, & Lieb, 2008) and 4.3% (Norton, Zvolensky, Bonn-Miller, Cox & Norton, 2008) of people experience panic attacks of sufficient severity to meet diagnostic criteria (DSM-IV & ICD-10). Panic disorder represents a more severe condition where an individual suffers from multiple panic attacks over a period of time and such attacks impair normal functioning and mental wellbeing. Research has indicated lifetime prevalence of between 2% and 4% (Wittchen *et al.*, 2008; Katerndahl & Realini, 1993; Kessler McGonagle, Zhao, Nelson, Hughes, Eshleman, Wittchen *et al.*, 1994; Regier, Rae, Narrow, Kaelber,

& Schatzberg, 1998) of panic disorder in the general population, with females often being at least twice as likely to suffer. The impact of a diagnosis of panic disorder is associated with significant suffering and disability in social, physical, and occupational domains (Leon, Portera, & Weissman, 1995). As such any steps taken to prevent the onset of either panic attacks or panic disorder would seem a sensible aim in reducing suffering.

2.1 Cannabis and anxiety

There are over 400 chemical compounds contained within cannabis of which 66 are unique to the plant and are labelled ‘cannabinoids’ (Earleywine, 2005). The major psychoactive ingredient in cannabis is Delta-9 Tetrahydrocannabinol (THC) however; Delta-8 THC, cannabitol and cannabidiol (CBD) are also commonly found. Brain regions such as the hippocampus, cortex and amygdala which are directly involved in the regulation of mood and emotional behaviour, have been found to have high concentrations of CB1 receptors (Witkin Tzavara & Nomikos 2005).

Over the last fifteen years an increasing amount of research has examined the effect of cannabinoids in animal samples. Much of the data from research with animals provides evidence of dose-dependent bidirectional modulation of anxiety by the cannabinoid system in that both anxiogenic and anxiolytic effects have been observed (Viveros, Marco & File 2005).

With regards to human studies, a series of experiments have shown that administration of high doses of THC produce a neurological syndrome (abnormal movement and hyperreflexia) in nearly all subjects (Tassinari, Ambrosetto, Peraita-Adrado & Gastaut, 1999). Indeed, more recent research has shown THC to have a greater effect on euphoria and anxiety symptoms (Williamson & Evans, 2000). Zuardi, Shirakawa, Finkelfarb and Karniol (1982) research has demonstrated that THC given in a pure form has a marked anxiogenic and psychotic effect on healthy human subjects. D’Souza, Perry, MacDougall, Ammerman, Cooper, Wu, *et al.* (2004) found similar effects. Paradoxically, cannabidiol, another important cannabinoid, has been found to reduce or block the anxiety reaction caused by THC (Karniol, 1974; Carlini & Santos, 1970) and anxiety in clinically anxious

outpatients (Fabre & McLendon, 1981). Indeed there is recent evidence of CBD acting as a general anxiolytic and anti-psychotic (Zuardi *et al.*, 2006), as well as a neuroprotective (Hermann, Sartorious, Welzel, Walter, Skopp, Ende, *et al.*, 2007).

Higher potency cannabis (sensimillia or skunk) has been available for over ten years in the UK and potentially longer in countries such as Holland where cultivation and use is de-criminalised. Despite this, it has taken the academic community some time to recognise that this new ‘high potency cannabis’ may be qualitatively different to cannabis resin or traditional herbal cannabis or ‘weed’. In fact the changing potency of cannabis has been largely ignored by current research (Smith, 2005). Only two very recent studies, known to the present author have considered the different types of cannabis in their research (Di Forti, Morgan, Dazzan, Pariante, Modelli, Marques, *et al.*, 2009; Morgan & Curran, 2008)

Cannabis sinsemilla or ‘skunk’ (a name derived from the smell being likened to the animal) has much higher levels of THC than cannabis resin, which has higher levels of cannabinalol, and traditional herbal cannabis (Hardwick & King, 2005; BBC, 2008; Potter *et al.*, 2008). Before the year 2000 the most commonly occurring cannabis in the UK was cannabis resin, which constituted about 70% of the samples seized by police. By 2005 cannabis resin accounted for only 16% of the market with sinsemillia making up 81% (Hardwick & King, 2005). As such THC levels in available cannabis have been rising over recent years (Smith, 2005) whilst CBD levels have been remaining relatively consistent. Harwick and King (2008) found that UK samples of cannabis sinsemilla had very high concentrations of THC (median 15%) and contrastingly low concentrations of CBD (0.1%). Cannabis resin and traditional herbal cannabis had much lower concentrations of THC (median 5%) and higher levels of CBD (mean 3.5%).

Therefore, the cannabis skunk used today has much higher levels of THC and thus logically could precipitate stronger anxiety and panic reactions among users. Thus the issue of THC potency among users of cannabis is most worthy of investigation.

2.2 Cannabis and Panic research

From around the 1970's small-scale research and case studies began to emerge linking the use of cannabis with panic attacks (Gale & Guenther, 1971). Indeed panic reactions to marijuana consumption were found in five cases presented by Ganz and Volkmar (1976). Such case reports continued through the 1990s (Langs, Fabisch, Fabish & Zapotoczky 1997) and beyond (Deas, Gerding & Hazy, 2000) with authors suggesting a panic vulnerability model whereby certain individuals may react negatively to cannabis consumption with consequences of extreme anxiety and panic.

Surveys of cannabis users have shown that over a third of habitual cannabis smokers report anxiety reactions (including panic) along with potentially anxiogenic effects such as paranoia (Negrete, 1974). Further survey research into cannabis users' experiences discovered that common negative reactions to cannabis include anxiety and panic, with approx 22% of cannabis users reporting having experienced a panic attack related to such use (Thomas, 1996). Female users reported significantly more panic attacks than males and this has also been found elsewhere (Zvolensky, Berstein, Sachs-Ericsson, Schmidt, Buckner & Bonn-Miller, 2006a). Hathaway (2003) surveyed experienced and long-term cannabis users and found that 40% of weekly users reported having experienced at least one panic attack as a result of such use. This is significantly elevated considering the prevalence rates in the aforementioned general population, although it should be noted that strict panic attack diagnostic criteria were not employed in the above studies.

Other research suggesting a cannabis-panic link comes from the work of Dannon, Lowengrub, Amaiz, Grunhaus and Kotler (2003). They discovered that among 66 patients with panic disorder in their sample, 24 (36%) had experienced their first panic attack within 24 hours of using cannabis and had subsequently gone on to develop panic disorder. Szuster, Ponitus and Campos (1988) also found that panic disorder patients had negative cannabis use histories and experienced more anxiety reactions to cannabis use than patients with other diagnoses. These findings support the assertion by

Earleywine (2002) that acute intoxication with Cannabis can lead to panic reactions but mainly among naïve users.

More methodologically robust research in this area began with Zvolensky *et al.* (2006a). Lifetime associations between cannabis use, abuse, dependence and panic attacks were investigated in a large cross-sectional survey-based design. After controlling for poly-substance use (but not tobacco use) and demographic variables, among a representative sample of 4745 participants, lifetime history of cannabis dependence, but not use or abuse, (as defined by DSM-III diagnostic criteria) was significantly associated with an increased risk of panic attacks. Whilst this is an interesting finding, the nature of the direction of this use is so far unknown from the current empirical literature (Zvolensky, Lewinsohn, Bernstein, Schmidt, Buckner & Seeley, 2008).

However the putative cannabis-panic link is further complicated by research into tobacco smoking and panic. Prospective longitudinal research has shown a marked increased risk of future panic attacks in adults among regular adolescent smokers (Isensee, Wittchen, Stein, Hofler & Leib 2003), although steps taken to account for reverse or reciprocal causality are not clear. Indeed this was also the case for panic disorder (Isensee *et al.*, 2003; Johnson, Cohen, Pine, Klein, Kasen & Brook, 2000). Laboratory studies have also shown that smokers are more prone to anxious responding to bodily sensations than non-smokers (Zvolensky, Leen-Feldner, Feldner, Bonn-Miller, Lejuez, Kahler *et al.*, 2004). Nicotine consumption causes neurotransmitter release resulting in increased heart rate and blood pressure which can be interpreted as anxiety (Dilsaver, 1987) These finding is further complicated by the findings that cannabis is the most frequently used illicit substance among cigarette smokers (Smart & Ogbourne, 2000) and due to the other problem that cannabis is often consumed mixed with tobacco, which can enhances the psychoactive effects (Earlywine, 2002), isolating the causal elements of both substances in terms of their potential to induce panic attacks is a significant methodological challenge for current and future research. That said, some limited research has looked at Cannabis use among tobacco smokers and found that the

cannabis use incrementally predicts anxiety symptoms (Bonn-Miller, Zvolensky, Leen-Feldner, Feldner & Yartz 2005).

2.3 Cannabis and panic theory

As explored in detail above there is certainly a potential physiological explanation for why cannabis intoxication could induce panic attacks. This is even more the case due to the increasing potency of THC in modern cannabis varieties. However there are other psychological effects of cannabis which could contribute to increased levels of anxiety and potentially panic. Bonn-Miller *et al.* (2005) and Thomas (1996) found that regular cannabis users experience more symptoms of anxious arousal, dizziness and somatic tension than non-users. Dannon *et al.* (2004) and Troisi *et al.* (1998) have observed such differences also, in addition to reports of cognitive dyscontrol such as depersonalisation and paranoia (Brook, Cohen & Brook 2001). These effects can arguably, to a lesser or greater extent, be risk factors with regard to panic attack vulnerability (Zvolensky *et al.*, 2008).

Other authors have suggested that the potential cannabis-panic link may be due to severity of use. For example Zvolensky, Schmidt and McCreary (2003) have argued that severity of smoking patterns may modulate panic processes. Indeed much research has indicated that heavy users of cannabis report higher overall levels of anxiety symptoms and psychopathology compared to non-users (Bonn-Miller *et al.*, 2005; Thomas, 1996). Zvolensky, Bonn-Miller, Bernstein, McLeish, Feldner and Leen-Feldner (2006b) have also argued that repeated affect-relevant learning with aversive interoceptive cues may be a key risk mechanism for frequent cannabis users. It may be that cannabis withdrawal symptoms, an increasingly recognised phenomenon, play a role in panic vulnerability as users experience more concentrated interoceptive sensations and have an opportunity to misconstrue them as potentially dangerous; thus increasing risk of a panic attack (Zvolensky *et al.*, 2006b). Bonn Miller *et al.* (2005) add to this thread by suggesting that during intoxication, cannabis users may learn that certain interoceptive events are beyond their control and

cannot employ cognitive resources to modulate affect regulation. As such they may well feel ‘out of control’ and this can lead to anxiety symptoms and in some cases, panic (Zvolensky, Feldner, Eifert & Brown, 2001).

Zvolensky *et al.* (2006b) have argued that in the current research literature there is a lack of studies that firstly look at the individual differences in psychological panic vulnerability factors among cannabis users and secondly that look at anxiety factors beyond the effects of cigarette smoking. They argued that anxiety sensitivity (fear of fear and anxiety-related sensations) could theoretically predispose people to panic attacks; alongside individual’s tendency to appraise bodily sensations as threatening. Zvolensky *et al.*’s (2006) research investigated how these constructs varied among tobacco smokers who used cannabis or not. The results found that the cannabis users compared to non-users, who had high anxiety sensitivity, were at increased risk of both anxiety symptoms and catastrophic cognitions. These findings go some way to uncovering the psychological differences that may predispose some individuals to panic pathology.

3. Rationale and Hypotheses

3.1 Rationale

Whilst research into the links between cannabis and panic pathology has been enlightening, there have been a number of potential problems with it. Firstly much of the previous work has both not distinguished between anxiety and panic attacks in a systematic fashion, or used a consistent diagnostic framework to ascertain if reported panic attacks or panic disorder are valid (e.g. Thomas, 1996; Hathaway, 2003; Szuster *et al.*, 1988).

Secondly, much previous research has not adequately controlled for potential confounders in the form of poly-substance use or problematic alcohol use. Only few studies have considered the effects of tobacco smoking alongside cannabis use and their potential individual relation to panic.

Thirdly all the current research into cannabis and panic has taken place in the USA, Australia and New Zealand. There has been no research conducted in a UK population.

Fourthly, virtually no research has considered individual psychological differences in panic vulnerability among cannabis users who experience panic attacks.

Finally, and perhaps most importantly, no study (to the present author's knowledge) has considered the differing effects of the main three types of cannabis (skunk, resin, herbal) upon panic attack prevalence.

The way in which THC increases anxiety in humans may be primarily physiological, but the role of cognitive processes and underlying belief systems may indeed serve to modulate this anxiogenic reaction. The present author suggests that locus of control may play a part in how cannabis users react to the feelings of anxiety and the amount of catastrophic interpretations of bodily events they experience. It is proposed that those cannabis users with high, internal locus of control will be more likely to experience panic attacks due to finding the feeling of being out of control more aversive.

Therefore the present study will use DSM-IV diagnostic criteria and diagnostic psychometrics to gain increased accuracy of the occurrence of both panic attacks and panic disorder. Potential confounders in the form of poly-substance use and alcohol misuse will be appropriately considered. Participants will be asked about both their tobacco and cannabis use history in detail, including the types of cannabis consumed. Three groups (cannabis users, tobacco users, neither users) will be initially compared in terms of the occurrence (or not) of panic and then total frequency. Secondary analysis will consider the differences among the cannabis group split into the differing types of cannabis. Last, differences in psychological panic vulnerability factors will be investigated across the main three aforementioned groups.

3.2 Hypotheses

The sample obtain for the present study did not recruit enough tobacco only smokers to constitute a meaningful size group. As such hypotheses were altered to reflect the fact that comparison across three groups was not possible. Therefore the comparison is now between cannabis users and non-cannabis users for the majority of hypotheses.

Hypothesis one is one-tailed and states that ‘Cannabis users will be significantly more likely to have ever experienced a panic attack than non-users’.

Hypothesis two is one-tailed and states ‘Among those who have experienced at least one panic attack cannabis users will have experienced a significantly higher amount of lifetime panic attacks compared to non-users’.

Hypothesis three is one-tailed and stated ‘Cannabis users are significantly more likely to meet a diagnosis of panic disorder than non-users’.

Hypothesis four is one-tailed and states that ‘Among cannabis users, those who predominantly used sinsemilla (skunk) would be significantly more likely to have ever experienced a panic attack compared to those who primarily used herbal cannabis or resin’.

Hypothesis five is one-tailed and states that ‘Among cannabis users, who had experienced at least one panic attack, those who predominantly used sinsemilla (skunk) would experience significantly more total lifetime panic attacks compared to those who primarily used herbal cannabis or resin’.

Hypothesis six states that ‘Among those who have experienced at least one panic attack cannabis users will have significantly higher mean locus of control scores than non-cannabis users’.

Hypothesis seven states that ‘Among those who have experienced at least one panic attack cannabis users will have significantly higher mean anxiety sensitivity scores than non-cannabis users’.

Hypothesis eight states that ‘Among those who have experienced at least one panic attack cannabis users will have significantly higher mean catastrophic cognitions scores than non-cannabis users’.

4. Method

4.1 Design

The present cross-sectional study utilised an independent groups design to explore the primary hypothesis that cannabis use (IV) would be associated with a significantly higher probability of experiencing a panic attack (DV). Independent groups and association analyses were also utilised to explore the various secondary hypothesis listed above.

4.2 Participants

4.2.1 Power analysis

Because of the large number of IVs utilised in the present study, it was recognised that the number of participants in each cell for between-groups analyses would vary considerably depending on the statistical test utilised. Therefore sample size calculation is based on the original primary hypothesis. In order to detect a difference in lifetime panic attacks between 22% in the cannabis group (Thomas, 1996) and 7.7% in the smokers group (Johnson, 2000) with 80% power and alpha set at 5% one-sided error rate, and assuming a 2:1:2 ratio in the respective proportion of students in the groups (cannabis, tobacco, neither) required a total of 354 completed questionnaires.

4.2.2 Inclusion criteria

Inclusion criteria required all participants to:

- be at least 18years old
- a student at one of Leicester's universities

Inclusion criteria to be treated as a cannabis user:

- a single use (or more) of cannabis in a lifetime

Inclusion criteria to be treated as a smoker:

- Smoking greater than, or equal to, one ten pack of cigarettes per day currently, or for any 6 month period in the past.

Responses to specific items on the questionnaires provided a means of screening participants against inclusion/exclusion criteria.

4.2.3 Exclusion criteria

- A current alcohol dependency or history of alcohol abuse, due to the findings of Kushner, Abrams, Thuras & Hanson (2000) that showed alcohol abuse is associated with an increased risk of panic attacks.
- Significant poly-substance use (5+ uses of other substances) due to the potential confounding effects of other substance use on anxiety (Lejuez, Paulson, Daughters, Bornovalova & Zvolensky, 2006) and to be in line with other studies (Zvolensky *et al.*, 2006a)
- Under 18 years of age.

4.2.4 Sampling

The present study employed a quasi-opportunity based self-selected sampling strategy. This was far from the ideal; however such methods of recruitment are commonly employed in studies with substance users due to difficulties inherent in recruiting random samples within these populations (Stephens, 1999). Participants were recruited from both of Leicester's Universities.

4.2.5 Recruitment

Recruitment spanned from January 2009 to the end of June 2009. During the course of the study 700 questionnaire-packs were distributed. Completed questionnaires were received from 306 participants (response rate of 43.7%). This was particularly encouraging and higher than other

studies employing similar recruitment methodology (e.g. Kenna & Wood, 2008). The details of the recruitment methods are described in the procedure section.

4.3 Materials

4.3.1 Substance use measures

The Smoking History Questionnaire (SHQ)

A modified and shortened version of the SHQ was used to assess smoking history and patterns. This 30-item self-report measure (reduced to 16 items in the present study –see Appendix D) includes amongst other information items pertaining to smoking rate, age of onset of initiation and years of being a regular smoker. The SHQ has been used in previous studies as a descriptive measure of smoking history (Brown, Lejuez, Kahler, & Strong, 2002; Bonn-Miller, Zvolensky, Leen-Feldner, Feldner & Yartz, 2005). This measure was used as a primary index of ‘smoking exposure’. This is a non-validated questionnaire designed primarily for factual information gathering.

Marijuana Smoking History Questionnaire (MSHQ)

A modified and shortened version of the MSHQ was used to assess past and current cannabis use. The MSHQ has been used successfully by Zvolensky *et al.* (2006a; 2006b). This 18-item self report measure (reduced to 15 items in the present study – see Appendix D) includes amongst other information current and past use, attempts at abstinence and beliefs about the effect of cannabis on illness. Additional questions (and pictures) were added to the MSHQ regarding the type of cannabis used and the route of administration. Although Stephens (1999) noted the difficulties in obtaining accurate information about type and quantity of cannabis used, the present author believed that reasonably accurate information regarding type of cannabis (and therefore THC potency) and route of administration could be obtained.

The Brief Michigan Alcoholism Screening Test – (BMAST)

The Brief Michigan Alcoholism Screening Test (Appendix D) is a ten-item self-report inventory designed to screen for lifetime episodes of problematic dependent drinking. The sensitivity of the BMAST is 86-98% and specificity 81-95% (Nilssen & Cone, 1994). Pokorney, Miller and Kaplan (1972) found the BMAST to be as consistent and reliable as the original MAST. The BMAST was chosen over other alternative alcohol screens (e.g CAGE, AUDIT, TWEAK) due to its ability to detect both current and past alcohol problems, essential for the exclusion criteria (see above).

Poly-Substance Questionnaire

An eight item poly-substance questionnaire was created by the author (Appendix D) and used to try and cater for the potential effects of other substances and their relationships with anxiety and panic attacks. This was deemed necessary due to findings that multiple substances of abuse can be related to anxiety problems (Lejuez *et al.*, 2006).

4.3.2 Panic measures

Panic History Questionnaire

A ten item panic history questionnaire (Appendix D) firstly gave a detailed description of a panic attack experience based on that from the Panic Attack Questionnaire –IV (PAQ-IV) (Norton *et al.*, 2008) followed by self diagnosis guidance to the reader taken from DSM-IV. The contained questions were designed to help answer hypotheses one, two, four and five. This questionnaire was designed by the present author.

Panic disorder self report (PDSR)

This questionnaire designed by Newman, Holmes, Zuellig, Kachin, and Behar (2006) consists of 24 items and represents a self-report diagnostic measure of panic disorder based on the DSM-IV diagnostic criteria (see Appendix D). Receiver operating characteristic analyses revealed that the

PDSR showed 100% specificity and 89% sensitivity. The PDSR also demonstrated retest reliability, convergent and discriminate validity, and kappa agreement of .93 with a structured interview. Newman *et al.* (2006) reports that the PDSR demonstrates clinical validity. Students who were identified as having panic disorder using the PDSR did not have significantly different scores on the PDSR than a panic disordered community sample. Therefore the PDSR was a good psychometric for the purposes of diagnosing panic disorder and useful for extrapolating findings into the wider community.

4.3.3 Psychological measures

Anxiety Sensitivity Index (ASI)

The Anxiety Sensitivity Index (Reiss, Peterson, Gursky, & McNally, 1986) is a 16-item measure in which respondents indicate on a 5-point scale the degree to which they are concerned about possible negative consequences of anxiety symptoms (see Appendix D). Factor analysis of the scale indicates it has a hierarchical structure, with three first- order factors entitled AS- Physical concerns, AS- Mental Incapacitation Concerns, and AS-Social Concerns and a single, higher order general factor (Zinbarg, Barlow & Brown, 1997). The ASI has high levels of internal consistency for the global score (range of alpha coefficients = .79 -.90) and good test-retest reliability ($r = .70$ for 3 years; Peterson & Reiss, 1992). The ASI is unique from other measures of anxiety e.g. trait anxiety (Rapee & Medoro, 1994) and the construct is distinguishable from frequency of anxiety symptoms (McNally, 1996).

Multidimensional locus of control (MDLC)

The Multidimensional Locus of Control Scale (Levenson, 1972) is a 24-item self-report instrument that uses a seven-point Likert scale (see Appendix D) . This questionnaire measures internal vs external locus of control and splits external into two sub scales: powerful others and

chance. The MDLC is reported (Furnham and Steele, 1993) to have reasonable reliability (split-half .62-.64, test-retest .62-.91) and validity in terms of concurrent, construct and discriminate validity.

Catastrophic Cognitions Questionnaire (CCQ-M)

Catastrophic Cognitions Questionnaire Modified (Khawaja & Oei, 1992) is a 21-item self-report scale designed to assess catastrophic cognitions associated with panic disorder and agoraphobia (see Appendix D). This questionnaire measures over three subscales: emotional catastrophes, physical catastrophes, and mental catastrophes. Reliability is good as the range of alpha coefficients for this measure range from .86 -.94. (Khawaja, Oei, and Baglioni 1992)

4.4 Procedure

4.4.1 Pilot study

Prior to the final version of the questionnaire pack being agreed and distributed, a small pilot was carried out with approximately ten people participating. This was undertaken to help ensure that the questionnaire made sense to potential participants, could be completed in a reasonable length of time and could provide meaningful data for the study. Mean completion time for the questionnaire was 22 minutes. Suggestions were made by the participants of the pilot study and changes made on the basis of these comments (see Appendix E for pilot questionnaire pack). Changes were relatively minor, some ordering of questionnaires, clearer layout and wording of questions.

4.4.2 Procedure for data collection

Participants were recruited through three main recruitment methods. The first method involved making use of a system that requires first and second year psychology students to participate in a number of research experiments. Seventy participants were recruited through this method. Secondly, students were recruited through accessing student lectures. Individual departments across universities were contacted and asked for permission to enter lectures. Once permission was

obtained individual lecturers were contacted to gain their permission to enter lectures (see Appendix F for list of departments that took part). The principal researcher would enter at the end of a lecture and provide a standardised introduction (see Appendix G). Students would then take a questionnaire pack (see Appendix D) and return it to one of the many clearly labelled ‘drop-boxes’ located around University campuses. All participants were offered an optional entry into a prize draw for taking part in the study, with the total prize fund amounting to one hundred pounds.

4.5 Ethical Considerations

Ethical approval was originally sought from the Central Office of Research Ethics however after submission the Chair of the LREC committee deemed their involvement unnecessary (see Appendix H). Ethical approval was then sought through the University of Leicester’s research governance process where approval to conduct the study was granted following ethical review (see Appendix I). All participants were guaranteed confidentiality and no names or addresses were required for participation. Email addresses (given for the prize draw) were kept on a separate password protected encrypted document and deleted when no longer needed. In order to prevent universities becoming anxious about uncovering a cannabis use epidemic among their student population, data collection did not indicate from which university the participants were recruited. The last page of the questionnaire pack contained a removable information sheet (see Appendix J) which detailed useful contact information regarding where to go to seek help with drug problems or anxiety difficulties.

4.6 Data Analysis

Data was entered by the principal researcher and analysed using SPSS v16. SPSS v16 was used to calculate the scores on subscales of the questionnaires. Descriptive and frequency analysis were used to identify both missing data and entries made in error. Visual inspection of box-plots and histograms was initially undertaken to check for false outliers and normal distributions. Missing data for psychometric subscales were substituted with the mean of other scores across that subscale.

SPSS was also utilised to score the scales on the psychometric tests in an attempt to reduce the probability of manual scoring error.

A variety of parametric and non-parametric statistical tests were used to test the various research hypotheses; these included chi-squared, survival analysis, mann-whitney, and MANOVA.

5. Results

5.1 Descriptive data and data handling

A total of 306 participants took part in the study. Gender makeup of the sample was relatively balanced, with 129 (42.4%) males and 176 females (57.7%) taking part. Mean age for the whole sample was 20.04 years (SD 2.2 yrs), and mean age across groups of interest did not vary significantly (less than 1 yr). Table one below provides descriptive and psychometric data across the whole sample.

Table 1. Whole sample descriptive statistics and psychometric means	
N	306
Mean age years	20.04 (SD 2.2)
Gender ratio	129 M (42.4%) 176 F (57.7%)
Psychometric Mean Scores (SD):	
Power LOC	19.19 (7.5)
Chance LOC	19.78 (7.7)
Internal LOC	31.71 (5.9)
Physical ASI	4.09 (4.1)
Cognitive ASI	2.86 (4.1)
Social ASI	8.21 (4.7)
Total ASI	15.20 (10.8)
Emotion CCQ	13.33 (4.3)
Physical CCQ	25.35 (4.3)
Mental CCQ	19.42 (5.9)
Total CCQ	58.07 (11.9)

LOC = Locus of Control, ASI = Anxiety Sensitivity Index, CCQ = Catastrophic Cognitions Questionnaire. Unless given with %, numbers in brackets represent standard deviations.

A total of 146 participants (47.1%) had used cannabis at least once, 35 (11.4%) had used only tobacco at least once and 125 (40.84%) had used neither. With regards to panic, 88 participants (28.76%) had experienced at least one panic attack; with 24 (7.8%) meeting criteria for panic disorder. Table two and three provide descriptive data across groups of interest. In terms of previous treatment for mental health, 37 (12.2%) reported prior treatment for depression, 18 (5.9%) for anxiety, 12 (4%) for stress, 7 (2.3%) for drug/alcohol and 11 (3.6) for 'other mental health'.

Table 2. Descriptive and psychometric data across groups of interest.						
	Cannabis users	Tobacco Smokers	Neither use	Panic people >0 panic attacks	Non-Panic people	Panic Disorder
N (% of total sample)	146 (47.71%)	35 (11.43%)	125 (40.84%)	88 (28.76%)	217 (70.9%)	24 (7.8%)
Mean age years (SD)	20.17 (1.75)	20.17 (3.67)	19.75 (2.0)	20.08 (3.0)	20 (1.75)	21.23 (4.7)
Gender ratio	M= 70 (47.6 %) F=77 (52.4%)	M=10 (28.6%) F=25 (72.4%)	M = 49 (39.8%) F= 74 (60.2%)	M =29 (33%) F =59 (67%)	M=100 (46.1%) F= 117 (53.9%)	M=7 (28%) F=17 (72%)
Psychometric Subscales:						
Power LOC	19.7 (7.7)	19 (6.9)	18.7 (7.4)	20.4 (8.4)	18.7 (7.1)	22.8 (8.6)
Chance LOC	19.7 (7.8)	19.5 (6.5)	20 (7.9)	21 (8.3)	19.3 (7.4)	22.8 (8.8)
Internal LOC	31.6 (6.0)	32.3 (6.1)	31.6 (5.9)	31.1 (6.7)	31.9 (5.6)	31.7 (5.9)
Physical ASI	3.8 (4.2)	4.6 (3.8)	4.3 (4.0)	5.3 (4.9)	3.6 (3.6)	6.6 (6.1)
Cognitive ASI	2.7 (4.0)	3.9 (5.6)	2.8 (3.7)	3.6 (4.3)	2.5 (4)	4.3 (5.0)
Social ASI	7.8 (4.6)	8.7 (5.1)	8.6 (4.7)	8.8 (4.7)	7.9 (4.7)	9.9 (4.7)
Total ASI	14.3 (10.6)	17.1 (13.1)	15.7 (10.4)	17.7 (11.4)	14 (10.3)	20.9 (11.7)
Emotion CCQ	12.8 (4.3)	13.7 (4.5)	13.8 (4.2)	14.3 (4.2)	12.9 (4.2)	15.9 (4.8)
Physical CCQ	25.1 (4.6)	25 .1(4.4)	25.8 (4.0)	26.1 (4.3)	25 (4.3)	26.7 (5.5)
Mental CCQ	18.6 (6.0)	21.3 (5.9)	19.8 (5.6)	20 (6.0)	19.2 (5.8)	20 (6.7)
Total CCQ	56.4 (12.3)	60.2 (12.8)	59.4 (10.8)	60 (11.6)	57 (11)	62.6 (13.6)
N >0 panic attacks	50 (34%)	9 (25%)	29 (23%)			

Note – The tobacco only group contained in this table represents a looser definition of smoking than originally intended i.e. this group represents those that have smoked tobacco once or more as opposed to the original definition of a pack a day for 6 months or more. Unless given with %, numbers in brackets represent standard deviation.

Table 3. Cannabis users (n=146) descriptive data	
Mean age of first use of cannabis (SD)	15.9 (1.95)
Mean age of regular use among regular users (n=52) (SD)	14.1 (6.3)
Mean percentages of lifetime use by cannabis type:	
Sinsemilla	38%
Resin	17%
Herbal	44%
Main method of administration (%):	
Smoked with tobacco	80.6%
Smoked without tobacco	17.4%
Eaten	2.1%
Panic data:	
Percentage that had reported experiencing 1 or more panic attacks	34%
Percentage that had reporting experiencing a panic attack during cannabis intoxication (among cannabis-panickers)	20%
Percentage that reported ceasing cannabis use due to a panic reaction (among cannabis panickers)	27%

5.2 Data handling

In order to increase the methodological rigour of the results the author decided to exclude all cases where the first occurrence of a panic attack preceded first cannabis use. Therefore this represents an attempt to reduce probability of reverse causality in the prediction that cannabis has some potential causal element in panic pathology. All statistical tests were set at alpha 0.5. Distribution normality for the parametric psychometric data was checked with the Kolmogorov-Smirnov test. However due to the large sample size most of the results came back with spuriously abnormal distributions. This is not uncommon for large sample sizes (Field, 2009). As such visual distributions were produced which showed most variables to have acceptable distributions patterns, although not all. Under professional statistical advice outliers were not removed unless believed to

be false. In the absence of appropriate non-parametric tests for the hypotheses relating to the psychometric data the decision was taken to go ahead with the parametric tests as required.

5.3 Hypotheses testing

Since the sample did not contain enough tobacco-only smokers to compare all three groups adequately, the decision was taken to just compare the cannabis users group with the non-cannabis users group. This was not a desirable situation but practical as the sample contained much fewer tobacco only users than expected at the outset. Therefore the small tobacco only group became part of the non-cannabis user group for all statistical analyses.

5.3.1 Hypothesis one

Hypothesis one was one-tailed and stated that ‘Cannabis users will be significantly more likely to have ever experienced a panic attack than non-cannabis users’.

5.2.1.1 Primary analysis

Due to this being categorical data, Chi-squared analysis was used to test hypothesis one with problem-drinkers (n=14) and poly- substance users (n=57) removed from the analysis. Table 4 below displays the relevant contingency table. No significant association between being a cannabis user and having ever experienced a panic attack $\chi^2 (1) = 0.14$, $p = 0.709$, OR 1.126, 95% CI 0.605 - 2.093 was found. This means that whilst the association between cannabis use and ever having a panic attack is not statistically significant, using chi-squared, the cannabis users group were found to be 13% more likely to experience panic than the non-cannabis users group.

Table 4. Chi-Squared contingency table

		Ever had a panic attack		
		No	Yes	Total
Smoke cannabis	No	117	37	154
	Yes	59	21	80
	Total	176	58	234

5.2.1.2 Secondary analysis

In order to test the primary hypothesis with a more rigorous test of survival analysis, Cox regression with time-dependent covariates was employed. Cox regression is a more powerful parametric test that produces a hazard function in the form of an odds ratio over time. In this analysis age of first cannabis use, age of first panic attack and age at the time of data collection, represent time-dependent covariates used to calculate hazard ratios. This enabled a more sophisticated analysis to examine the length of time between first cannabis use and first panic attack (for further explanation of this statistical procedure see Appendix K). This test found a significant difference between the cannabis users group and the non-cannabis group $p=0.009$, in terms of hazard function and risk of subsequent panic attack(s), HR, 2.01, CI, 1.2-3.4. This means cannabis users, compared to the non-cannabis users were found to be 101% more likely to experience a panic attack.

5.2.2 Hypothesis two

Hypothesis two was one-tailed and stated ‘among those who have experienced at least one panic attack cannabis users will have experienced a significantly higher amount of lifetime panic attacks compared to non-users’. Due to the data collection methods providing ordinal data a Mann-Whitney test of difference was employed to test this hypothesis. Problem-drinkers ($n=6$) and poly- substance users ($n=16$) were removed from the analysis. Cannabis users ($n=21$) (mean rank=33.55) reported experiencing significantly more lifetime panic attacks than the ‘non-user’ ($n=37$) (mean

rank=27.20) group, $U=303.5$, $z=-1.787$, $p=0.038$, $r=.23$. This result represents a small/medium effect size.

5.2.3 Hypothesis three

Hypothesis three was one-tailed and stated ‘Cannabis users are significantly more likely to meet a diagnosis of panic disorder than non-users’. Due to this being categorical data, Chi-squared analysis was used to test hypothesis three with problem-drinkers ($n=14$) and poly-substance users ($n=57$) removed from the analysis. Table 5 (below) shows the contingency table. There was no significant association between being a cannabis user and the presence of panic disorder $\chi^2 (1) = 1.12$, $p=0.768$, OR 1.22, 95% CI 0.385 - 3.848. This means that whilst the association between cannabis use and a diagnosis of panic disorder is not statistically significant, the cannabis users group were found to be 22% more likely to have panic disorder than the ‘non-user’ group.

Survival analysis was not used for hypothesis three as panic disorder represents a diagnostic category, derived from the participants’ previous six months panic history among other criteria, rather than a discrete event such as a panic attack. As such the diagnostic questionnaire used (PDSR) was incapable of assessing lifetime occurrence of panic disorder and the participants’ age at the time of any previous diagnosis of panic disorder. Therefore there was insufficient data for a survival analysis to be performed. In addition the low base rate of confirmed panic disorder in the population would have made the chance of a type 2 error more likely.

Table 5. Chi-Squared contingency table

		Presence of Panic Disorder		
		No	Yes	Total
Smoke cannabis	No	146	8	154
	Yes	75	5	80
	Total	221	13	234

5.2.4 Hypothesis four

Hypothesis four was one-tailed and stated that ‘among cannabis users, those who predominantly used sinsemilla (skunk) would be significantly more likely to have experienced a panic attack compared to those who primarily used herbal cannabis or resin’. Cannabis users were assigned to either group based on percentage lifetime use i.e. >50% lifetime use of sinsemilla gave membership of the sinsemilla group; those that did not meet this criterion were allocated to the resin/herbal cannabis group. This being categorical data, Chi-squared analysis was used to test hypothesis four with problem-drinkers (n=14) and poly-substance users (n=57) removed from the analysis. Table 6 (below) shows the contingency table. There was no significant association between type of cannabis used and having ever experienced a panic attack $\chi^2 (1) = 1.77$, $p = 0.181$, OR 2.02, 95% CI 0.714 - 5.688. This means that whilst the association between cannabis use and a diagnosis of panic disorder is not statistically significant, the sinsemilla group were found to be 102% more likely to have experienced a panic attack than the resin/herbal group.

Table 6. Chi-Squared contingency table

		Ever had a Panic attack		
		No	Yes	Total
Main cannabis of choice	Sinsemilla	16	9	25
	Resin/herbal	43	12	55
	Total	59	21	80

5.2.5 Hypothesis five

Hypothesis five was one-tailed and stated that ‘among cannabis users, who had experienced at least one panic attack, those who predominantly used sinsemilla (skunk) would experience significantly more total lifetime panic attacks compared to those who used primarily herbal cannabis or resin’. Due to the data collection methods providing ordinal data a Mann-Whitney test

of difference was employed to test this hypothesis. Problem-drinkers (n=6) and poly- substance users (n=16) were removed from the analysis. Sinsemilla users (n=9) (mean rank=13.78) experienced significantly more lifetime panic attacks than the resin/herbal (n=12) (mean rank=8.92) group, $U=29$, $z=-2.035$, $p=0.025$, $r=.44$. This result represents a medium effect size.

5.2.7 Hypothesis six

Hypothesis six was stated that ‘among those who have experienced at least one panic attack cannabis users will have significantly higher mean locus of control scores than non-cannabis users’. As this hypothesis having two predictor variables and three outcome variables and parametric data, a MANOVA was employed. Using Pillai’s trace, there was no significant difference between cannabis users and non-users in relation to all three constructs of control, $V=0.066$, $F(1,85)=1.966$, $p>.125$. Between-subject tests (ANOVAs) also found no significant differences on each outcome variable independently. As both multivariate and univariate tests yielded no significant results no discriminate analysis was performed on the data.

5.2.7 Hypothesis seven

Hypothesis seven stated that ‘among those who have experienced at least one panic attack cannabis users will have significantly higher mean anxiety sensitivity scores than non-cannabis users’. As this hypothesis had two predictor variables and three outcome variables and parametric data, a MANOVA was employed. Using Pillai’s trace, there was no significant difference between cannabis users and non-users in relation to all three subscales of anxiety sensitivity, $V=0.002$, $F(1,85)=0.48$, $p>.986$. Between-subject tests (ANOVAs) also found no significant differences on each outcome variable independently. As both multivariate and univariate tests yielded no significant results no discriminate analysis was performed on the data.

5.2.8 Hypothesis eight

Hypothesis eight stated that ‘among those who have experienced at least one panic attack cannabis users will have significantly higher mean catastrophic cognitions scores than non-cannabis users’. As this hypothesis had two predictor variables and three outcome variables and parametric data, a MANOVA was employed. Using Pillai’s trace, there was no significant difference between cannabis users and non-users in relations to all three subscales of catastrophic cognitions, $V=0.055$, $F(1,85)=1.602$, $p>.195$. Between subject tests (ANOVA’s) also found no significant differences on each outcome variable independently, although subscale mental catastrophic cognitions approached significance $p>.057$. As both multivariate and univariate tests yielded no significant results no discriminate analysis was performed on the data.

6. Discussion

The present study aimed to build on and expand the current literature relating to cannabis and tobacco use and their potential relationship with panic attacks. The results found predominantly support hypotheses proposed and current research findings. The study adds new findings in terms of the different types of cannabis and their unique relationship to panic.

6.1 Summary of Results

The results support the primary hypothesis that cannabis users would be more likely than non users to experience a panic attack. Whilst the first chi-squared analysis found no such significant association and only an increased risk of experiencing a panic attack of 13% among cannabis users, the secondary survival analysis did find a significant relationship. This more powerful parametric test found a significant increased hazard ratio for cannabis users resulting in a 101% increase risk of panic attack compared to non users. This will be discussed further below.

As hypothesised, Cannabis users were significantly more likely to suffer from more total lifetime panic attacks than non-users. Contrary to prediction, cannabis users were not significantly more likely to suffer from panic disorder. However, odd ratios indicate that cannabis users have a 22% increased risk of suffering from panic disorder.

There was no effect found of cannabis type on risk of panic. However the odds ratio indicated a 102% increased risk for sinsemillia users in terms of experiencing a panic attack. However, cannabis users who predominantly used sinsemillia experienced significantly more lifetime panic attacks than those who used resin or herbal cannabis

Contrary to prediction, the various psychological and cognitive measures were not found to influence the incidence of panic in this population.

6.2 Key findings

There are four key results from the present study that are worthy of attention. Firstly, the results show that the use of cannabis, even on perhaps just one occasion, can significantly increase (double) the odds of an individual experiencing a panic attack. This finding broadly supports the work of other studies in this area linking cannabis use with both heightened anxiety symptoms, the triggering of panic attacks and panic pathology (Langs *et al.*, 1997; Deas, Gerding & Hazy 2000; Gale and Guenther, 1971; Ganz and Volkmar, 1976; Thomas, 1996; Hathaway, 2003; Dannon *et al.*, 2003, Szuster *et al.*, 1988, Zvolensky *et al.*, 2006). However whilst some research that has shown this link with more dependent cannabis users (Zvolensky *et al.*, 2006a) and those with existing panic disorder (Dannon *et al.*, 2003), the present study takes this further to include those that are inexperienced with cannabis use also.

Secondly, the present study showed that cannabis users not only have a greater chance of experiencing a panic attack *per se* but also experience a significantly greater number of lifetime panic attacks than non-cannabis users. Whilst the effect size for this result was small this is still a useful and novel discovery. To the author's knowledge this represents an interesting new finding in that no known research to date has investigated the differences in lifetime panic attacks between cannabis users and abstainers.

Thirdly, whilst there was no significant difference between cannabis users differentiated by cannabis type/ potency in terms of the likelihood of experiencing a panic attack, the odds were still double for those who predominantly smoked sensimillia.

Fourthly, building on the differences between cannabis types, the present study discovered that cannabis users who smoked predominantly sensimillia experienced significantly more lifetime panic attacks than those that smoked resin or herbal cannabis. Again, to the author's knowledge these represent important new findings in that no known research to date has investigated the differences between different types of cannabis with regards to panic. Indeed only two studies

known to the author have investigated the different types of cannabis and differential effects on mental health (Di Forti *et al.*, 2009; Morgan & Curran, 2008).

Overall 34% of cannabis users reported experiencing at least one panic attack, which is somewhat higher than the 22% reported by Thomas (1996). It is possible that this difference represents the difference in availability of sensimillia at the time of respective studies. Indeed Thomas's (1996) work was with a New Zealand sample, and studies of THC potency around that time (Poulsen & Suterhland, 2000) showed THC content at under half that of UK samples. However it is also possible that the differences are due to self-selection bias.

The finding that panic disorder is not significantly associated with cannabis use is contrary to previous studies (Szuster *et al.*, 1988; Zvolensky *et al.*, 2008). Whilst a 22% increased risk for cannabis users was observed, the lack of significance may well be due to small numbers in the panic disorder group making the test underpowered.

The complete lack of differences between groups in regards to locus of control, anxiety sensitivity and catastrophic cognitions is an unexpected finding of the present study. Previous work has looked into the role of catastrophic cognitions and anxiety sensitivity (Zvolensky *et al.*, 2006b; Bonn-Miller *et al.*, 2005) among cannabis users but has not compared group means directly. It would have seemed logical that those who experienced panic attacks and who also used cannabis might have elevated levels of residual anxiety sensitivity or catastrophic cognitions, due to finding that cannabis users both had an increased likelihood of experiencing a panic attack and experienced more panic attacks in general. However as this was not the case in terms of the findings of the present study, it would point towards a more bio-chemical cause of the increased levels of panic found. Thus the proposed model of the THC present in cannabis having an anxiogenic effect on the CNS, leading to increased catastrophic cognitions about bodily events among those high in anxiety sensitivity and in turn this leading to panic is not supported by this study.

What is perhaps more probable is that THC, or a combination of THC and nicotine are direct causal agents in panic reaction due to their effect on neurotransmitter release and acting as CNS stimulants. That said, this study far from rules out a cognitive component among the cannabis users who panic. Indeed there are countless psychological variables not measured in the present study.

However it is important to recognise that all the findings of the present study do not prove a causal link between cannabis use and panic nor the direction of association. The findings could be interpreted in reverse, in that people who experienced panic are more likely to go on to use cannabis, either with a self medication motivation or due to other variables that result in predisposing people to both panic and cannabis use independently of each other. There is also the potential for a reciprocal causality relationship between cannabis use and panic, as recently postulated by Bonn-miller *et al.* (2007). They propose that cannabis users with high anxiety sensitivity are more likely to use the drug primarily for coping, such that a ‘forward feed cycle’ may begin where cannabis offers short term relief for aversive affect yet ironically adds to longer-term risk of problems with anxiety and panic. A pertinent analogy for this relationship might be an ever increasing bank overdraft!

6.3 Limitations and Strengths

The present study was subject to a number of potential methodological weaknesses as well as strengths. These shall be explored in turn below.

6.3.1 Limitations

Perhaps the most significant limitation was the cross sectional nature of the research design. This limits the ability to form potential causal inferences from the associations found within the data. Whilst ‘proving’ causality is a luxury found only within the physical sciences, other research designs using prospective longitudinal data whilst controlling for all known confounding variables would help unpick the direction of any associations found. That said, the survival analysis used in

the present study along with the steps taken to minimise potential reverse causality represent reasonable attempts to generate useful findings from within the limitations of a cross-sectional design.

The sample population employed in the present study represents a significant methodological weakness in terms of ecological validity. As all participants were university students the results found cannot readily be extrapolated to the general population, or cannabis users therein. That said, unless there is something unique about university students that would distinguish them as a very different population with regards to their physical and psychological response to cannabis or panic attacks, to say no extrapolation could be made would seem unduly cautious. No data were gathered on participants' ethnicity, however it is reasonable to assume that this would broadly reflect that of Leicester's universities which are likely to contain larger percentages of minority ethnic groups due to the population make-up of the local area and a significant number of students from overseas.

There is also the potential problem of the sampling technique. Whilst reasonable attempts were made to obtain a good cross section of university students, the very nature of the study encouraged self-selection bias. It could be argued that students who used cannabis and students who experienced panic attacks were more likely to take part in the study resulting in an over-representation of both in the overall sample. There is also the strong possibility that through answering the questionnaires participants would have worked out the hypotheses of the study and that this knowledge may have influenced their answers. Those that are pro-cannabis may have falsely not disclosed any panic psychopathology, whilst those that may be anti-cannabis may have fabricated panic psychopathology.

The use of self-report data through questionnaires also serves as a methodological weakness, although not an uncommon one within psychological research. One could argue that it's reasonable to expect participants to be able to recall if they *ever* suffered a panic attack, due significance of the psychological distress suffered. However the accuracy of recall of *lifetime* panic attacks is more

questionable. The use of self report data in the area of drug research is also problematic due to problems with shared method variance and problems of retrospective recall among drug users (Stephens, 1999). Other relevant research has used diagnostic interviews to increase reliability but this was beyond the scope of the present study.

Turning to the psychometric measures, scores on these can be affected by current mood state, social desirability, impression management and even the temperature of the room all can distort the reliability of the data obtained. The Panic History questionnaire used to gauge whether a participant's experience qualified as a panic attack (see Appendix D) did follow DSM-IV criteria but it still remains a non-validated psychometric measure. This is also the case for the Marijuana Smoking History Questionnaire (MSHQ) and the Smoking History Questionnaire (SHQ); however as these are primarily factual information gathering tools, the validation issue is less problematic. Whilst the Panic disorder diagnostic psychometric was a robust tool, it does only assess for panic disorder over the last six months. Therefore there may well have been participants with previous panic disorder not detected by this study.

A final limitation in the interpretation of the results of the study is the issue of the confounding effects of smoking. As daily smoking has been linked in some prospective studies to panic attacks, and as 80% of the sample reported their main method of consumption being used with tobacco it is possible that the effects observed are attributable to tobacco use rather than cannabis. Asking different questions of the data set could examine this problem but is outside the scope of the present study. Nevertheless there were differences found in lifetime panic attacks between users of different types of cannabis and both these groups used tobacco with cannabis. This makes it less likely that it is just tobacco having the observed effect on panic frequency. In addition, the majority of the sample were not heavy smokers, of either cannabis or tobacco and it was among daily smokers and dependent cannabis users that the links with panic were reported (Zvolensky *et al.*, 2006a; Isensee *et al.*, 2003).

However very recent research (Zvolensky *et al.*, 2008) using a prospective longitudinal design found that cannabis use and dependence did not significantly increase the odds of panic attack or panic disorder over and above the effects of daily tobacco use. However this study still had the problem of accurately separating out variables of cannabis use and tobacco smoking due to their high rates of co-occurrence. It seems that more work is needed to separate out the potential individual effects of both tobacco and cannabis use.

6.3.2 Strengths

Whilst not counteracting or nullifying the aforementioned limitations of the study, there were some methodological strengths present. Within the questionnaire battery attempts were made to help participants quantify whether their experience met diagnostic criteria for both panic attacks and panic disorder. A validated psychometric was used to assess panic disorder and DSM-IV criteria were used to construct the panic history questionnaire. These steps have not always been taken in previous research (e.g. Thomas, 1996; Hathaway, 2003).

Attempts were made to reduce potential reverse causality by excluding participants who experienced panic attacks prior to their first use of cannabis. Certainly, not all previous research has done so (e.g. Thomas, 1996; Hathaway, 2003; Zvolensky *et al.*, 2006). Potential confounding variables were also controlled for where possible, anyone with a history of problem drinking or poly-substance use were excluded from statistical analysis. Such steps have not always been taken in prior research.

Whilst the use of anonymous questionnaires has its limitations, it can be argued that compared to information gathered through interview, they can encourage more forthright disclosures of sensitive, illegal or socially disapproved behaviour. As such the use of questionnaires may have proved of benefit to the reliability of the data gathered.

The author also took steps to ensure the reliability of the questionnaire data that were entered onto the SPSS database. All entries were double checked once entered by hand and the author did all of the data entry, thus reducing the possibility of error through others completing this role. In addition SPSS was utilised to score the various psychometric data, eliminating the possibility of human error in calculations.

Perhaps the most significant strength of the study was the attempt made to gain data on the different types of cannabis consumed by users. Very few, and only recent studies, have made such attempts or indeed recognised the importance of doing so (Forti *et al.*, 2009; Morgan & Curran, 2008). None, to the author's knowledge, have done so in the area of panic attacks. Accurate identification of the type of cannabis smoked was assisted through pictures, description and street cost. Whilst this is no guarantee of accuracy, it was the most practical method within the questionnaire design.

7. Implications

7.1 Clinical Implications

The findings of the present study along with those of previous research suggest a number of implications for clinicians. In the area of direct substance misuse work drug workers may require updated training with regards to the potential effects of cannabis use upon panic psychopathology. Indeed, whilst further research is needed into the differential effects of cannabis sinsemilla compared to resin and herbal cannabis, it would be useful for drug workers to be aware of the potential heightened risks associated with use of higher potency cannabis. This information could, in turn be passed onto users and may help in objectives such as harm reduction through encouraging ceasing use or moving to a lower THC variety. As studies have found a significant proportion of tobacco users also consume cannabis, it might be pertinent to educate smoking cessation workers in this regard also. This need for updated education seems especially important in view of some professionals' views about cannabis and mental health (Clutterbuck, Tobin, Orford, Copello, Preece & Birchwood, 2009).

With regard to mental health professionals and IAPT workers, including clinical psychologists, it would seem wise to enquire into cannabis use with clients who present with panic anxiety and panic disorder. Again it would appear good practice to quantify the type of cannabis being consumed by patients (if any) and advise of the potential of cannabis exacerbating panic symptoms and increasing the overall frequency of panic, particularly with sinsemilla. As contemporary research has indicated, cannabis users high in anxiety sensitivity may be a particular higher risk subgroup; patients with panic pathology who use cannabis could be screened using the ASI psychometric to assess for further elevations in risk.

Professionals involved in drug education of younger adults could also benefit from updated knowledge of the potential cannabis-panic association. As the present study discovered the mean age of first use being 15.9 yrs and regular users being younger at 14.1 years it could be useful from

a mental health prevention perspective to educate young people about the new findings of the dangers of cannabis use. This could come in the form of drug information leaflets and direct contact by youth workers, teachers and youth offending teams.

Finally there is the possibility of potentially developing targeted treatment methods for panic attacks with co-morbid cannabis use. This could combine cognitive therapy for panic disorder with motivational interviewing for reducing or ceasing cannabis use in an integrative intervention, both approaches having empirical efficacy when used independently with the possibility of being effective when combined. Of course any such treatment design would have to be trialled and tested empirically.

7.2 Wider implications

The classification of cannabis is an area of contemporary debate and controversy. High profile scientists have resigned from posts due to the government not following their advice, which has largely been to lower the severity classification of cannabis due to arguments that its use is not more harmful than alcohol use or smoking tobacco. Certainly whilst it is not uncommon for science and government to be in dispute, the findings of the present study do emphasise the potential dangers of the use of cannabis upon panic pathology and this should be added to the ever increasing evidence base in this area. Since the consumption of sensimillia is the main cannabis of choice in the UK, contemporary studies are more likely to have valid data than those pre 2000. This fact is supported by the WHO (WHO, 1997) report which stated that the majority of cannabis research pre 1997 does not apply to what current users consume today. As such both scientific advisory bodies and government policy decisions need to be mindful of this fact.

It could also be useful as part of a harm reduction policy for countries where cannabis cultivation is legal to promote a policy of growing varieties of cannabis which have a higher proportion of CBD and a lower proportion of THC. This could have the effect of protecting users from the

anxiogenic properties of THC and as such reduce associated anxiety/panic pathology from cannabis use.

8. Future research

The present study points towards several areas of further research. Firstly research could build on the present study by gathering a large and more representative sample from the general population; this would enable more generalisability and hopefully provide the originally intended three groups for comparison. The larger numbers would also help to apply more statistical power to test the hypothesis around panic disorder. Although gender effects were not directly tested by the present study they are worthy of future investigation also. It would be worthwhile trying to discover why, as found in previous research, women are more at risk than men in terms of the negative panic reactions to cannabis. Do, for example, women have a different number or distribution of cannabis receptors in the brain?

Future research could also look at increasing the reliability of the various measures of the present study. To aid retrospective recall, techniques such as the Timeline-Followback technique (Sobell & Sobell, 1996) or Retrospective Alcohol and Other Substance Use Measure (RETROSUB) (Windle, 2005) could be employed. These could be applied to both panic attacks and cannabis use. In terms of increasing the reliability of cannabis self-report there are historical problems associated with this (Stephens, 1999), however urine samples can detect cannabis for up to 28 days from consumption and hair samples can not only detect cannabis, but also the various concentrations of THC and CBD.

The ability of hair samples to detect such differences would be particularly useful for further research into the different types of cannabis, which is undoubtedly an area worthy of further investigation. Future research could use larger samples and try and find cannabis users who have used one type of cannabis exclusively, as this would help increase the likelihood that the differences observed were due to the different cannabis types. In addition, in order to separate out the different effects of cannabis and tobacco, it would be best to find cannabis users who do not smoke cannabis

with tobacco. This would take some searching but would be crucial to untangle the complex chemical interplay.

Ultimately, the ‘fairy-tale’ best option would be to combine all of the ideas above in to a prospective longitudinal study measuring as many theoretical covariates as possible. This would be best placed within a birth cohort study using techniques such as Mendelian randomization to account for genetic confounders brought to light through potential longitudinal twin studies. Attempts to minimise both reverse causality and intoxication effects would be crucial. However it is unlikely the author would secure funding for such an endeavour!

9. Conclusion

At the time of writing, to the author's knowledge, this study is the first British study to explore the relationship between cannabis and panic. Indeed this study represents the first known worldwide attempt to investigate the different types of cannabis and their effect on panic pathology.

In accordance with some previous research (Thomas, 1996; Hathaway, 2003; Zvolensky *et al.*,; 2006a; Langs *et al.*, 1997; Deas *et al.*, 2000; Gale and Guenther, 1971; Ganz and Volkmar, 1976; Zvolensky *et al.*, 2008) the present study found a significant relationship between cannabis use and panic attacks in terms of cannabis users being at increased risk of experiencing a panic attack compared to non-users. In a novel finding, the present study discovered that cannabis users also experience significantly greater lifetime panic attacks than non users.

Other novel findings concern the type of cannabis smoked. The present study showed that cannabis users who smoked primarily sensimillia (skunk) experienced significantly more lifetime panic attacks than those who smoked mainly other types of cannabis. The need for research to recognise the differences in types of cannabis across mental health research is, in the author's view, most pressing.

Whilst further research is required into this area, the results of this study show that a drug which has had a long history of association with relaxation can, paradoxically, lead to just the opposite.

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Section D

CRITICAL APPRAISAL

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By

David Ward

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1. Introduction

This section outlines my experiences and reflections of conducting this research and demonstrates learning points, decisions and debates throughout the research process. It describes among other things the origin and development of the study, selection of area for the literature review, challenges met along the way and the experience of conducting research. It also covers key decision in the design, selection of measures and reflections on the process of data collection and analysis. This part of the thesis was aided by a research journal that I kept throughout the research process and took along to every supervision session to scribble in frantically!

2. Selection of overarching area of Inquiry

My interest in cannabis research did not so much begin at a specific point in time, but rather was born out of an accumulation of observations and events from the age of 16. I could remember during school and undergraduate study quite a few people who had used cannabis, some frequently and others more occasionally at parties. I can remember initially having a very relaxed attitude to my friends' use, seeing cannabis very much as a 'soft drug' which was the reputation of the time. This was especially so as when I first had friends using cannabis all that was available to them was cannabis resin. However, over a series of events my attitude to cannabis began to change. As time progressed I saw the occasional person having some quite unexpected and unusual (in my eyes) reactions to the drug, in the form of extreme fear. These events I now look back on with more informed eyes and realise that some people were experiencing panic attacks and other negative effects of cannabis use.

The individual occurrences described above were beginning to change my mind about the 'softness' of cannabis. I had some close friends at university who were quite heavy users and I observed how some became extremely apathetic and constantly 'stoned' whilst another experienced an acute psychotic reaction believing that he had 'destroyed his brain, memory and intellectual ability' resulting in a brief spell on a psychiatric ward. In addition, I witnessed one of my family members have an acute panic reaction to cannabis use and I believe this was the triggering point for a period of recurring panic attacks for them.

After undergraduate study I worked for the probation service which led me into working with substance misuse. Anecdotal reports of offenders' experiences with cannabis further enriched my interest in the area. My probation experience led to a senior practitioner role within a community drug and alcohol team, I knew that I wanted to conduct my doctoral research in the area of cannabis use.

2.1 Selection of topic for literature review

Narrowing down the area of cannabis I wanted to explore was not an easy decision. I was interested in the neuro-cognitive residual effects of prolonged use (due to the claims of my friend – above), but having read a meta-analysis by Grant *et al.* (2002) in the area I felt that within the limits of a DClinPsy research project I would not be able to add anything useful to the area. I knew that there had been a plethora of research into cannabis and psychosis so I avoided that area of inquiry.

I knew from undergraduate study that what motivated me most to do research was inquiring into novel areas where little work had been done. As such I decided to look into the area of cannabis and anxiety, as after searching, I could not find a literature review dedicated to this theme.

2.2 Reflections on the literature review process

The process of the literature review was a new and unique challenge for me. There was so much information to read, absorb, question and critique. In order to make this process actually possible within my own cognitive limitations I made good use of data extraction tools and constructed a table that made sense to me. This enabled me to organise the different studies by methodology and subject of inquiry. I also created a separate document in which I summarised the study in a paragraph and added my own thoughts and critique. Reflecting on this process now I can see its value but also how I could have probably made it easier for myself through reduction of replication of work. I am also aware that colleagues of mine managed to collate research without such tools.

What did strike me as I read paper after paper and then the references of the relevant papers is just how limited some research is. To begin, one of the things I found most frustrating was the way research is often presented and utilised. The abstracts of papers tell nothing about the quality of the research that has been completed, yet this is exactly what is reported upon by most news sources when presented to the public. Some authors will give an honest and frank critique of their work but others are less forthcoming. However, what I found confusing is that sometimes the limitations are

pushed to one side when drawing conclusions about the study and of course it is these conclusions that end up in the abstract.

Another anomaly that caught my attention occurred when I checked the references contained in the introduction sections. I sometimes found that there was quite a liberal use of a reference to support an argument and on occasion a questionable representation. Combined problems like this with publication bias (a phenomenon not compatible with scientific objectivity) led me to find the experience slightly alarming. Considering the papers I read were quantitative, which generally resides in the positive school of science which implies objectivity of the researcher and generally ignores the possibility of the research influencing the outcome, I felt my alarm was warranted (see Goldacre, 2009 for discussion on biases in the reporting and interpretation of scientific research).

Appraising all the literature for the literature review left me quite confused and discombobulated. I can remember becoming quite frustrated trying to compare all the different designs, methods, measures and outcomes used. I found it difficult to compare studies and to draw meaningful conclusions from the current state of research and was at times convinced I did not have the skill required. Reflecting on this I can see that there was somewhat of a parallel process occurring where my mental state at the time reflected the state of the literature i.e. confused, frustrated and searching for an answer with limited evidence to base any on. However, there was one area that seemed an emerging and promising area and that was cannabis and panic.

2.3 Selecting the focused inquiry of the research report

I decided that from the literature review that I wanted to bring something new to the area by expanding on the work of others and including some new, novel elements. What I had noticed from the literature review was that all authors were treating cannabis as only coming in one variety, rather than the diversity of forms in which cannabis can be purchased. This caused me considerable surprise as I thought it common knowledge that cannabis came in different varieties and potency. I found only one reference (Smith, 2005) that alluded to this fact. Combining what I had read about

THC, CBD and what I knew from the panic research and theory I had a 'eureka' moment where I realised that finding a way to compare higher potency cannabis users versus lower potency users in terms of their experience of panic attacks was to be a focus of my study. Encouragement from research supervisors cemented this topic of enquiry.

3. Constructing the proposal

The construction of the research proposal ended up being a hugely time consuming task, which was not my original expectation. I think I completed eight drafts before settling on a proposal with my supervisors. The following sections shed light on what was involved.

3.1 Decisions concerning the methodology and population

A qualitative method may have proved to be a more valid way of gaining insight into the nuance and detail of experience in terms of cannabis users and their experience of panic attacks. However a quantitative method was selected for what I still now consider to be valid reasons. Firstly, it allowed for more systematic testing of hypotheses that I was very interested in. Secondly, the more empirical method (despite all its aforementioned limitations) is more influential in the field of clinical psychology and psychology in general. Both potential publication and research dissemination should prove more reliably successful and ultimately that any useful clinical implications of my work would stand the best chance of being implemented. Of course this fact is tied up in the whole ethos of psychology trying its very hardest to be a science in order to be taken seriously by the world, but that's a discussion for a different day.

I would like to be able to say that I selected university students due to some pertinent theoretically relevant finding that made them the perfect population of choice. However I believe that it was more to do with the fact that I needed a big sample (from the power calculations) and my project was already behind schedule. Originally I had wanted to recruit just tobacco smokers from the general population and then divide them into cannabis users and non-cannabis users (as a way round the tobacco-panic links). However this seemed implausible given the time-frame and resources available. I had also hoped that I could go through University ethics, which should have been a more appropriate route than NHS ethics procedures. This was a priority of mine after constructing a final agreed proposal had taken so long.

3.2 Decisions concerning design and measures

In an ideal world I would have liked to have embarked on a cohort based longitudinal investigation with a full research team at my side. Fortunately I realised early on that I was doing a clinical psychology doctorate and such ambitions were just slightly out of reach. Having reviewed the literature in the area I was aware of the variety of research designs employed and as such felt that a cross-sectional study would be an acceptable way forward. Encouragement from supervisors helped me settle on this method.

Considerable time was spent trying to work out how I could compare the different users of cannabis by type of cannabis they smoked. From reading reports and some news headlines I had convinced myself that I would be unable to compare groups due to the fact that I believed the cannabis market to be saturated with sensimillia (skunk). Initially I had hoped to overcome this problem by recruiting an older sample through users of cannabis websites. Indeed I spent some time exploring this option and contacted several website owners to this end. However this idea was eventually abandoned due to concerns over the poor uptake of internet based research and the methodological problems of comparing an older group of cannabis users with a younger group in terms of panic attack likelihood.

Choosing the right measures was time consuming and at times difficult. I was conscious of my desire to use some of the same measures (ASI, CCQ) that previous research in the field had used, primarily because I had felt that there was a lot of inconsistency in terms of the measures used across studies. I investigated all the panic related questionnaires using the book by Antony *et al.* (2001) which gave a very useful summary of the various psychometric properties as well as copies of actual questionnaires. One problem I found was that most of the panic-specific questionnaires were just far too lengthy and I wanted to maximise participant uptake by keeping the questionnaire pack completion time as short as possible. Other research has used trained interviewers to diagnose panic attacks and panic disorders, but for the numbers I needed this was not a feasible solution.

However in retrospect it would have been potentially a better option to have used interviews although the trade off probably would have been a smaller overall sample, due to reasons of participants opting out due to the lack of anonymity and the constraints of me being the sole researcher on the project.

In the absence of a suitable psychometric I created my own panic history questionnaire with an emphasis on helping the participant self-diagnose an incident of panic using DSM-IV criteria and a descriptive element amalgamated from various sources. This self made questionnaire was coupled with a short validated panic disorder questionnaire (PDSR) that met the need for brevity quite well. Although the panic disorder questionnaire was unable to diagnose panic disorder in someone's history, it was able to cover the previous 6 months and in the absence of no suitable alternative it was the best option I had.

I also needed a brief alcohol screening questionnaire to be able to exclude problem drinkers. I had originally intended to use the SADQ, however this had two main problems. Firstly, it is lengthy and is really supposed to be administered by a clinician. Secondly, I needed to be able to exclude past as well as current alcohol use in a quick measure. I looked elsewhere to alcohol screening tools that might be used by GPs or within primary care settings. This resulted in selecting the B-MAST which was just what I needed as the questions were framed in such a way as to cover current problem drinking as well as historical episodes.

Thinking about what I would change in respect to the measures, I would probably put more time into finding a more suitable locus of control measure. The Levenson LOC measure did not, upon reflection, really tap into what I was trying to measure in regards to control. In essence I was not so interested in how in control people felt about their lives or how much control they had over life events. I was actually more trying to measure how important it was for them to feel, or be in control over themselves and their bodies and minds. Essentially I wondered if those for whom being in

control was so important, would react more negatively in terms of a foreign substance (in this case cannabis) being present in their system and this predisposing them to panic.

In retrospect I would have also used a different catastrophic cognitions questionnaire or perhaps not included one at all. The feedback from the pilot study was that people generally found the questions of the CCQ difficult to answer. However at that time there was not sufficient scope to search for a replacement for the CCQ and gain permission for using it as there was a pressing need to begin data collection. The other problem with such questionnaires in my research was that probably none of the participants were intoxicated with cannabis at the time. As such only I was unable to measure catastrophic cognitions when they may have been playing a key role in panic among cannabis users. This was not such a problem for the ASI as this is meant to be a more stable construct.

3.3 Ethics

After thinking I could bypass NHS ethics I was informed that this would not be the case and I saw the logic to the argument made. Therefore I spent time going through the NHS ethics procedure, filling in the lengthy repetitive form and taking advice from the lead research manager in the employing trust who oversees NHS applications. My supervisors and I were certain that due to my status as an NHS employee I would have to use this method to obtain approval. However, once this was reviewed by the panel it was deemed an unnecessary application. I then began a fresh application through University ethics to secure approval for my project. At the time I found this all rather frustrating as I was behind schedule anyway. In hindsight I can see the benefit of having gone through filling in the lengthy ethics form, as the experience will be useful for any further NHS research I may engage in. I did however miss out on facing the ethics panel, which may well have been a useful experience.

I think I made reasonable attempts to deal with ethical issues in my research and I felt good that I provided a 'tear off sheet' at the back of the questionnaire detailing how and where help and

information for both substance use and panic attacks could be best found. Some questionnaires came back without this sheet attached and at the time I felt some concern that completing the questionnaire may have highlighted problems for people who were not necessarily aware. Looking back now I am glad that I was able to give participants this information as for some it may have been the first step to gaining support for any difficulties.

4. Undertaking the research

4.1 Use of the EPR system

I was fortunate enough to be able to recruit some participants from the Experiment Participation Requirement system that requires first and second year psychology students to participate in a number of research studies of their choosing. I got about 70 participants through this method. It was fast and reliable and I was somewhat disappointed at the time that I could not have more hours allocated to me to assist in fast data collection. However in hindsight having only a small proportion of the sample from psychology was a positive result as through other recruitment methods I was able to gain a more representative sample of the student population.

4.2 Recruitment through lectures

Recruitment through lectures was my main sampling strategy. This was far more complicated than I thought it was going to be! Somehow when I thought of this I thought I would go into a couple of well attended lectures and get my sample in a flash. How wrong I was! In actuality I had to contact the head of department for each subject I was trying to recruit from, which ended up being all of them. After gaining permission I then had to coordinate attending the various lectures on my research days and ask individual lecturers if I could attend their lecture at the end for a few minutes to give an introduction and hand out questionnaires to willing participants. Standing up in front of a large group of students and talking, even for just a minute, I found very anxiety provoking.

4.3 Other recruitment methods

I tried to get a 'stall' through the student union marketing department in the main area where businesses and organisations often advertise or hand out various free goods. However, these spaces get booked up months in advance and so I had to take what was given to me, which was a table outside the student bar. I did not recruit many people through this medium, partly due to my anxiety

making it difficult to approach people as they went past. In hindsight I should have thought of booking the stall earlier on as it could have been a very successful recruitment method.

4.4 Decision to end recruitment

The sample size target was approx 350 participants based on quite strict sample size calculations. After about four months I had reached approx 300 and was struggling to find new lectures to attend, students were beginning to prepare for exams and teaching was wrapping up. As such I decided to stop recruitment as my supervisors' and I were pleased with the numbers I had obtained for the study.

In retrospect I could have probably stopped a little sooner as I did not obtain more than 20 new participants in the last month. Ending earlier may have helped the overall speed of project completion.

5. Reflections on data handling and analysis

5.1 Statistics

Professional statistics advice was elicited through the University's medical research department. I was grateful for their assistance as although I had statistics experience from undergraduate studies, that learning was many years back and I was more than a little rusty. In fact, without their help I would have never considered the survival analysis statistic as it is not commonly used in psychological research. I was also grateful for the statistics help from supervisors, who helped me take a practical approach when getting bogged down in stats theory.

Thinking about the tests of choice now I think I probably could have done a logistical regression with covariates such as alcohol, poly-substance use and gender. However at the time the advice given was not to violate parametric assumptions, which I have some sympathy with.

With regards to the power calculation, if I had chosen to use Cohen (1991) as most psychologists do I could have had a much lower sample size target. However the professional advice given was that Cohen's power tables were based on some assumptions that lacked statistical rigour and as such I was advised not to use them. Again I could have probably gone against this advice and achieved my desired sample far more quickly but rightly or wrongly I went with my tendency for accuracy over speed. In addition the whole statistics part of the work caused me a lot of stress and anxiety. I did not feel confident in my use of statistics, which is not uncommon amongst psychologists. That said I am really glad I did a quantitative design and used statistics as by the end of my work I felt more confident and a sense of achievement in being able to understand and question statistical tests and assumptions.

5.2 Data Inputting

One drawback to having a large sample was the amount of time taken to input the data by hand. Whilst I had worked out a way for SPSS to score the psychometrics for me the sheer number of

variables entered was huge. In retrospect I probably should have gone with the Cohen power calculations and the lower number would have meant saving two thirds of the time it took to input all the data. Certainly if I had to do it again I would strongly consider that option.

I also could have used some volunteers that the department had a list of to help input some of the data. At the time I felt uncomfortable about this for two main reasons. Firstly because I did not think the volunteers would get much out of the experience, other than perhaps some SPSS knowledge. Secondly, I felt very invested in the outcome of the research, so much so that I did not want an error on the database to potentially effect the results. Reviewing that decision now I can see that I was perhaps being a little too precious about my data and was perhaps making other people's decisions for them regarding whether they would find an experience useful. Countless days spent typing data into a database has a powerful way of making one reflect on one's decisions!

5.3 Staying focused on research questions

Certainly I did lose some time playing with data purely for curiosity sake. I had wanted to ask even more research questions than the substantial amount already covered and had the data to do so. I have to thank my academic supervisor for encouraging me to stay focused on the research questions and not to come to supervision sessions with endless SPSS output. In addition asking too many questions of the same data set can lead to type 1 errors, through the increased chance of finding spurious significant results.

6. Learning points

6.1 Why it took so long

This review would not be complete without due mention of the time it took to complete my research. In fact to not make mention of it would be extremely remiss of me. Why it took so long is a question I often asked myself, perhaps wanting to look for a simple answer. However like a good formulation the answer is often far from simple. There were some technical obstacles in the journey such as the problems with going through two ethics applications, the time it took to find suitable measures and the many months of data collection and data entry. I think these difficulties reflected the over-ambitious nature of the project from conception to completion. If I were to embark on a research project again I would temper my ambitions.

There were also personal barriers in the research process. My ongoing difficulties with dyslexia certainly served as a barrier to the speed of my work from constructing the proposal, to all the reading for the lit review and of course the time for the final write up. I also have a tendency to dislike not understanding an aspect of something I am reading and this can lead me off in tangents trying to find answers. These difficulties were most likely made more difficult through my perfectionist tendencies that can lead to procrastination and actually not producing as good a piece of work as you might have.

Often I lacked faith in my own abilities, despite my achievements to date, and I think this served as a barrier to progress. I think that in retrospect I should have made more use of deadlines to get things done. These tend to have more impact when supervisors are involved in them too as I learnt during the write-up stage.

6.2 Becoming a better researcher

There is no doubt in my mind that I have become a better researcher as a result of my project. I have learnt to try and come to terms with the feelings of uncertainty and ‘not knowing’. I realised

that through learning more and more about an area one often ends up with more questions and when you begin to answer them some more are created in a kind of mushroom effect. Certainly I learnt for the future the need to keep focused on the task and subject in hand and to be aware of any risk of hubris over what I could achieve.

I feel I have learnt the hard way just how time-consuming conducting research primarily by oneself can be. In the current era of the NHS there appears to be less and less time for research and I think for clinical psychologists to use their research skills well it would be wise to perhaps work collaboratively on manageable projects. This does of course depend on the NHS valuing research in the current epoch of target-driven patient contact based competitive markets.

7. Impact of self

My final reflections concern how I was affected by the research process. This is a difficult variable, set of variables, to isolate because of course mostly the process is co-occurring with clinical training. That said I, can reflect on how the two parallel experiences had parallel process occurring somewhat simultaneously. Certainly the image that comes to mind is that of a roller-coaster, an emotional roller coaster that is! I experienced totally polar experiences throughout the project, as if I was tossed between two ends of one of Kelly's (1951) personal constructs. There were times of excitement, for example when I realised I had found some undiscovered territory. There were times of sheer tedium when I was entering data into SPSS for hour upon hour, which reminded me of some of the soul destroying jobs I did whilst a young student. Finally there were undoubtedly many times of stress, anxiety and conversely, apathy. The research project certainly took its toll on my physical health, relationships and the new patches of grey that appeared on my temples!

Ultimately considering it all I feel an amazing sense of achievement reaching the end. There were many times I really considered going back to old careers and lifestyles; I think if I had taken that choice I may have regretted it. However, I saw it through to the end and I think that in doing so I have not just developed as a researcher but as a clinical psychologist and a person too.

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Section E Appendices

Appendix A

AUTHOR GUIDELINES

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Appendix B

Extraction tool	
Author & Date	
N	
Mean age	
Sample population	
Aims	
Design	
Measures	
Findings/conclusions relevant to current paper	
Critique	

Critical Appraisal Tables

Longitudinal Research

<i>Author & Date</i>	<i>N</i>	<i>Mean age</i>	<i>Sample population</i>	<i>Aims</i>	<i>Design</i>	<i>Measures</i>	<i>Findings/conclusions relevant to current paper</i>
Patton et al (2002)	1601 (731 male) (859 female)	14.5 (SD 0.5) at wave 1. At wave 7 20.7 (SD 0.5).	Students from Australian secondary school	To determine whether cannabis use in adolescence predisposes to higher rates of depression and anxiety in young adulthood	Opportunity sample Seven wave prospective longitudinal cohort study over six years	Computerised revised clinical interview schedule (CIS-R) used to measure depression and anxiety at wave 1 and 7 by telephone interviews.	Daily use in young women was associated with an over fivefold increase in the odds of depression and anxiety after adjustment for intercurrent use of other substances. Weekly or more frequent cannabis use predicted an approximately twofold increase for later depression and anxiety for women. Self-medication hypothesis not supported.
Brook, Cohen and Brook (1998)	976 in 1975, by age 27 709 followed up.	No data	Randomly selected children from ages 1-10 yrs old from two counties in up-state New York, USA.	To examine temporal priority in the relationship between psychiatric disorders and drug use	Random, stratified sample. Longitudinal design.	Diagnostic Interview Schedule for Children Version 1 (DISC-1) Interviews used for substance use. Various other demographic measures.	At no point in time was there a reliable correlation between level of cannabis use and rates of anxiety disorder. There was no evidence that anxiety disorders in late adolescence have an influence on later drug use, controlling for earlier drug use.
McGee, Williams, Poulton & Moffitt (2000)	1037 (gender mix not stated)	Assessed at ages 15, 18 & 21 yrs old	New Zealand cohort born between 1 st April 1972 and 31 st of March 1973	To examine the longitudinal association between cannabis use and mental health.	Longitudinal prospective cohort study over 6 years	Self-report cannabis use, alcohol and tobacco questionnaire. Diagnostic Interview Schedule for Children (DISC-C). Diagnostic Interview Schedule (DISC) Revised problem behaviour checklist (RBPC). Parent attachment, family background measures, behaviour problems.	At age 15 cannabis use was significantly higher amongst those with anxiety/depressive disorders, when compared to those with no disorder. At ages 18 and 21 cannabis use was not significantly higher amongst those with anxiety or depressive disorders. Cannabis use at age 15 did not predict anxiety or depressive disorders at age 18. Cannabis use at 18 also did not predict anxiety or depressive disorder at age 21. Authors argue that the self-medication hypothesis is not supported with this study.

Appendix C

<i>Author & Date</i>	<i>N</i>	<i>Mean age</i>	<i>Sample population</i>	<i>Aims</i>	<i>Design</i>	<i>Measures</i>	<i>Findings/conclusions relevant to current paper</i>
Ferguson and Horwood (1997)	1265 at start of birth cohort. No data on number at age 18.	No data	New Zealand cohort born between 1 st April 1972 and 31 st of March 1973	To examine the relationship between early onset cannabis use and later psychosocial adjustment.	Longitudinal cohort study followed over 18 years	Composite International Diagnostic Interview (CIDI) DSM-IV Various other measures too extensive to list here	There was an association between frequency of cannabis use and anxiety disorders. However when other factors were taken into account there was no significant association found. It appears that linkages between cannabis use and other aspects of mental health arose because those who elected to use cannabis at an early age were a high risk population which, independently of cannabis use, would have been at higher than average risk of later adjustment difficulties

Appendix C

Cross-Sectional/Epidemiological Research

<i>Author & Date</i>	<i>N</i>	<i>Mean age</i>	<i>Sample population</i>	<i>Aims</i>	<i>Design</i>	<i>Measures</i>	<i>Findings/conclusions relevant to current paper</i>
Tournier et al (2003)	79 (24m) (55f) participants selected cannabis users out of 685 (586 female) (63 male)	20 (SD 3) 95.7% were single	Undergraduate Psychology students in a French University	To investigate in a non-clinical population the association between cannabis use anxiety in daily life using the Experience Sampling Method (ESM)	Opportunity sample, cross-sectional	ESM used to collect data on cannabis use and state-anxiety in daily life. DSM-IV was used for diagnoses in structured clinical interview.	No significant association between the level of state anxiety and cannabis use in daily life. However a diagnosis of anxiety disorder was associated with an increased likelihood to use cannabis. A diagnosis of agoraphobia was significantly associated with increased likelihood of cannabis use.
Troisi et al. (1998)	133 (male)	No data, although all likely to be over 16.	Italian army draftees. Those with poly-substance were excluded.	To assess the prevalence of DSM-III-R axes I and II disorders in cannabis only users.	Stratified, opportunity sample. Cross-sectional design of cannabis use, abuse and dependence.	Structured clinical interview for DSM-III-R. Beck depression inventory (BDI) Speilberger State-Trait Anxiety index. Toronto Alexithymia scale (TAS-20)	Chronic cannabis use was associated with a high prevalence of co-morbid psychiatric disorders. Significant differences in reported anxiety between users, abusers and dependent cannabis use. The severity of both state and trait anxiety increased with frequency of cannabis use, suggesting a dose relevant effect. Interestingly, only one participant met the criteria for an axis I anxiety disorder.
Clough, d' Abbs, Cairney, Gray, Maruff, Parker & O'Reilly (2005)	103 (60 male)	22 (SD 5.8)	Current cannabis users in indigenous communities in Arnhem Land, Northern Territory, Australia	To investigate the association of cannabis use with adverse mental health effects.	Convenience sampling. Cross-sectional design. Qualitative cluster analysis along with multiple regressions.	Study specific interview questions that drew from the Composite International Diagnostic Interview (CIDI), Mini-international neuropsychiatric interview (MINI), Cut down, annoyed, Guilty, Eye opener (CAGE)	After controlling for age, gender and alcohol use, the 'anxiety-dependency' cluster was significantly positively associated with number of 'cones' smoked per week. There was a incremental effect observed whereby the more 'cones' smoked per week the more 'anxiety-dependency' symptoms
Bonn-Miller et al. (2005)	202 (100 female) of which 147 were current cannabis users.	22.5 (SD 7.9)	Regular cigarette smokers recruited through local adverts from greater Berlington , Vermont USA	To evaluate the incremental validity of regular marijuana use in relation to anxiety and depressive symptoms among young adult tobacco smokers	Stratified (all smokers, no previous psychiatric history, no current psychoactive medication) cross-sectional sample	Smoking history questionnaire (<i>SHQ</i>) Marijuana and alcohol assessment (<i>MAA</i>) Anxiety sensitivity index(<i>ASI</i>) Positive affect negative affect scale (<i>PANAS</i>) Mood and Anxiety symptom questionnaire (MASQ)	After controlling for cigarette use, alcohol use and affect factors (anxiety sensitivity), marijuana use and frequency was related to anxiety symptoms. Regular cannabis users reported more anxiety symptoms than both occasional and non-users.

Appendix C

Author & Date	N	Mean age	Sample population	Aims	Design	Measures	Findings/conclusions relevant to current paper
Degenhardt et al. (2001)	10,641 no data on gender mix	No mean ages given, all over 18 yrs.	Australian adults who took part in the national representative sample of the 'National Survey of Mental Health and Well Being'.	To compare relationships between alcohol cannabis and tobacco and indicators of mental health problems in the general population.	Survey design. Opportunity sample.	Interviews used to gather substance use. Cannabis use consumption categorised into 4 categories. Mental health disorders assessed with modified version of the CIDI (gives both ICD-10 and DSM-IV disorders)	Tobacco and cannabis use were both associated with increased rates of all mental health problems examined. However after controlling for other drug use, neuroticism and demographics, any level of cannabis was not associated with anxiety or affective disorders.
Sethi et al (1986)	50 (male) experimental group, 50 male matched control group. 20 albino mice. 40 albino rats.	Not reported	An unspecified sample of male Indian chronic (>5 years regular use) Cannabis users, compared with a matched control group.	To involve clinical, behavioural and biochemical studies to elucidate the probable mechanism of the observed anti-anxiety effects of cannabis	Stratified, opportunity sample (chronic cannabis users). Experimental Lab-based study	Taylor's Manifest Anxiety Scale. Behavioural observation with the mice. Vogel test with the rats.	Chronic cannabis users were found to report significantly ($p<0.01$) less anxiety than controls. The dose dependent manipulation in the animal study showed that low doses of cannabis created more anxiety responses whereas a high dose created an anti-anxiety effect.
Stewart, Karp, Pihl & Peterson (1997)	Experiment 1 229. (98 male 131 female) Experiment 2 219. (58 male 161 female)	Exp 1 – 18.6 Exp 2 – 20.9	University psychology students at George Washington University, Halifax, Canada.	To examine the relationship between anxiety sensitivity, drug use and reasons for drug use.	Opportunity sample. Cross-sectional correlation approach.	Anxiety sensitivity index (ASI) Author designed questionnaire for demographics and drug use.	Users of marijuana or hashish were found to score significantly lower on the anxiety sensitivity index than non-users.
Wilson & Maguire (1985)	125 no gender data stated	No data	Students at Temple University	To investigate the effects of high and low self-esteem on the subjective experiences of experienced marijuana users.	Opportunity sample. Questionnaire based survey design.	Rosenbergs (1965) self-esteem scale. Tart's (1971) items to measure marijuana effects	Marijuana users with low-self esteem were more likely to experience anxiety about losing control than those with high-self esteem.

Appendix C

Panic Studies (all designs)

<i>Author & Date</i>	<i>N</i>	<i>Mean age</i>	<i>Sample population</i>	<i>Aims</i>	<i>Design</i>	<i>Measures</i>	<i>Findings/conclusions relevant to current paper</i>
Zvolensky et al (2006)	4745 (52% female) 84% Caucasian	42.6 (SD 17.5)	Adult population who took part in the Colorado Social Health Survey, USA	To evaluate lifetime associations between cannabis use, abuse, dependence and panic attacks.	Cross-sectional design. Participants were contacted using randomly sampled household addresses.	Interviews that took place in participants' homes. DSM-III was used for diagnoses in structured clinical interview	Lifetime history of cannabis dependence but not use or abuse was significantly related to an increased risk of panic attacks. Also the onset of panic attacks for participants with a lifetime history of cannabis use was significantly earlier than those without cannabis use. Postulates a cannabis-panic self medication model.
Thomas (1996)	528 (199 admitted cannabis use, 102 male, 95 female)	27 (SD 5.2)	Adults residing in Hastings, New Zealand	To survey the various adverse effects of cannabis use.	Randomised opportunity sample, survey design, cross-sectional.	Questionnaire devised by Thomas (1993)	Female cannabis users reported statistically significantly more panic attacks than male users. Ex-users reported significantly more panic attacks over the last week than current users. No significant differences were found with panic attacks for dependent versus non-dependent users.
Szuster, Pontius & Campos (1988)	Experimental group = 25 Control group 1 = 22 Control group 2 = 25	Experimental group = 29.84 (SD 6.44) Control group 1 = 33.05 (SD 8.98) Control group 2 = 32.96 (SD 5.99)	Patients receiving outpatient treatment for panic disorder. Patients receiving outpatient treatment for depression. General hospital patients, not receiving psychiatric treatment.	To explore the relationship between marijuana smoking and panic anxiety with both panic disorder patients and non-disorder controls.	Opportunity, stratified sample. Experimental, questionnaire based design. Cross-sectional.	Diagnoses confirmed using DSM-III specifications. Questionnaire used to assess demographic information and history of marijuana use.	Patient's diagnoses with panic anxiety reported significantly more anxiety reactions to smoking marijuana than either depressed or non-patient controls. The majority of panic patients ceased use of marijuana due to experiencing anxiety reactions.
Dannon, Lowengrub, Amiaz, Grunhaus & Kotler (2004)	66 (32 female, 34 male)	32.3 (SD 14.2)	Patients receiving treatment for panic disorder at an outpatient facility in Israel.	To compare the treatment of panic disorder in patients with or without cannabis use according to response, relapse and side effects.	Opportunity, stratified sample. Experimental design.	DSM-IV criteria used for diagnosis. Individual clinical histories used for info on cannabis use.	Acute cannabis use can be associated with the onset of panic attacks and panic disorder.

Appendix C

Author & Date	N	Mean age	Sample population	Aims	Design	Measures	Findings/conclusions relevant to current paper
Zvolensky, Bonn-Miller, Bernstein, Mcleish, Feldner & Leen-Feldner. (2006)	265 (137 female) 94% Caucasian. 73% (195) were current marijuana smokers.	22.06 (SD 7.19)	Regular cigarette smokers recruited through local adverts from greater Burlington, Vermont USA	To evaluate whether anxiety sensitivity interacts with marijuana use in relation to panic-relevant variables among young adult tobacco smokers.	Opportunity, stratified sample. Cross sectional design.	Smoking history questionnaire (SHQ); Marijuana and Alcohol Assessment (MAA); Anxiety Sensitivity index (ASI); Positive Affect Negative Affect symptoms Questionnaire (PANAS); Mood and Anxiety Symptom Questionnaire (MASQ); Agoraphobic Cognitions Questionnaire (ACQ)	Marijuana users compared to non-users were at increased risk for anxiety symptoms and catastrophic thinking about bodily events among cigarette smokers high but not low in anxiety sensitivity. This effect was observed after controlling for amount of tobacco use, negative affectivity and alcohol use.
Strohle, Muller & Rupprecht (1998)	1	16 (SD n/a)	16 yr old male joiner, living in Germany with no previous psychiatric history.	To report on a single case of with an onset of panic disorder with agoraphobia related to cannabis use.	Case Report	Assessment by a Psychiatrist using DSM-IV for diagnosis	Authors argue that consumption of marijuana was a causal factor in the development of panic disorder in this case. Panic attacks continued in the absence of cannabis use. They postulate towards a possible genetic link or underlying vulnerability.
Deas, Gerding & Hazy (2000)	1	15	15 yr old American male with no previous psychiatric history	To report on a single case of panic disorder without agoraphobia following cannabis use.	Case report	Assessment by psychiatrist using DSM-IV for diagnoses	Authors argue that consumption of cannabis directly contributed to the development of panic disorder in this case. Panic attacks continued in the absence of cannabis (with urine screening confirming this). Authors postulate that cannabis use may trigger an underlying vulnerability in some users.
Ganz & Volkmar (1976)	5	1. 19 2. 20 3. 21 4. 19 5. 29	Students at Stanford University, USA	To report on the adverse effects of Marijuana use.	Case report on five individuals who experiences adverse reactions to cannabis	Psychiatrist assessment using DSM-IV criteria for diagnosis	Among other negative reactions anxiety occurred in all cases reported and a panic reaction in three others.
Langs et al (1997)	3	1. 53 (f) 2. 30 (m) 3. 35 (f)	Three American adults that presented to mental health treatment services.	To report on some clinical cases where cannabis use has appeared to precipitate panic attacks.	Case report of three individuals who had adverse reactions to cannabis use.	DSM-IV was used to classify panic disorder.	Authors suggest that cannabis may trigger the onset of recurrent panic attacks and uncover latent panic disorders in vulnerable adults.

Appendix C

User surveys/subjective experience

<i>Author & Date</i>	<i>N</i>	<i>Mean age</i>	<i>Sample population</i>	<i>Aims</i>	<i>Design</i>	<i>Measures</i>	<i>Findings/conclusions relevant to current paper</i>
Smart & Adalf (1982)	986 no data on gender ratios	Equal measures of students in grades 5,7,9,11,13	All public and separate school students in Ontario, Canada.	To investigate adverse reactions to cannabis use across frequency of use. Also to investigate the amount of users who seek medical attention for adverse reactions	Cross-sectional, survey design. Stratified single-stage cluster design with paired selection of cluster replicates.	The Drug Use Questionnaire	About 45% of all users and 60% of daily users reported adverse effects of anxiety and confusion. 25% of all users and 56.5% of daily users reported a recurrence of the experience. Interestingly the best predictors of anxiety and confusion reactions were daily use and being female.
Thomas (1996)	528 (199 admitted cannabis use, 102 male, 95 female)	27 (SD 5.2)	Adults residing in Hastings, New Zealand	To survey the various adverse effects of cannabis use.	Randomised opportunity sample, survey design, cross-sectional.	Questionnaire devised by Thomas (1993)	22% of cannabis users reported having experienced a panic attack (not using diagnostic criteria). Female cannabis users reported statistically significantly more panic attacks than male users. Ex-users reported significantly more panic attacks over the last week than current users. No significant differences were found with panic attacks for dependent versus non-dependent users.
Reilly, Didcott, Swift & Hall (1998)	268 (59% male)	36.4 (SD 7.5 years)	Long-term cannabis users residing in New South Wales, Australia	To investigate the characteristics and patterns of cannabis and other drug-use among long-term cannabis users in an Australian rural area.	Snowball sampling, survey design	Structured interview schedule used for data collection	The most frequent cited reasons for using cannabis were to relieve tension and achieve relaxation (61%). The most common reported negative effects were anxiety, depression or paranoia (21%). The majority (72%) of users believe the positive effects outweighed the negative.
Hathaway (2003)	104 (64 male and 40 female)	34 (SD not reported)	Experienced cannabis users residing in Toronto, Canada.	To examine the perceived costs and benefits of cannabis consumption among experienced users	Stratified, opportunity sample. Cross-sectional.	Structured interviews used, with answers rated on a likert scale.	40% of weekly users reported having at least one panic attack related to such use. Relevant findings include the main reasons for cannabis use were enhancement of recreation and coping with stress and anxiety (95%). Although in contrast 50% reported anxiety as a negative side-effect.

Appendix C

Disorder specific studies –all designs

Author & Date	N	Mean age	Sample population	Aims	Design	Measures	Findings/conclusions relevant to current paper
Buckner et al. (2006)	123 (73 female), 110 after exclusion criteria applied. 59.3% White	20.8 (SD 4.1)	Undergraduates from a state University in Florida, USA	To investigate potential moderators in the relationship between cannabis use disorders and social anxiety disorder in relation to the tension-reduction models of addiction.	Opportunity sample. Participants recruited through posters around campus. Lab based experimental study	Physiological reactivity measured via skin conductance response (SCR). Coping measured through the subjective psychophysiological reaction questionnaire (SPRQ) DSM-IV axis 1 dimensions measured using SCID-I/NP structured interview	Cannabis use disorders symptomology was found to be associated with social anxiety disorder symptoms. Only perceived coping moderated the relationship.
Oyefeso (1991)	253	23.4 yrs (SD 2.8)	Undergraduate male cannabis users attending five different universities in Nigeria.	To examine personality differences among five categories of usage in male undergraduate cannabis users.	Opportunity stratified sample. Cross-sectional questionnaire based study.	Edward's (1953) need for autonomy scale. Watson and Friend's (1969) Social avoidance and distress scale. Spielberger's (1970) State-Trait Anxiety Inventory. Adanijo and Oyefeso's (1986) Self-esteem scale.	Involvement in cannabis use was not related to trait anxiety need for autonomy or self-esteem. However cannabis use was related to higher social anxiety with daily users being higher in social anxiety than other users.
Lynskey, Heath, Nelson, Bucholz, Madden, Slutske et al. (2002)	6265 (3445 women, 2779 males)	30 (at age of assessment)	Young adult male and female Australian twins born between 1964 and 1971.	To examine the genetic and environmental contributions to risk of cannabis dependence.	Longitudinal cohort design. Stratified sample.	SSAGA adapted from alcohol genetic studies (Bucholz <i>et al.</i> 1994). DSM-IV used for diagnoses	Social anxiety correlated with cannabis dependence. Social anxiety was found to be a risk factor of cannabis dependence.
Degonda & Angst (1992)	591 (at commencement of study, no data on gender)	19 yrs old for males and 20 yrs old for females at beginning of study	General population of Zurich, Switzerland	To investigate the problems associated with agoraphobia and social phobia.	Representative sample, stratified for age at beginning of study. Longitudinal design.	SPIKE interview SCL-90-R Freiburg Personality Inventory Diagnoses based on DSM-III criteria	Assessment of co-morbidity and phobias and other disorders revealed that agoraphobia was associated with cannabis use. Social phobia showed no such association.

Appendix D

Pack No. _____

Questionnaire Battery

Dear Participant,

Contained in this questionnaire pack are a number of different measures asking questions about cannabis use, smoking habits, panic attacks, as well as thoughts, feelings and opinions you may have. Please take your time when answering the questions and try to be as accurate and honest as possible in your answers. Please complete all questions and sections unless directed to leave any out.

Participation in this study is, of course, entirely voluntary. If you do not wish to participate then please do not take or complete a questionnaire; thank you for your interest though. If at any time during the completion of the questionnaire pack you decide you no longer wish to participate, please dispose of the questionnaire appropriately.

The time taken to complete this questionnaire pack will vary from person to person however it should be in the region of 15-35 minutes, based on a pilot study. If you have any questions please just ask me, or if I am not present when you complete the questionnaires, please email me at djd2@le.ac.uk (if you do not mind disclosing your email address, messages will not be kept over 7 days).

The questionnaires are confidential and **anonymous** and do not ask for any identifiable details from you. Thank you for agreeing to take part in this study; and for your valuable time.

David Ward
Trainee Clinical Psychologist

Prize Draw

There is an optional prize draw for taking part in the study. 1st prize is £50, 2nd is £30, 3rd is £20.

If you wish to be included in the prize draw please leave an email address where you can be contacted to inform you of your win! To help ensure your anonymity it might be best to use an address that is innocuous, not one with your full name. Email addresses will be stored on a password protected file on my personal computer (which has full internet firewall and virus security). This file will be deleted as soon as all data is collected. Winners will be chosen at random.

Email _____

If you would like to be informed of the results of the study please leave your email below
Email _____

Returning your Questionnaire

‘Drop-boxes’ for posting your questionnaire are located at:

1. 3rd floor of the Attenborough Tower (Sociology dept)
2. Psychology General Office – Henry Welcome Building
3. Bennet – Outside lecture theatres (downstairs)

Date ____/____/____

Section 1

Below are some questions regarding use of the drug Cannabis. Please give accurate and honest answers. Thank you for your participation.

For each question below, please write the number of the answer on the blank line(s) to the **right** of each item.

1. Do you currently or have you ever smoked Cannabis? _____

If **NO**, skip the remainder of this section and move onto 'section 2'

2. Please rate your Cannabis use in the past 30 days using the scale below.

0	1	2	3	4	5	6	7	8	_____
No use				Once a week				More than once a day	

3. In your lifetime how many days have you smoked Cannabis?

1-10	10-20	20-30	30-40	40-50	50-60	60-70	70-80	80-90	100+	200+	300+	_____
------	-------	-------	-------	-------	-------	-------	-------	-------	------	------	------	-------

4. How old were you when you first smoked Cannabis? (years & months) _____

5. If you have now **ceased** use of cannabis, how old were you when you stopped? _____

6. How old were you when you started regular daily Cannabis smoking? (years) _____

7. For how many years, altogether, have you been a regular, daily Cannabis smoker? _____

8. Think about your smoking during the last month, how much Cannabis did you smoke in an average **week**?

Under 1/16th or 1.75g, 1/16th or 1.75g, 1/8th or 3.5g, 1/4 or 7g, 1/2 or 14g 1oz or 28g _____

9. Think about your smoking during the last week, how often did you smoke Cannabis in an average **day**? (please answer in number of joints/spliffs) _____

10. When were you smoking the heaviest? (year) _____

11. Have you in the past had a disease or illness you believe was caused or aggravated by you smoking Cannabis? 1 = YES 0 = NO _____

12. Do you have any symptoms now that you believe are caused by your smoking Cannabis? 1 = YES 0 = NO _____

13. Do you have a disease or illness now that you believe is caused by or aggravated by your smoking Cannabis? 1 = YES 0 = NO _____

14. Do you think you have ever had an illness that has been improved through your use of Cannabis? 1 = YES 0 = NO _____

If your answer is yes, please state the illness or problem _____

15. Please indicate on a percentage basis over the course of your entire cannabis smoking history how often you would consume cannabis via the following routes. (note – the four totals should amount to 100% over the four questions).

Smoking with tobacco

Smoking *without* tobacco (either in rizla or through a bong/pipe etc)

Eating

Using a vaporizer

%

Types of cannabis use

The following information is given to assist you in answering the subsequent questions.

Cannabis is generally available in three main varieties.

1. Cannabis Skunk – this is a green bud of the plant which often has a strong smell (similar to the smell of the skunk animal) and retails at around £20-£25 for 1/8th of an ounce or 3.5g.



2. Cannabis Hash or Resin is a black-brown lump made from the resin of the plant and used to be the commonest form of cannabis in the UK. It's sometimes squidgy, but usually hard until heated. It costs around £10-£15 for 1/8th of an ounce or 3.5g.



3. Cannabis Grass or Weed (traditional herbal cannabis) is made from the dried leaves of the plant and looks like tightly packed dried herbs. This is often brown or dark green in colour and looks and smells different (less strong or potent) to skunk. . It costs around £15 for 1/8th



Questions

1. In terms of the types of cannabis you have smoked over your cannabis smoking history, what percentages have you smoked the following types of cannabis.

- a. Skunk 10% 20% 30% 40% 50% 60% 70% 80% 90% 100%
- b. Hash or Resin 10% 20% 30% 40% 50% 60% 70% 80% 90% 100%
- c. Weed 10% 20% 30% 40% 50% 60% 70% 80% 90% 100%

(note – the three totals should amount to 100% over the three questions)

2. In terms of number of days smoking cannabis on how many days have you smoked the following:
Please circle your answer

- a. Skunk 1-10 10-20 20-30 30-40 40-50 50-60 60-70 70-80 80-90 100+ 200+ 300+
- b. Resin 1-10 10-20 20-30 30-40 40-50 50-60 60-70 70-80 80-90 100+ 200+ 300+
- c. Weed 1-10 10-20 20-30 30-40 40-50 50-60 60-70 70-80 80-90 100+ 200+ 300+

3. When you **first** ever smoked cannabis what type was it that you smoked. (please circle)

- a. Skunk
- b. Resin
- c. Weed
- d. Uncertain

Section 2

Below are some questions regarding the use of tobacco. Please give accurate and honest answers. Thank you for your participation.

For each question below, please write the number of the answer on the blank line(s) to the right of each item.

1. 1. Do you currently or have you ever smoked tobacco? _____

If **NO**, skip the remainder of this section and move onto 'section 3'

2. How old were you when you **first** smoked a cigarette? (years/months) _____

3. How old were you when you started regular daily cigarette smoking? (years) _____

4. For how many years, altogether, have you been a regular daily smoker? _____

5. Since you started regular daily smoking, what is the average number of cigarettes you smoked per day? _____

6. Think about your smoking during the **last week**, how many cigarettes did you smoke on an average day? _____

7. When were you smoking the **heaviest**? (which year/s) _____

8. When smoking the heaviest, how many cigarettes did you smoke per day? _____

9. When smoking the lightest, how many cigarettes did you smoke per day? _____

10. How many years have you smoked? (total number of years) _____

11. Have you in the **past** had a disease or illness you believe was caused or aggravated by your smoking? 1 = YES 0 = NO _____

12. Do you have any symptoms **now** that you believe are caused by your smoking? 1 = YES 0 = NO _____

13. Do you have a disease or illness **now** that you believe is caused by or aggravated by your smoking? 1 = YES 0 = NO _____

14. Were you smoking 12 months ago? 1 = YES 0 = NO _____

15. Where you smoking 6 months ago? 1 = YES 0 = NO _____

16. Do you/did you smoke rolling tobacco or ordinary cigarettes? _____

17. Do you/did you use filters with your cigarettes? _____

Section 3

For the purposes of this questionnaire please use the following definitions of panic. Use the criteria of symptoms to ascertain if you have experienced a panic attack.

“A panic attack is the sudden onset of intense fear or terror, often associated with feelings of impending doom, that is not a result of a real danger. Often the symptoms experienced during an attack are: shortness of breath, chest pain or discomfort, dizziness and trembling or shaking. A panic attack is different from feelings of fear, anxiety, or worry that build up over time, and also differs from moderate feelings of fear or anxiety. Instead, it involves quick hitting feelings of extreme terror or fear.”

*“To qualify as a full panic attack, you must have experienced a sudden unexpected increase in anxiety with at least **four** of the following symptoms occurring at the same time.”*

- palpitations, pounding heart, or accelerated heart rate
- sweating
- trembling or shaking
- sensations of shortness of breath or smothering
- feeling of choking
- chest pain or discomfort
- nausea or abdominal distress
- feeling dizzy, unsteady, lightheaded, or faint
- feelings of unreality or being detached from oneself
- fear of losing control or going crazy
- fear of dying
- numbness or tingling sensations
- chills or hot flushes

1. What is your current age? _____

2. Please state your gender (circle answer) Male/ Female

3. Were you ever treated in the past (drugs, psychotherapy, hospitalization) for any of the following?
Please tick response

YES NO

- ___ ___ depression
- ___ ___ anxiety or nervous disorders
- ___ ___ other psychological disorders (Type? _____)
- ___ ___ heart problems (Type? _____)
- ___ ___ migraines or tension headaches
- ___ ___ stress related disorders (e.g. ulcers, hypertension)
- ___ ___ alcohol or drug problems
- ___ ___ neurological problems (e.g. inner ear disturbance)

Please use the definition of Panic Attacks above to answer the following questions

4. How many Panic attacks have you experienced in your lifetime? (circle your answer below)

0 1-10 11-20 21-30 31-40 41-50 51-60 61-70 71-80 81-90 100+

If the answer is zero then please move onto Section 5

5. How old were you when you first experienced a panic attack? Years____Months____

6. Was your first Panic attack experienced after taking Cannabis? (please circle answer)

Yes/No

If 'Yes' what type of cannabis were you smoking (if known – please refer to types described earlier) (please circle response)

Skunk Resin Weed

7. Did you experience your first Panic attack within 30 days of your first (or subsequent uses) of cannabis? (please circle answer)

Yes No

8. If you have experienced Panic attacks, how long have you/did you suffer/ed from them? (please answer in months and years)

9. Have any of your panic attacks experienced when intoxicated with Cannabis, if so how many? Please circle your answers.

0 1 2 3 4 5 6 7 8 9 10 over10 (how many?____)

10. Did you ever cease smoking cannabis due to experiencing a panic attack or anxiety reaction?

Yes/No

Section 4

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Panic attacks are discrete episodes of intense fear, apprehension, or terror that are accompanied by a number of physical symptoms. Panic attacks can either occur for no apparent reason (spontaneously) or upon entering into or being in situations which have become associated with them (for example, long lines, travels, etc.) Do not consider fear to be a panic attack if it lasts most of the day.

- 1) During the last six months, have you had a panic attack or a sudden rush of intense fear or anxiety? (Circle your answer) YES NO

When was the most recent time this occurred?

Date _____

If NO (you have not experienced a panic attack), please leave the remainder of this section blank. If YES, please continue.

- 2) Was at least one panic attack unexpected, as if it came out of the blue? YES NO
- 3) Did it happen more than once? YES NO
- 4) **If YES** to 3, approximately how many panic attacks have you had in your lifetime? _____

If YES to 1, 2, and 3, please answer the following questions:

If NO to 1, 2, and 3, please leave the remainder of this form blank.

- 5) Have you ever worried a lot (for at least one month) about having another panic attack? YES NO
- 6) Have you ever worried a lot (at least one month) that having the attacks meant you were losing control, going crazy, having a heart attack, seriously ill, etc.? YES NO
- 7) Did you ever change your behaviour or do something different (for at least one month) because of the attacks? YES NO

If YES to 5, 6 OR 7 please answer the following questions:

Think back to your most severe panic attack. Did you experience any of the following symptoms?:

- 8) Shortness of breath or smothering sensations? YES NO
- 9) Feeling dizzy, unsteady, lightheaded, or faint? YES NO
- 10) Palpitations, pounding heart, or rapid heart rate? YES NO
- 11) Trembling or shaking? YES NO

- | | | |
|--|-----|----|
| 12) Sweating? | YES | NO |
| 13) Feelings of choking? | YES | NO |
| 14) Nausea or abdominal distress? | YES | NO |
| 15) Numbness or tingling sensations? | YES | NO |
| 16) Flushes (hot flashes) or chills | YES | NO |
| 17) Chest pain or discomfort? | YES | NO |
| 18) Fear of dying? | YES | NO |
| 19) Fear of going crazy or doing something uncontrolled? | YES | NO |

20) How much do these symptoms interfere with your daily functioning? (Circle one)

0	1	2	3	4
No	Mild	Moderate	Severe	Very Severe
Interference	Interference	Interference	Interference	Interference

21) How distressing do you find these symptoms? (Circle one)

0	1	2	3	4
No	Mild	Moderate	Severe	Very Severe
Interference	Interference	Interference	Interference	Interference

- 22) When you have bad panic attacks, does it often take **less than ten minutes** from the point at which the attack begins, to the point at which it reaches a peak or becomes most intense? YES NO
- 23) Just before you began having panic attacks, were you taking any drugs or excessive amounts (more than 4 cups daily) of stimulants (e.g., coffee, tea, or cola with caffeine)? YES NO
- a) If YES, what was it that you were taking? _____
- b) How much of it were you taking (in cups, etc.)? _____
- 24) Have you ever been diagnosed with a medical problem (hyperthyroidism, a seizure or cardiac condition, etc.) that could have caused your panic symptoms? YES NO

Section 5

This questionnaire contains other substances that you may have used in the past or are doing so currently. Please answer the questions below as honestly and accurately as possible. Remember that your responses will be anonymous.

Please indicate the number of occasions (if any) that you have used the following substances in your lifetime. (please circle your answer)

Amphetamines (speed, whiz etc)	0	1	2	3	4	5	6	7	8	9	10+
Barbiturates	0	1	2	3	4	5	6	7	8	9	10+
Tranquilizers (benzo's, moggies)	0	1	2	3	4	5	6	7	8	9	10+
Cocaine	0	1	2	3	4	5	6	7	8	9	10+
Heroin	0	1	2	3	4	5	6	7	8	9	10+
LSD (acid)	0	1	2	3	4	5	6	7	8	9	10+
Ectasy (MDMA)	0	1	2	3	4	5	6	7	8	9	10+
Poppers	0	1	2	3	4	5	6	7	8	9	10+

Section 6

Below are a series of questions regarding drinking behaviour. Please indicate your answer to each question by circling the appropriate Yes/No response. Please be accurate and honest with all your answers and complete all questions leaving none out.

If you do not drink or have **never** consumed alcohol then skip this questionnaire and move onto the next. Thank you for your time.

Please Circle

1	Do you feel you are a normal drinker?	Yes	No
2	Do friends or relatives think you are a normal drinker?	Yes	No
3	Have you ever attended a meeting of Alcoholics Anonymous?	Yes	No
4	Have you ever lost friends or girlfriends/ boyfriends because of drinking?	Yes	No
5	Have you ever got into trouble at work because of drinking?	Yes	No
6	Have you ever neglected your obligations, your family, or your work for 2 or more days in a row because you were drinking?	Yes	No
7	Have you ever had delirium tremens, severe shaking, heard voices or hallucinated after heavy drinking?	Yes	No
8	Have you ever gone to anyone for help about your drinking?	Yes	No
9	Have you ever been in hospital because of drinking?	Yes	No
10	Have you ever been arrested for drunk driving or driving after drinking?	Yes	No

Section 7

Below are a series of attitude statements. Each represents a commonly held opinion. There are no right or wrong answers. You will probably agree with some items and disagree with others. We are interested in the extent to which you agree or disagree with such matters of opinion. Read each statement carefully. Then indicate the extent to which you agree or disagree by circling the number following each statement. The numbers and their meanings are indicated below:

If you agree strongly : circle +3
 If you agree somewhat: circle +2
 If you agree slightly: circle +1

If you disagree slightly: circle -1
 If you disagree somewhat: circle -2
 If you disagree strongly : circle -3

First Impressions are usually the best. Read each statement, decide if you agree or disagree and the strength of your opinion, and then circle the appropriate number.

PLEASE GIVE YOUR OPINION ON EVERY STATEMENT.

If you find that the numbers to be used in answering do not adequately reflect your own opinion, use the one that is closest to the way you feel. Thank you.

	<i>Strongly disagree</i>	<i>Disagree</i>	<i>Slightly Disagree</i>	<i>Slightly Agree</i>	<i>Agree</i>	<i>Strongly Agree</i>
1. Whether or not I get to be a leader depends mostly on my ability.	-3	-2	-1	+1	+2	+3
2. To a great extent my life is controlled by accidental happenings.	-3	-2	-1	+1	+2	+3
3. I feel like what happens in my life is mostly determined by powerful people.	-3	-2	-1	+1	+2	+3
4. Whether or not I get into a car accident depends mostly on how good a driver I am.	-3	-2	-1	+1	+2	+3
5. When I make plans, I am almost certain to make them work.	-3	-2	-1	+1	+2	+3
6. Often there is no chance of protecting my personal interests from bad luck happenings.	-3	-2	-1	+1	+2	+3
7. When I get what I want, it's usually because I'm lucky.	-3	-2	-1	+1	+2	+3
8. Although I might have good ability, I will not be given leadership responsibility without appealing to those in positions of power.	-3	-2	-1	+1	+2	+3
9. How many friends I have depends on how nice a person I am.	-3	-2	-1	+1	+2	+3

	<i>Strongly disagree</i>	<i>Disagree</i>	<i>Slightly Disagree</i>	<i>Slightly Agree</i>	<i>Agree</i>	<i>Strongly Agree</i>
10. I have often found that what is going to happen will happen.	-3	-2	-1	+1	+2	+3
11. My life is chiefly controlled by powerful others.	-3	-2	-1	+1	+2	+3
12. Whether or not I get into a car accident is mostly a matter of luck.	-3	-2	-1	+1	+2	+3
13. People like myself have very little chance of protecting our personal interests when they conflict with those of strong pressure groups.	-3	-2	-1	+1	+2	+3
14. It's not always wise for me to plan too far ahead because many things turn out to be a matter of good or bad fortune.	-3	-2	-1	+1	+2	+3
15. Getting what I want requires pleasing those people above me.	-3	-2	-1	+1	+2	+3
16. Whether or not I get to be a leader depends on whether I'm lucky enough to be in the right place at the right time.	-3	-2	-1	+1	+2	+3
17. If important people were to decide they didn't like me, I probably wouldn't make many friends.	-3	-2	-1	+1	+2	+3
18. I can pretty much determine what will happen in my life.	-3	-2	-1	+1	+2	+3
19. I am usually able to protect my personal interests.	-3	-2	-1	+1	+2	+3
20. Whether or not I get into a car accident depends mostly on the other driver.	-3	-2	-1	+1	+2	+3
21. When I get what I want, it's usually because I worked hard for it.	-3	-2	-1	+1	+2	+3
22. In order to have my plans work, I make sure that they fit in with the desires of people who have power over me.	-3	-2	-1	+1	+2	+3
23. My life is determined by my own actions.	-3	-2	-1	+1	+2	+3
24. It's chiefly a matter of fate whether or not I have a few friends or many friends.	-3	-2	-1	+1	+2	+3

Section 8

Please circle the number that best corresponds to how much you agree with each item. If any items concern something that you have never experienced (e.g., fainting in public), then answer on the basis of how you think you might feel *if you had* such an experience. Otherwise, answer all items on the basis of your own experience. Be careful to circle only one number for each item and please answer all items.

	Very little	A little	Some	Much	Very much
1. It is important for me not to appear nervous.	0	1	2	3	4
2. When I cannot keep my mind on a task, I worry that I might be going crazy.	0	1	2	3	4
3. It scares me when my heart beats rapidly.	0	1	2	3	4
4. When my stomach is upset, I worry that I might be seriously ill.	0	1	2	3	4
5. It scares me when I am unable to keep my mind on a task.	0	1	2	3	4
6. When I tremble in the presence of others, I fear what people might think of me.	0	1	2	3	4
7. When my chest feels tight, I get scared that I won't be able to breathe properly.	0	1	2	3	4
8. When I feel pain in my chest, I worry that I'm going to have a heart attack.	0	1	2	3	4
9. I worry that other people will notice my anxiety.	0	1	2	3	4
10. When I feel "spacey" or spaced out I worry that I may be mentally ill.	0	1	2	3	4
11. It scares me when I blush in front of people.	0	1	2	3	4
12. When I notice my heart skipping a beat, I worry that there is something seriously wrong with me.	0	1	2	3	4
13. When I begin to sweat in a social situation, I fear people will think negatively of me.	0	1	2	3	4
14. When my thoughts seem to speed up, I worry that I might be going crazy.	0	1	2	3	4
15. When my throat feels tight, I worry that I could choke to death.	0	1	2	3	4
16. When I have trouble thinking clearly, I worry that there is something wrong with me.	0	1	2	3	4
17. I think it would be horrible for me to faint in public.	0	1	2	3	4
18. When my mind goes blank, I worry there is something terribly wrong with me.	0	1	2	3	4

Section 9

The items listed below aim to measure your beliefs and thoughts regarding the following items. Sometimes these items are believed to be *DANGEROUS*. Please read each item carefully and using the scale given below, rate it by circling the appropriate answer.

	Not at all Dangerous	A Little Dangerous	Quite Dangerous	Very Dangerous	Extremely Dangerous
1. Feeling edgy	1	2	3	4	5
2. Having an accident	1	2	3	4	5
3. Mind not functioning normally	1	2	3	4	5
4. Being miserable	1	2	3	4	5
5. Being injured	1	2	3	4	5
6. Unable to think rationally	1	2	3	4	5
7. Feeling shaky	1	2	3	4	5
8. Having a stroke	1	2	3	4	5
9. Unable to control thinking	1	2	3	4	5
10. Being agitated	1	2	3	4	5
11. Being ill	1	2	3	4	5
12. Losing memory	1	2	3	4	5
13. Unable to relax	1	2	3	4	5
14. Being suffocated	1	2	3	4	5
15. Being mentally blocked	1	2	3	4	5
16. Being alarmed	1	2	3	4	5
17. Being attacked	1	2	3	4	5
18. Being out of senses	1	2	3	4	5
19. Being angry	1	2	3	4	5
20. Losing sight	1	2	3	4	5
21. Being mentally blurred	1	2	3	4	5

PLEASE TEAR OFF AND TAKE THIS SHEET WITH YOU IF YOU WISH

Drug and Panic Information

If you have any concerns about drug use and would like more information or help please make use of the information below:

The 'talktofrank' service give information about drugs and alcohol and what services are on offer in your area to help you.

www.talktofrank.com ☐ [PHONE 0800 77 66 00](tel:0800776600)

If you have concerns about excessive anxiety, panic or other mental health problems please use the information provided below:

Mental Health Care

Comprehensive information about mental illnesses.

Email: subscribe@mentalhealth.org.uk

Website: www.mentalhealthcare.org.uk

Mind

Offers many services including helplines, drop-in centres, supported housing, counselling, befriending, advocacy, employment and training schemes.

Information line: 0845 766 0163

Email: contact@mind.org.uk

Website: www.mind.org.uk

Rethink

Support and advice for everyone affected by severe mental illness.

General enquiries: 0845 456 0455

National advice service: 020 8974 6814

Email: info@rethink.org

Website: www.rethink.org

Samaritans

Provides 24-hour, confidential emotional support to any person who is suicidal or despairing.

UK helpline: 08457 909090

ROI helpline: 1850 609090

Email: jo@samaritans.org

Website: www.samaritans.org.uk

No Panic

Help for people with panic attacks, phobias, obsessive-compulsive disorders and general anxiety disorders.

Helpline: 0808 808 0545

Email: ceo@nopanic.org.uk

Website: www.nopanic.org.uk

AnxietyUK (formerly the National Phobics Society)

A user-led organisation dealing with anxiety disorders.

Tel: 08444 775 774

Email: info@anxietyuk.org.uk

Website: www.anxietyuk.org.uk

Appendix E

Pack No. _____

Questionnaire Pack

Dear Participant,

Thank you for agreeing to take part in this study. Contained in this questionnaire pack are a number of different measures asking questions about smoking habits, cannabis use, panic attacks, as well as thoughts, feelings and opinions you may have. Please take your time when answering the questions and try to be as accurate and honest as possible in your answers. Also please complete all questions unless directed to leave any out.

The time taken to complete this questionnaire pack will vary from person to person however it should be in the region of 15-30 minutes. If you have any questions please just ask me, or if I am not present please email me at djd2@le.ac.uk.

The questionnaires are **anonymous** and do not ask for any identifiable details from you. The entry to the prize draw is optional and your email address will be stored electronically until the end of the study when it will be permanently deleted.

Prize Draw

There is an optional prize draw for taking part in the study. 1st prize is £50, 2nd is £30, 3rd is £20.

If you wish to be included in the prize draw please leave an email address where you can be contacted to inform you of your win!

Email _____

Date ____/____/____

CANNABIS SMOKING HISTORY QUESTIONNAIRE

Below are some questions regarding use of the drug Cannabis. Please give accurate and honest answers. Thank you for your participation.

For each question below, please write the number of the answer on the blank line(s) to the **right** of each item.

1. Do you currently or have you ever smoked Cannabis? _____

If **NO**, skip the remainder of this questionnaire and move onto the next questionnaire.

2. Please rate your Cannabis use in the past 30 days using the scale below.

0	1	2	3	4	5	6	7	8	_____
No use				Once a week				More than once a day	

3. In your lifetime how many days have you smoked Cannabis?

1-10	10-20	20-30	30-40	40-50	50-60	60-70	70-80	80-90	100+	200+	300+	_____
------	-------	-------	-------	-------	-------	-------	-------	-------	------	------	------	-------

4. How old were you when you **first** smoked Cannabis? (years & months) _____

5. If you have now **ceased** use of cannabis, how old were you when you stopped? _____

6. How old were you when you started regular daily Cannabis smoking? (years) _____

7. For how many years, altogether, have you been a regular, daily Cannabis smoker? _____

8. Think about your smoking during the **last month**, how much Cannabis did you smoke in an average **week**?

Under 1/16 th or 1.75g,	1/16 th or 1.75g,	1/8 th or 3.5g,	1/4 or 7g,	1/2 or 14g	1oz or 28g	_____
------------------------------------	------------------------------	----------------------------	------------	------------	------------	-------

9. Think about your smoking during the **last week**, how often did you smoke Cannabis in an average **day**? (please answer in number of joints/spliffs) _____

10. When were you smoking the **heaviest**? (year) _____

11. Have you in the **past** had a disease or illness you believe was caused or aggravated by you smoking Cannabis? 1 = YES 0 = NO _____

12. Do you have any symptoms **now** that you believe are caused by your smoking Cannabis? 1 = YES 0 = NO _____

13. Do you have a disease or illness **now** that you believe is caused by or aggravated by your smoking Cannabis? 1 = YES 0 = NO _____

14. Do you think you have ever had an illness that has been improved through your use of Cannabis? 1 = YES 0 = NO _____

If your answer is yes, please state the illness or problem _____

15. Please indicate on a percentage basis over the course of your entire cannabis smoking history how often you would consume cannabis via the following routes. (note – the three totals should amount to 100% over the four questions).

Smoking with tobacco

Smoking *without* tobacco (either in rizla or through a bong/pipe etc)

Eating

Using a vaporizer

%

Types of cannabis use

The following information is given to assist you in answering the subsequent questions.

Cannabis is generally available in three main varieties.

1. Cannabis Skunk – this is a green bud of the plant which often has a strong smell (similar to the smell of the skunk animal) and retails at around £20-£25 for 1/8th of an ounce or 3.5g.



2. Cannabis Hash or Resin is a black-brown lump made from the resin of the plant and used to be the commonest form of cannabis in the UK. It's sometimes squidgy, but usually hard until heated. It costs around £10-£15 for 1/8th of an ounce or 3.5g.



3. Cannabis Grass or Weed (traditional herbal cannabis) is made from the dried leaves of the plant and looks like tightly packed dried herbs. This is often brown or dark green in colour and looks and smells different (less strong or potent) to skunk.



Questions

1. In terms of the types of cannabis you have smoked over your cannabis smoking history what percentages have you smoked the following types of cannabis.

- a. Skunk 10% 20% 30% 40% 50% 60% 70% 80% 90% 100%
- b. Hash or Resin 10% 20% 30% 40% 50% 60% 70% 80% 90% 100%
- c. Weed 10% 20% 30% 40% 50% 60% 70% 80% 90% 100%

(note – the three totals should amount to 100% over the three questions)

2. In terms of number of occasions smoking cannabis on how many times have you smoked the following: Please circle your answer

- a. Skunk 1-10 10-20 20-30 30-40 40-50 50-60 60-70 70-80 80-90 100+ 200+ 300+
- b. Resin 1-10 10-20 20-30 30-40 40-50 50-60 60-70 70-80 80-90 100+ 200+ 300+
- c. Weed 1-10 10-20 20-30 30-40 40-50 50-60 60-70 70-80 80-90 100+ 200+ 300+

3. When you **first** ever smoked cannabis what type was it that you smoked. (please circle)

- a. Skunk
- b. Resin
- c. Weed
- d. Uncertain

TOBACCO SMOKING HISTORY QUESTIONNAIRE

Below are some questions regarding use of the drug Cannabis. Please give accurate and honest answers. Thank you for your participation.

For each question below, please write the number of the answer on the blank line(s) to the right of each item.

If you have **never** smoked a cigarette please leave this questionnaire blank and move onto the next questionnaire.

1. How old were you when you **first** smoked a cigarette? (years/months) _____
2. How old were you when you started regular daily cigarette smoking? (years) _____
3. For how many years, **altogether**, have you been a regular daily smoker? _____
4. Since you **started** regular daily smoking, what is the average number of cigarettes you smoked per day? _____
7. Think about your smoking during the **last week**, how many cigarettes did you smoke in an average day? _____
8. When were you smoking the **heaviest**? (which year/s) _____
9. **When smoking the heaviest**, how many cigarettes did you smoke per day? _____
10. **When smoking the lightest**, how many cigarettes did you smoke per day? _____
11. How many years have you smoked? (total number of years) _____
12. Have you in the **past** had a disease or illness you believe was caused or aggravated by your smoking? 1 = YES 0 = NO _____
13. Do you have any symptoms **now** that you believe are caused by your smoking? 1 = YES 0 = NO _____
14. Do you have a disease or illness **now** that you believe is caused by or aggravated by your smoking? 1 = YES 0 = NO _____
15. Were you smoking 12 months ago? 1 = YES 0 = NO _____
16. Where you smoking 6 months ago? 1 = YES 0 = NO _____
17. Do you/did you smoke rolling tobacco or ordinary cigarettes? _____
18. Do you/did you use filters with your cigarettes? _____

Panic History Questionnaire

For the purposes of this questionnaire please use the following definition of Panic.

“A panic attack is the sudden onset of intense fear or terror, often associated with feelings of impending doom, that is not a result of a real danger. Some of the most common symptoms experienced during an attack are: dizziness, shortness of breath, chest pain or discomfort, and trembling or shaking. A panic attack differs from feelings of fear, anxiety, or worry that build up over time, and also differs from moderate feelings of fear or anxiety. Rather, it involves quick hitting feelings of extreme terror or fear.”

*“To qualify as a full panic attack, you must have experienced a sudden unexpected increase in anxiety with at least **four** of the following symptoms occurring at the same time.”*

- ☐ ☐ *palpitations, pounding heart, or accelerated heart rate*
- ☐ ☐ *sweating*
- ☐ ☐ *trembling or shaking*
- ☐ ☐ *sensations of shortness of breath or smothering*
- ☐ ☐ *feeling of choking*
- ☐ ☐ *chest pain or discomfort*
- ☐ ☐ *nausea or abdominal distress*
- ☐ ☐ *feeling dizzy, unsteady, lightheaded, or faint*
- ☐ ☐ *feelings of unreality or being detached from oneself*
- ☐ ☐ *fear of losing control or going crazy*
- ☐ ☐ *fear of dying*
- ☐ ☐ *numbness or tingling sensations*
- ☐ ☐ *chills or hot flushes*

1. Were you ever treated in the past (drugs, psychotherapy, hospitalization) for any of the following?
Please tick response

YES NO

- ☐ ☐ depression
- ☐ ☐ anxiety or nervous disorders
- ☐ ☐ other psychological disorders (Type? _____)
- ☐ ☐ heart problems (Type? _____)
- ☐ ☐ migraines or tension headaches
- ☐ ☐ stress related disorders (e.g. ulcers, hypertension)
- ☐ ☐ alcohol or drug problems
- ☐ ☐ neurological problems (e.g. inner ear disturbance)

2. Please state your gender (circle answer)

Male Female

3. How many Panic attacks have you experienced in your lifetime? (please circle your answer below)

0 1-10 10-20 20-30 30-40 40-50 50-60 60-70 70-80 80-90 100+

4. How old were you when you first experienced a panic attack? Years____Months____

5. Was your first Panic attack experienced when intoxicated with Cannabis? (please circle answer)

Yes/No

If 'Yes' what type of cannabis were you smoking (if known – please refer to types described earlier) (please circle response)

Skunk Resin Weed

6. Did you experience your first Panic attack within 30 days of your first (or subsequent uses) of cannabis? (please circle answer)

Yes No

7. If you have experienced Panic attacks, how long have you suffered from them? (please answer in months and years)

8. Have any of your panic attacks experienced when intoxicated with Cannabis, if so how many?

0 1 2 3 4 5 6 7 8 9 10+

9. Did you ever cease smoking cannabis due to experiencing a panic attack or anxiety reaction?

Yes/No

10. What is your current age? _____

Poly substance questionnaire

This questionnaire contains other substances that you may have used in the past or are doing so currently. Please answer the questions below as honestly and accurately as possible. Remember that your responses will be anonymous.

Please indicate the number of occasions (if any) that you have used the following substances in your lifetime. (please circle your answer)

Amphetamines (speed, whiz etc)	0	1	2	3	4	5	6	7	8	9	10+
Barbiturates	0	1	2	3	4	5	6	7	8	9	10+
Tranquilizers (benzo's, moggies)	0	1	2	3	4	5	6	7	8	9	10+
Cocaine	0	1	2	3	4	5	6	7	8	9	10+
Heroin	0	1	2	3	4	5	6	7	8	9	10+
LSD (acid)	0	1	2	3	4	5	6	7	8	9	10+
Ectasy (MDMA)	0	1	2	3	4	5	6	7	8	9	10+
Poppers	0	1	2	3	4	5	6	7	8	9	10+

MAST

Below are a series of questions regarding drinking behaviour. Please indicate your answer to each question by circling the appropriate Yes/No response. Please be accurate and honest with all your answers and complete all questions leaving none out.

If you do not drink or have never consumed alcohol then skip this questionnaire and move onto the next. Thank you for your time.

1	Do you feel you are a normal drinker?	Yes	No
2	Do friends or relatives think you are a normal drinker?	Yes	No
3	Have you ever attended a meeting of Alcoholics Anonymous?	Yes	No
4	Have you ever lost friends or girlfriends/ boyfriends because of drinking?	Yes	No
5	Have you ever got into trouble at work because of drinking?	Yes	No
6	Have you ever neglected your obligations, your family, or your work for 2 or more days in a row because you were drinking?	Yes	No
7	Have you ever had delirium tremens, severe shaking, heard voices or hallucinated after heavy drinking?	Yes	No
8	Have you ever gone to anyone for help about your drinking?	Yes	No
9	Have you ever been in hospital because of drinking?	Yes	No
10	Have you ever been arrested for drunk driving or driving after drinking?	Yes	No

MDLOC scale

Below are a series of attitude statements. Each represents a commonly held opinion. There are no right or wrong answers. You will probably agree with some items and disagree with others. We are interested in the extent to which you agree or disagree with such matters of opinion. Read each statement carefully. Then indicate the extent to which you agree or disagree by circling the number following each statement. The numbers and their meanings are indicated below:

If you agree strongly : circle +3

If you agree somewhat: circle +2

If you agree slightly: circle +1

If you disagree slightly: circle -1

If you disagree somewhat: circle -2

If you disagree strongly : circle -3

First Impressions are usually the best. Read each statement, decide if you agree or disagree and the strength of your opinion, and then circle the appropriate number.

PLEASE GIVE YOUR OPINION ON EVERY STATEMENT.

If you find that the numbers to be used in answering do not adequately reflect your own opinion, use the one that is closest to the way you feel. Thank you.

	<i>Strongly disagree</i>	<i>Disagree</i>	<i>Slightly Disagree</i>	<i>Slightly Agree</i>	<i>Agree</i>	<i>Strongly Agree</i>
1. Whether or not I get to be a leader depends mostly on my ability.	-3	-2	-1	+1	+2	+3
2. To a great extent my life is controlled by accidental happenings.	-3	-2	-1	+1	+2	+3
3. I feel like what happens in my life is mostly determined by powerful people.	-3	-2	-1	+1	+2	+3
4. Whether or not I get into a car accident depends mostly on how good a driver I am.	-3	-2	-1	+1	+2	+3
5. When I make plans, I am almost certain to make them work.	-3	-2	-1	+1	+2	+3
6. Often there is no chance of protecting my personal interests from bad luck happenings.	-3	-2	-1	+1	+2	+3
7. When I get what I want, it's usually because I'm lucky.	-3	-2	-1	+1	+2	+3
8. Although I might have good ability, I will not be given leadership responsibility without appealing to those in positions of power.	-3	-2	-1	+1	+2	+3
9. How many friends I have depends on how nice a person I am.	-3	-2	-1	+1	+2	+3
10. I have often found that what is going to happen will happen.	-3	-2	-1	+1	+2	+3

11. My life is chiefly controlled by powerful others.	-3	-2	-1	+1	+2	+3
12. Whether or not I get into a car accident is mostly a matter of luck.	-3	-2	-1	+1	+2	+3
13. People like myself have very little chance of protecting our personal interests when they conflict with those of strong pressure groups.	-3	-2	-1	+1	+2	+3
14. It's not always wise for me to plan too far ahead because many things turn out to be a matter of good or bad fortune.	-3	-2	-1	+1	+2	+3
15. Getting what I want requires pleasing those people above me.	-3	-2	-1	+1	+2	+3
16. Whether or not I get to be a leader depends on whether I'm lucky enough to be in the right place at the right time.	-3	-2	-1	+1	+2	+3
17. If important people were to decide they didn't like me, I probably wouldn't make many friends.	-3	-2	-1	+1	+2	+3
18. I can pretty much determine what will happen in my life.	-3	-2	-1	+1	+2	+3
19. I am usually able to protect my personal interests.	-3	-2	-1	+1	+2	+3
20. Whether or not I get into a car accident depends mostly on the other driver.	-3	-2	-1	+1	+2	+3
21. When I get what I want, it's usually because I worked hard for it.	-3	-2	-1	+1	+2	+3
22. In order to have my plans work, I make sure that they fit in with the desires of people who have power over me.	-3	-2	-1	+1	+2	+3
23. My life is determined by my own actions.	-3	-2	-1	+1	+2	+3
24. It's chiefly a matter of fate whether or not I have a few friends or many friends.	-3	-2	-1	+1	+2	+3

ASI-3

Please circle the number that best corresponds to how much you agree with each item. If any items concern something that you have never experienced (e.g., fainting in public), then answer on the basis of how you think you might feel *if you had* such an experience. Otherwise, answer all items on the basis of your own experience. Be careful to circle only one number for each item and please answer all items.

	Very little	A little	Some	Much	Very much
1. It is important for me not to appear nervous.	0	1	2	3	4
2. When I cannot keep my mind on a task, I worry that I might be going crazy.	0	1	2	3	4
3. It scares me when my heart beats rapidly.	0	1	2	3	4
4. When my stomach is upset, I worry that I might be seriously ill.	0	1	2	3	4
5. It scares me when I am unable to keep my mind on a task.	0	1	2	3	4
6. When I tremble in the presence of others, I fear what people might think of me.	0	1	2	3	4
7. When my chest feels tight, I get scared that I won't be able to breathe properly.	0	1	2	3	4
8. When I feel pain in my chest, I worry that I'm going to have a heart attack.	0	1	2	3	4
9. I worry that other people will notice my anxiety.	0	1	2	3	4
10. When I feel "spacey" or spaced out I worry that I may be mentally ill.	0	1	2	3	4
11. It scares me when I blush in front of people.	0	1	2	3	4
12. When I notice my heart skipping a beat, I worry that there is something seriously wrong with me.	0	1	2	3	4
13. When I begin to sweat in a social situation, I fear people will think negatively of me.	0	1	2	3	4
14. When my thoughts seem to speed up, I worry that I might be going crazy.	0	1	2	3	4
15. When my throat feels tight, I worry that I could choke to death.	0	1	2	3	4
16. When I have trouble thinking clearly, I worry that there is something wrong with me.	0	1	2	3	4
17. I think it would be horrible for me to faint in public.	0	1	2	3	4
18. When my mind goes blank, I worry there is something terribly wrong with me.	0	1	2	3	4

Appendix F

Lectures/Deaprtments attended for participant recruitment

1. History
2. Sociology
3. Psychology
4. Social Work
5. Chemistry
6. Geography
7. Law
8. Computer Science
9. English
10. Engineering

Departments that refused participation or had no reply from

1. Economics
2. School of management
3. Medical school

Appendix G

Standardised 'spiel' for study introduction in lectures/ EPR

Good morning/afternoon, my name is David Ward and I am a trainee clinical psychologist at Leicester university. I am conducting my Doctoral thesis research in the area of cannabis use, tobacco smoking and panic attacks. I have some questionnaires that I would be grateful if you would complete for me. The estimated time taken to complete the questionnaires is between 15-35 minutes, based on a pilot study. The data gathered from the questionnaires is confidential and anonymous. Participation in this study is, of course, entirely voluntary. If you do not wish to participate then please do not take a questionnaire.

If at any time during the completion of the questionnaire pack you decide you no longer wish to participate then please dispose of the questionnaire appropriately.

If you do decide to participate then please either pass the questionnaire directly to me once you have completed it or if you are taking it away to complete later then please return it into one of the marked 'drop-boxes' located at

1. Attenborough tower – 3rd floor
2. Bennett building – lower floor outside lecture theatres
3. Henry welcome building – outside the psychology general office

There is a **prize draw** for participants with prizes of **50, 30 and 20 pounds cash!**

Again entry into the prize draw is entirely voluntary, however in order to be contacted of your win (should you be a lucky winner) you will need to leave an email address. To help ensure your anonymity it might be best to use an address that is innocuous, not one with your full name. Email addresses will be stored on a password protected file on my personal computer (which has full internet firewall and virus security). This file will be deleted as soon as all data is collected and winners notified. Winners will be chosen at random.

If you have any questions please ask now or email me later at djdw2@le.ac.uk.

Thank you for your time

David Ward

Appendix H

From: McKie Jeannie - - NCTPCT [Jeannie.McKie@nottspct.nhs.uk]
Sent: 20 October 2008 09:33
To: djdw2@leicester.ac.uk
Subject: FW: For Chair's approval

Dear David,

Please could you take a look at Dr Edward's comments below and discuss them with your supervisor. If he insists on REC review please contact me again. Alternatively I can issue you a letter confirming that the protocol has been reviewed by the Chair and REC approval is not required.

Kind Regards
Jeannie McKie
Committee Coordinator
Leicestershire, Northamptonshire and Rutland Research Ethics Committee 1 & 2
1 Standard Court
Park Row
Nottingham, NG1 6GN
Tel: 0115 9123344 ext 39428
Fax: 0115 9123300
www.nres.npsa.nhs.uk

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From: Carl Edwards [mailto:carledwards101@hotmail.com]
Sent: 16 October 2008 07:59
To: McKie Jeannie - - NCTPCT; anne.edwards@em-nhs-hub.org
Subject: RE: For Chair's approval

Hi Jeannie,

this really does fall out of the remit of the REC I think.

Whilst in principle I haven't a problem with him coming for an opinion to the REC I think his supervisor needs to reconsider the ethical scrutiny that this project requires? It's an anonymous questionnaire survey and whilst the subject may be contentious the participants are guaranteed anonymity and the protocol does address some of the potential Institutional effects of the research but this wouldn't normally be a consideration for the REC.

Given that the RECs are currently very busy I think that it's best dealt with by the usual Leicester University Departmental ethics committee? I can have a word with his supervisor if he wants?

atb,

Carl

Appendix I

Dear Mr. David Ward,

Your project "Exploring the relationship between cannabis use, tobacco smoking and panic attacks" has been approved by the Psychology Research Ethics Committee.

This e-mail is the official document of ethical approval and should be printed out and kept for your records or attached to the research report if required - this includes all undergraduate and postgraduate research. This approval is valid for three years. For research projects lasting more than one year a yearly statement must be sent to the Chair of the Psychology Research Ethics Committee confirming that the research project has not been changed.

We wish you every success with your study.

Carlo De Lillo
Psychology Research Ethics Committee Chair

Dr. Carlo De Lillo
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School of Psychology
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Tel. +44-0116-229-7193
Fax +44-0116-229 7196
E-mail cd12@le.ac.uk
Web-page: <http://www.le.ac.uk/pc/cd12/>

Appendix J

PLEASE TEAR OFF AND TAKE THIS SHEET WITH YOU IF YOU WISH

Drug and Panic Information

If you have any concerns about drug use and would like more information or help please make use of the information below:

The 'talktofrank' service give information about drugs and alcohol and what services are on offer in your area to help you.

www.talktofrank.com PHONE 0800 77 66 00

If you have concerns about excessive anxiety, panic or other mental health problems please use the information provided below:

Mental Health Care

Comprehensive information about mental illnesses.

Email: subscribe@mentalhealth.org.uk

Website: www.mentalhealthcare.org.uk

Mind

Offers many services including helplines, drop-in centres, supported housing, counselling, befriending, advocacy, employment and training schemes.

Information line: 0845 766 0163

Email: contact@mind.org.uk

Website: www.mind.org.uk

Rethink

Support and advice for everyone affected by severe mental illness.

General enquiries: 0845 456 0455

National advice service: 020 8974 6814

Email: info@rethink.org

Website: www.rethink.org

Samaritans

Provides 24-hour, confidential emotional support to any person who is suicidal or despairing.

UK helpline: 08457 909090

ROI helpline: 1850 609090

Email: jo@samaritans.org

Website: www.samaritans.org.uk

No Panic

Help for people with panic attacks, phobias, obsessive-compulsive disorders and general anxiety disorders.

Helpline: 0808 808 0545

Email: ceo@nopanic.org.uk

Website: www.nopanic.org.uk

AnxietyUK (formerly the National Phobics Society)

A user-led organisation dealing with anxiety disorders.

Tel: 08444 775 774

Email: info@anxietyuk.org.uk

Website: www.anxietyuk.org.uk

Appendix K

Explanation of Cox regression survival analysis (taken from Norušis, 2008)

Cox regression (or proportional hazards regression) is method for investigating the effect of several variables upon the time a specified event takes to happen. In the context of an outcome such as death this is known as Cox regression for survival analysis. Cox regression is used to determine the relationship between survival and several independent explanatory variables. Cox regression is useful for modelling the time to a specific event based upon the value of a given covariate. Survival analysis is a method of analyzing whether or not an event will happen. Cox regression provides an estimate of the treatment on the survival rate, after adjustment of the explanatory variable. In Cox regression, we estimate the coefficient of the explanatory variable. The basic model for Cox regression produces the proportional hazard function, which can be extended through the specifications of a strata variable or time-dependent covariates.

Unlike ordinary regression models Cox regression models can be used when there are observations for whom the event has not occurred. If you want to evaluate the effect of one or more covariates on time to an event, this is the procedure of choice.

Hazard function

Hazard is the event of interests occurring. For example, in medical research usually death is a hazard. Another example would be in the onset of a particular disease. In other words, we can say that the probability of the endpoint of an event of interest is called the hazard. Hazard function is also known as the Cox proportional hazard function. A hazard ratio is essentially an odds ratio, in that it represents the probability of an event occurring in time.

Status variable

In Cox regression, the status variable is the dependent variable. In Cox regression, the status variable is binary in nature. For example, we will assign code 1 for events that happen, and 0 for events that do not happen.

Time variable

In Cox regression, the time variable measures the duration of the status variable. Time variable is simply the counter unit of time since the series started.

Covariate

In Cox regression, covariates are the independent variables. In Cox regression, covariates can be categorical or a dummy.

Hazard In Cox regression, hazard is the event of interest occurring.

Hazard rate or hazard ratio

In Cox regression, hazard ratio is also called the odd ratio. Hazard ratio is the probability of events happening in time $t+1$.

References

Norušis, M. (2008). *SPSS statistics 17.0: advanced statistical procedures companion*. Pearson Education. New York.