HUMAN PSYCHOPHARMACOLOGY
Hum. Psychopharmacol Clin Exp 2008; **23**: 475–485
Published online 30 April 2008 in Wiley InterScience
(www.interscience.wiley.com) **DOI**: 10.1002/hup.950

Polydrug use, cannabis, and psychosis-like symptoms

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Objective To examine psychosis-like symptoms in users of legal and illicit drugs.
Methods Schizotypal Personality Questionnaire (SPQ) scores were compared in groups of people with different exposure to cannabis, with the use of other drugs serving as a covariate. Supplemental analyses compared users of legal and illicit drugs with cannabis use as a covariate.
Results Weekly ($n = 111$) and monthly ($n = 136$) cannabis users had higher scores on the SPQ than former ($n = 143$) and non-users ($n = 81$). The use of other drugs accounted for the links between cannabis and schizotypy. Lifetime use of psychomotor stimulant drugs plus ecstasy accounted for associations between cannabis and scores on the SPQ and its different subscales. Dividing groups by type of drug use revealed that those who used only cannabis and legal drugs (CLDs) ($n = 126$) were no different from those who used only legal drugs (LDs) ($n = 74$) but both groups scored significantly lower on the SPQ than polydrug users ($n = 247$). When controlling for marijuana use in the last month, the significant difference across drug use groups remained.
Conclusions The results suggest that research on marijuana and schizotypy requires careful assessment of the use of other drugs, especially psychomotor stimulants and ecstasy. Copyright © 2008 John Wiley & Sons, Ltd.

KEY WORDS — cannabis; psychosis; Schizotypal Personality Questionnaire; schizotypy

INTRODUCTION

The precipitation hypothesis suggests that cannabis might contribute to psychosis, especially in individuals who are predisposed (Hall, 2006; Hall *et al*., 2004; Johns, 2001; Macleod *et al*., 2007; Schuckit, 2006; Zullino *et al*., 2007). The strength of this contribution remains controversial (Arsenault *et al*., 2004; Moore *et al*., 2007; Smit *et al*., 2004). Moore *et al*. (2007) recently conducted a systematic review and suggest that cannabis increases psychotic symptomatology, but Macleod *et al*. (2007) counter that their review does not indicate causality and may suffer from small-study bias, given only seven different studies. Zammit *et al*. (2007) admit ''For any individual, use of cannabis is quite unlikely to lead to psychotic illness, even if the relation is causal.'' Without considering total drug use, any assessment of the contributions of cannabis to psychosis is marred with methodological limitations. Most of the studies reviewed by Moore *et al*. (2007) controlled for total drug use but failed to examine individual drugs or drug classes. One potential drug class is the psychomotor stimulants (e.g., cocaine, crack, amphetamine, methamphetamine). These drugs increase dopamine activity, potentially increasing the risk of psychosis (Berry *et al*., 2003). The seven studies included in the review also varied in their assessment of psychotic symptoms. Given the range of symptom severity, infrequency of schizophrenia, and the complications associated with severe psychotic symptomatology, examinations of early psychosis-like symptoms, rather than full-blown schizophrenia, can prove heuristic. Therefore, a particularly salient area of research may involve prodromal, or less severe phases of psychosis.

Schizotypy: a potential prodrome to schizophrenia

Schizotypal personality disorder (SPD) has consistently shown an etiological relation to the schizophrenia spectrum (Livesley, 2001; Raine, 2006).

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Received 10 December 2007
Accepted 19 March 2008

Approximately 40% of individuals with SPD progress to schizophrenia over 15 years (Fenton and McGlashan, 1989), and more than 40% of prodromal schizophrenics with SPD-like symptoms have a psychotic break within a year (Yung *et al*., 2003). In addition, the heritability of SPD (and its traits) parallels that of Schizophrenia (Meehl, 1990; Raine, 2006; Raine and Benishay, 1995; Vollema *et al*., 2002). There is an important clinical relationship between schizotypal signs and psychosis, especially in the link between psychosis and cannabis (Verdoux *et al*., 2002).

The Schizotypal Personality Questionnaire (SPQ; Raine, 1991) is not intended to diagnose SPD, but indicates schizotypal symptoms. Fifty-five per cent of individuals scoring in the top 10% of the SPQ have a clinical diagnosis of SPD as assessed by the SCID (Raine, 1991), suggesting a base rate 5.5% of SPD, which is quite comparable to the DSM-IV-TR's estimate of about 3% (American Psychiatric Association, 2000).

Schizotypy and cannabis

Cannabis administration can cause psychosis-like experiences (Barkus *et al*., 2006) and cannabis users report more psychotic symptoms (Bailey and Swallow, 2004; Dumas *et al*., 2002; Mass *et al*., 2001). The purported link between cannabis use and schizotypal traits could arise for multiple reasons. Symptoms of schizotypy may predispose individuals to use cannabis or cannabis may lead to the development of schizotypy or some third variable might account for both. Subjects cannot be randomly assigned to use cannabis, so correlates of cannabis use may account for its link with schizotypy. Raine (2006) suggests, ''some schizotypals may use drugs to self-medicate their symptoms and social impairment.''

Schizotypy and other drugs

One generally unexplored correlate of cannabis that may also relate to schizotypal symptoms is the use of other drugs. Alcohol alone and in combination with cannabis can predict some symptoms of schizotypy (Larrison *et al*., 1999; Nunn *et al*., 2001). Nicotine and caffeine also relate to schizotypy (Larrison *et al*., 1999). Although the vast majority of people who try cannabis do not use hard drugs (see Earleywine, 2002), important subsets of cannabis users try cocaine, amphetamines, hallucinogens, and others. Given the role of the dopamine system in the development of psychotic disorders (Berry *et al*., 2003), examination of other drugs, particularly those known to affect the dopamine system (Gardner, 2005; Robbins and Everitt, 1999; Volkow, 2005), could help explain the link between cannabis use and psychotic symptomatology. Some research on cannabis and schizophrenia assesses and controls for the use of other drugs, but often individual drugs or groups of drugs have not been examined (e.g., Arsenault *et al*., 2002; Henquet *et al*., 2005; van Os *et al*., 2002). Many studies of cannabis and schizotypal characteristics do not control for other drug use at all (Bailey and Swallow, 2004; Dumas *et al*., 2002; Schiffman *et al*., 2005).

The SPQ (Raine, 1991) and its short form (SPQ-B; Raine and Benishay, 1995) are the most commonly used questionnaires that assess symptoms of SPD. At least five previous studies reveal links between cannabis consumption and responses to the SPQ or SPQ-B, but only two of these studies control for the potential confound of other drugs, and they have very small samples. Skosnik *et al*. (2001) found large differences ($d = 1.8$) in SPQ scores across small groups ($n \leq 15$) with varying cannabis exposure (current users, former users, never used), while excluding users of other drugs. Mass *et al*. (2001) report significant differences between cannabis users ($n = 20$) and non-users ($n = 20$) for only the Eccentric Behavior sub-factor of the SPQ. These researchers report excluding on the basis of other drug use and report no differences in alcohol use between groups. Dumas *et al*. (2002) report medium ($d = 0.4$–0.6) effects on the full SPQ in a larger sample ($n = 232$) of current, former, and never using undergraduates. They report significantly increased schizotypy as cannabis use increases for SPQ total and four of the SPQ's nine sub-factors. Bailey and Swallow (2004) found a large difference ($d = 1.2$) between undergraduate cannabis users ($n = 30$) and non-users ($n = 30$). Schiffman *et al*. (2005) found medium ($d = 0.4$) effects on the Cognitive Perceptual (CP) and Disorganized (D) subscales of the SPQ-B, but not of the total SPQ-B in a sample of 189 undergraduate students. These last three studies, two of which have some of the largest sample sizes of an examination of SPQ scores and cannabis use to our knowledge, fail to control for the use of other drugs.

Current study

The current study examines responses on the SPQ in current, former, and non-users of cannabis in a large

DOI: 10.1002/hup

internet sample, while also assessing the potential contributions of other drugs to the differences between subsets of cannabis users and non-users. The study examines the relationship between frequency of cannabis use and symptoms of schizotypy. In addition, we examined SPQ scores across drug use groups (legal drug (LD) only, cannabis and legal drug (CLD) only, and cannabis polydrug (CPD) use).

METHOD

Procedure

Participants responded to an e-mail request to complete an internet survey on cannabis use and attitudes. To target potential cannabis users, the initial e-mail was sent to members of a class on drug effects at a university in upstate New York. The e-mail stated that participants could complete the internet questionnaire and forward it to others (the ''snowballing'' technique; Callow, 1996), for a chance to win a $100 gift certificate to Amazon.com. Participants had the option to enter their e-mail address if they wanted to be eligible for the prize. We downloaded e-mail addresses with data, but immediately placed them in a file separate from the data to ensure anonymity. The first online page of the study stated that continued participation beyond the first page implied participant consent to the experiment. All procedures were in accordance with and approved by the local Investigational Review Board.

Data screening

The internet survey model provides potential advantages and disadvantages over other studies on this topic, most of which concern sampling and response bias. At least two recent studies provide rationale and support for the internet method in examining illicit drug use (Rodgers *et al*., 2001; Rodgers *et al*., 2003). We considered fraudulent data entry (due to lack of concern or impairment) and duplicate individual responses as the largest threats to the integrity of the data (Rodgers *et al*., 2001). We carefully screened the data for inconsistencies in responses and duplicate data sets. Overall, 36 individuals were removed for inconsistency or what was perceived as inauthentic response. Individuals were removed for lack of consent ($n = 1$), inconsistency in self-report of alcohol use ($n = 11$), inconsistency in self-report of cannabis use ($n = 19$), and an unrealistic self-report of first age of cannabis use ($n = 5$). Inconsistencies in alcohol or cannabis use included reporting never using, and then subsequently giving a number of uses per week, month, year, lifetime, etc., and reporting a weekly, monthly, yearly number of uses that was higher than monthly, yearly, or lifetime uses (respectively). Unrealistic self-report of first age of cannabis use consisted of individuals who stated an age of less than 5 years old. Earliest episodic memories are recalled on average between 3 and 5 years of age (Mullen, 1994). After eliminating erroneous responses, we screened for duplicate cases; none were evident. Although screening data by Interpersonal (IP) address is common (Rodgers *et al*., 2001), the current study did not employ this method because of the sample. Preventing duplicate responses from a single IP address would have limited students using campus computers or friend's computers that had already been involved in the study.

Participants

To maximize the potential to elucidate interactive effects between cannabis and other drugs, we performed two sets of primary group analyses. Both of these analyses were equally important as they provided unique information about the effects of cannabis frequency and type of drug use (respectively) on psychosis-like symptoms. The first group analysis had four groups based on cannabis use frequency. These groups included: non-users ($n = 81$), former users ($n = 143$), monthly users ($n = 136$), and weekly users ($n = 111$). Across cannabis use groups, participants were primarily female (67%), with an average age of 28.7 years (SD = 10.5). The average age of first cannabis use was 16.4 years (SD = 3.2). The group was primarily Caucasian (83%), with African American (4.7%), other (4.3%), Asian (4.1%), Hispanic (3.2%), and Native American (0.6%) far behind. The sample was also well educated (87.8% reported education beyond high school; 34.1% were college graduates; 31.1% had at least some graduate education; and 11.5% held advanced degrees).

To examine potential confounds arising from other drug use, the second analytic strategy divided subjects by type of drugs used, an approach that is gaining favor (e.g., Bedi and Redman, 2008). Respondents were grouped based on reported lifetime drug use: LD only users ($n = 74$), CLD only users ($n = 126$), and CPD users ($n = 247$). Polydrug use was defined as the use of at least one or more illicit substance other than cannabis.

DOI: 10.1002/hup

MEASURES

Schizotypy

Participants completed the 74 True/False item version of the SPQ (Raine, 1991). The SPQ has excellent psychometric properties, addresses symptoms of SPD (Mata *et al*., 2005; Reynolds *et al*., 2001), and covaries with genetic vulnerability to schizophrenia (Vollema *et al*., 2002). The factor structure of the SPQ has proven stable across gender, ethnicity, religion, and social groups (Reynolds *et al*., 2001) and has been extensively tested across a range of community and clinical samples (Raine, 2006).

Instructions stated explicitly for participants not to include any drug-induced or drug-related experiences in their answers, a precaution not mentioned in previous work. Administration of the full SPQ permitted examination of two different subscale classifications: the Raine (1991) subscales and the Vollema and Hoijtink (2000) subscales, components of each showing specific relationships to psychosis (Siever, 1995; Vollema *et al*., 2002).

Raine subscales

Raine (1991) divided the SPQ into three major subscales: CP, IP, and D. The CP subscale includes 33 items that focus on magical thinking and paranoid ideation. Sample items include ''Are you sometimes sure that other people can tell what you are thinking?'' and ''Do you often have to keep an eye out to stop people from taking advantage of you?'' The IP subscale includes 33 items that focus primarily on social anxiety and discomfort. Sample items include ''I feel very uneasy talking to people I do not know well,'' and ''I tend to keep in the background on social occasions.'' The D subscale includes 16 items that assess odd or unusual behavior. Sample items include, ''Some people think that I am a very bizarre person,'' and ''People sometimes comment on my unusual mannerisms and habits.'' The CP subscale of the Raine (1991) model accounts for dopaminergic increases observed in SPD (Siever, 1995) and has strong face validity with regard to psychotic features (Raine, 2006). In addition, non-psychotic relatives of schizophrenics reporting increased scores relative to controls on the IP and D subscales (Raine, 2006).

Vollema and Hoijtink subscales

Vollema and Hoijtink (2000) also divided the SPQ into three major subscales comparable to facets of schizophrenia. The subscales include Positive Symptoms, Negative Symptoms, and Disorganized Symptoms. Individual items can receive a score of 0, 1, or 2 based on the strength of each items representativeness of the overall scale. Items in the Positive Symptom subscale represent positive symptoms of schizophrenia and include items such as ''I often hear a voice speaking my thoughts aloud,'' and ''Have you ever seen things invisible to other people?'' Total scores can range from 0 to 38. The Negative Symptoms subscale reflects negative symptoms of schizophrenia. It includes items such as, ''Some people think that I am a very bizarre person,'' and ''I attach little importance to having close friends.'' Scores range from 0 to 44. The Disorganized Symptoms subscale reflects disorganized thoughts and behaviors and includes items such as, ''I sometimes jump quickly from one topic to another when speaking,'' and ''Do you ever suddenly feel distracted by distant sounds that you are not normally aware of?'' This subscale has scores that range from 0 to 19.

The Positive Symptoms subscale of the Vollema and Hoijtink (2000) model varies with genetic vulnerability to schizophrenia (Vollema *et al*., 2002) and has strong face validity as a measure of psychotic features. In addition, non-psychotic relatives of schizophrenics have higher scores on the Negative Symptoms and D subscales.

Cannabis consumption

Participants answered ''yes'' or ''no'' to the question ''Have you used marijuana (cannabis, pot, weed, grass) in your lifetime?'' Those who answered ''yes'' were asked about use in the past year and frequency of use per month and week. The number of days marijuana was used per month (from 0 to 31) served as a covariate in the second primary analyses comparing across drug use groups. Individuals who reported no lifetime use composed the non-user group; individuals who reported lifetime use but not in the past year composed the former users group; individuals who reported at least once a month but fewer than four times a month composed the monthly users group; and the weekly users group included individuals who reported using four times or more a month and at least one time per week.

Legal drug use

Participants responded ''yes'' or ''no'' with regards to lifetime use and use within the last year, as well as estimating the number of days of drinking within the

DOI: 10.1002/hup

last month. A standard drink was defined as a 12 oz beer, a 5 oz glass of wine, or a 1.5 oz shot of 80 proof liquors. Participants also responded ''yes'' or ''no'' to questions concerning lifetime use, and monthly use for nicotine and caffeine.

Illicit drug use

Participants answered ''yes'' or ''no'' to questions concerning last month and lifetime use of nine illicit drugs: cocaine, crack, amphetamine, methamphetamine, heroin, LSD, ecstasy, mushrooms, and inhalants. The first four were labeled ''psychomotor stimulants'' due to the direct activity of cocaine and amphetamine on the mesocorticolimbic dopamine system (Everitt and Wolf, 2002) and the close chemical relationship between crack and methamphetime to cocaine and amphetamine, respectively. Given the relationship between MDMA and dopamine biochemistry (Colado *et al*., 2004) and that ''ecstasy'' tablets have been noted to contain stimulants like amphetamine and methamphetamine (Cole and Sumnall, 2003; Parrott, 2004), lifetime ecstasy use was considered an adjunct to the psychomotor stimulants. Ecstasy use was not directly included as a psychomotor stimulant due to the primary selective serotonergic action of MDMA (Colado *et al*., 2004). Although a lifetime measure of multiple drugs only provides a rough estimate of involvement, any findings with a measure this broad would suggest that further work with more detailed and expensive assessments could prove useful.

Statistical analysis

We investigated differences in demographics across cannabis groups and across drug use groups in outcome variables with chi-square tests of independence and analyses of variance (ANOVA). Demographic variables that significantly differed across cannabis group or drug use group were included in respective ANCOVA analyses to determine if they significantly altered results. We also used ANCOVA analyses to determine the impact of other drug use as potentially contributing to the relation between cannabis use and schizotypy scores. Differences in SPQ scores across drug use groups were compared using ANOVA. In consideration of the likelihood that individuals in the CPD group use more cannabis than individuals in the CLD group (Milani *et al*., 2005; Parrott *et al*., 2001), we compared cannabis use reported for the last month across these two groups using ANCOVA analyses. Effect size estimates were computed using Cohen's *d*. Bonferroni corrections were used when appropriate. All analyses were performed using SPSS 15.0.

RESULTS

Demographics

Overall illicit substance use. Total illicit drug use in the last month ranged from 0 to 4 (Median = 0, M = 0.15, SD = 0.48). The percentage of monthly illicit drug use was as follows: marijuana 48.1%, cocaine 6.1%, amphetamine 2%, mushrooms 1.6%, ecstasy 1.2%, inhalants 0.8%, heroin 0.7%, methamphetamine 0.5%, LSD 0.3%, crack 0.3%. Total lifetime illicit drug use ranged from 0 to 9 (Median = 1, M = 2.0, SD = 2.4). The percentage of lifetime illicit drug use was: marijuana 82.8%, cocaine 29.8%, mushrooms 28.0%, ecstasy 23.6%, LSD 21.6%, amphetamine 18.9%, inhalants 14.5%, methamphetamine 7.2%, crack 6.6%, heroin 6.1%.

To examine potential current drug-induced psychosis, we divided the sample into two groups according to likelihood of having SPD. Individuals scoring in the top 10% of scores on the SPQ comprised one group (having a 55% likelihood of SPD as reported by Raine, 1991), and the rest of the sample comprised the other group. An ANOVA of illicit drug use in the last month revealed no significant differences between those individuals having a higher likelihood of SPD than the rest of the group, $p > 0.3$.

Cannabis use groups. Cannabis groups did not differ on gender or ethnicity. There was, however, a significant difference between groups in age, $F(3, 466) = 11.36$, $p < 0.001$, and in education, $\chi^2(6, 471) = 20.71$, $p < 0.01$. Neither age nor education served as a significant covariate with cannabis group in SPQ total scores ($p > 0.2$).

Drug use groups. Drug use groups did not differ in gender, race, or education (LD only, CLD only, CPD), but they did differ in age, $F(2, 441) = 5.58$, $p < 0.02$. *Post-hoc* LSD comparisons with Bonferroni corrections revealed differences between the LD group (M = 30.6, SD = 12.5) and CLD group (M = 26.2, SD = 8.4) and the CLD group and CPD group (M = 29.4, SD = 10.5), but not the LD and CPD groups. Since age did not covary with schizotypy ($r = -0.07$), it was not included as a covariate.

DOI: 10.1002/hup

Table 1. Means, standard deviation, and effect size differences across groups for the Raine (1991) model

Scale	1 Weekly ($n = 111$)	2 Monthly ($n = 136$)	3 Former ($n = 143$)	4 Non-users ($n = 81$)	Largest between group d	*Post-hoc*[a]
SPQ	25.9 (13.9)	23.4 (13.5)	21.4 (14.4)	18.3 (12.4)	0.43[b]	1,2 > 4
CP	11.23 (7.29)	9.74 (6.75)	8.33 (7.06)	7.44 (6.47)	0.42[b]	1 > 3,4
IP	10.77 (7.01)	10.30 (7.10)	10.38 (7.98)	8.65 (6.21)	0.31	n.s.
D	6.46 (4.24)	5.81 (4.37)	4.86 (3.97)	4.14 (3.62)	0.44[c]	1,2 > 4, 1 > 3

SPQ, total score for Schizotypal Personality Questionnaire; CP, Cognitive Perceptual subscale of SPQ; IP, Interpersonal subscale of SPQ; D, Disorganized subscale of SPQ.
[a]Only significant differences are reported according to LSD *post-hoc* comparisons with Bonferroni corrections to set the p-value equivalent to $p < 0.05$ ($p = 0.008$); [b]$p < 0.01$; [c]$p < 0.001$.

Cannabis group schizotypy scores

Schizotypal Personality Questionnaire total score. Internal consistency for the full scale was very high (Cronbach's $\alpha = 0.95$). Cannabis use groups differed on SPQ total scores, $F(3.\ 470) = 5.36$, $p = 0.001$. Details appear in Table 1. Use of caffeine, nicotine, or alcohol did not serve as significant covariates, all $p > 0.4$. Illicit drug use within the last month (both for psychomotor stimulants alone and for all illicit drugs) did not serve as significant covariates in schizotypy scores across cannabis groups. Lifetime psychomotor stimulant use (cocaine, crack, amphetamine, methamphetamine) served as a significant covariate, $F(1,\ 450) = 6.31$, $p < 0.05$, and eliminated the effect of cannabis use, $F(3,\ 450) = 2.40$, $p = 0.07$.

Raine subscales

Cognitive Perceptual subscale. Internal consistency (Cronbach's α) was 0.91. The cannabis groups differed significantly on this subscale, $F(3,\ 470) = 5.93$, $p = 0.001$. Details appear in Table 1. Lifetime psychomotor stimulant use served as a significant covariate, $F(1,\ 450) = 6.71$, $p < 0.05$, and reduced but did not eliminate the difference between cannabis groups, $F(3,\ 450) = 3.18$, $p = 0.02$. Nevertheless, lifetime psychomotor stimulant plus ecstasy use served as a significant covariate, $F(1,446) = 7.35$, $p < 0.01$, and eliminated the difference between cannabis groups, $F(3,446) = 2.37$, $p = 0.07$.

Interpersonal subscale. Internal consistency (Cronbach's α) was 0.92. The cannabis groups did not differ on the IP subscale, $F(3,\ 470) = 1.50$, $p > 0.2$.

Disorganized subscale. Internal consistency (Cronbach's α) was 0.87. The cannabis groups differed significantly on the D subscale, $F(3,\ 470) = 6.32$, $p < 0.001$. Details appear in Table 1. Lifetime psychomotor stimulant use served as a significant covariate, $F(1,\ 450) = 4.58$, $p < 0.05$, and reduced, but did not eliminate the cannabis group effect, $F(3,\ 450) = 3.65$, $p = 0.01$. While lifetime psychomotor stimulant plus ecstasy use served as a significant covariate, $F(1,446) = 4.88$, $p < 0.05$, it did not eliminate the cannabis group effect, $F(1,446) = 3.17$, $p = 0.02$. When all lifetime illicit drug use was examined, it served as a significant covariate, $F(1,\ 450) = 7.81$, $p < 0.01$, and eliminated the cannabis group effect, $F(3,\ 450) = 2.53$, $p = 0.06$.

Vollema and Hoijtink subscales

Positive Symptoms subscale. Internal consistency (Cronbach's α) was 0.91. The cannabis groups differed significantly on the Positive Symptom subscale, $F(3,\ 470) = 6.39$, $p < 0.001$. Details appear in Table 2. Lifetime psychomotor stimulant use again served as a significant covariate, $F(1,\ 450) = 6.71$, $p < 0.05$, reducing but not eliminating the cannabis group effect, $F(3,\ 450) = 3.18$, $p = 0.02$. Lifetime psychomotor stimulant plus ecstasy use again served as a significant covariate, $F(1,\ 446) = 7.99$, $p < 0.01$, eliminating the cannabis group effect, $F(3,\ 446) = 2.59$, $p = 0.05$.

Negative Symptoms subscale. Internal consistency (Cronbach's α) was 0.92. The cannabis groups did not differ significantly on the Negative Symptoms subscale, $F(3,\ 470) = 1.77$, $p > 0.1$.

Disorganized subscale. Internal consistency (Cronbach's α) was 0.85. Cannabis groups differed significantly on the D subscale, $F(3,\ 470) = 5.12$, $p = 0.002$. Details appear in Table 2. Psychomotor stimulant use served as a significant covariate, $F(1,\ 450) = 4.22$, $p < 0.05$, that reduced, but did not eliminate the cannabis group effect, $F(3,\ 450) = 2.86$, $p = 0.036$. In contrast to the D subscale of Raine's (1991) model, here lifetime psychomotor

DOI: 10.1002/hup

Table 2. Means, standard deviation, and effect size differences across groups for the Vollema and Hoijtink (2000) model

Scale	1 Weekly ($n = 111$)	2 Monthly ($n = 136$)	3 Former ($n = 143$)	4 Non-users ($n = 81$)	Largest between group d	*Post-hoc*[a]
SPQ	25.9 (13.9)	23.4 (13.5)	21.4 (14.4)	18.3 (12.4)	0.43[b]	1,2 > 4
Pos Sx	12.39 (8.34)	10.49 (7.52)	8.91 (7.90)	7.99 (7.24)	0.44[c]	1 > 3,4
Neg Sx	14.36 (8.99)	13.63 (8.99)	13.27 (10.10)	11.35 (8.16)	0.31	n.s.
Disorg	7.63 (5.02)	7.02 (5.26)	5.94 (4.81)	5.16 (4.18)	0.41[b]	1,2 > 4, 1 > 3

SPQ, total score for Schizotypal Personality Questionnaire; Pos Sx, Positive Symptom subscale of SPQ; Neg Sx, Negative Symptom subscale of SPQ; Disorg, Disorganized subscale of SPQ.
[a]Only significant differences are reported according to LSD *post-hoc* comparisons with Bonferroni corrections to set the p-value equivalent to $p < 0.05$ ($p = 0.008$); [b]$p < 0.01$; [c]$p < 0.001$.

stimulant plus ecstasy use served as a significant covariate, $F(1, 446) = 4.35$, $p < 0.05$, that eliminated the cannabis group effect, $F(3, 446) = 2.50$, $p = 0.06$.

Drug use group schizotypy scores. Drug use groups (LD only, CLDs only, CPD use) differed in total schizotypy scores and all of the subscales. Details appear in Tables 3 and 4. LSD *post-hoc* analyses using Bonferroni corrections indicated no significant differences in schizotypy scores between the LD and CLD groups, but significant differences in schizotypy scores between the CPD and LD, and CPD and CLD groups.

Groups differed in monthly cannabis use, with members of the CPD group ($M = 8.1$, $SD = 11.5$) reporting more cannabis use per month than members of the CLD group ($M = 2.2$, $SD = 5.6$), $F(1, 366) = 28.72$, $p < 0.001$. This finding is consistent with previous work that indicates that polydrug users use more cannabis (e.g., Milani *et al*., 2005; Parrott

Table 3. Means, standard deviation, and effect size differences across drug use groups for the Raine (1991) model

Scale	1 LD ($n = 74$)	2 CLD ($n = 126$)	3 CPD ($n = 247$)	Largest between group d	*Post-hoc*[a]
Mo. THC use	—	2.2 (5.6)	8.1 (11.5)[b]	0.65	—
SPQ	17.9 (12.2)	19.9 (12.4)	25.2 (14.7)[c]	0.54	3 > 1,2
CP	7.1 (6.4)	8.1 (6.0)	10.4 (7.6)[c]	0.47	3 > 1,2
IP	8.7 (6.2)	9.5 (6.9)	11.0 (7.7)[b]	0.33	n.s.
D	4.1 (3.7)	4.5 (3.8)	6.3 (4.3)[c]	0.55	3 > 1,2

LD, legal drug group; CLD, cannabis legal drug group; CPD, cannabis polydrug group; SPQ, total score for Schizotypal Personality Questionnaire; CP, Cognitive Perceptual subscale of SPQ; IP, Interpersonal subscale of SPQ; D, Disorganized subscale of SPQ.
[a]Only significant differences are reported according to LSD *post-hoc* comparisons with Bonferroni corrections to set the p-value equivalent to $p < 0.05$ ($p = 0.017$); [b]$p < 0.05$; [c]$p < 0.001$.

Table 4. Means, standard deviation, and effect size differences across drug use groups for the Vollema and Hoijtink (2000) model

Scale	1 LD ($n = 74$)	2 CLD ($n = 126$)	3 CPD ($n = 247$)	Largest between group d	*Post-hoc*[a]
Mo. THC use	—	2.2 (5.6)	8.1 (11.5)[b]	0.65	—
SPQ	17.9 (12.2)	19.9 (12.4)	25.2 (14.7)[c]	0.54	3 > 1,2
Pos Sx	7.6 (7.1)	8.6 (6.6)	11.4 (8.6)[c]	0.48	3 > 1,2
Neg Sx	11.2 (8.1)	12.4 (8.8)	14.4 (9.8)[d]	0.36	n.s.
Disorg	5.2 (4.3)	5.6 (4.6)	7.5 (5.2)[c]	0.48	3 > 1,2

LD, legal drug group; CLD, cannabis legal drug group; CPD, cannabis polydrug group; SPQ, total score for Schizotypal Personality Questionnaire; Pos Sx, Positive Symptom subscale of SPQ; Neg Sx, Negative Symptom subscale of SPQ; Disorganized, Disorganized subscale of SPQ.
[a]Only significant differences are reported according to LSD *post-hoc* comparisons with Bonferroni corrections to set the p-value equivalent to $p < 0.05$ ($p = 0.017$); [b]$p < 0.05$; [c]$p < 0.001$; [d]$p < 0.01$.

DOI: 10.1002/hup

et al., 2001). Nevertheless, reported monthly cannabis use did not serve as a significant covariate in CLD versus CPD comparisons for all schizotypal measures ($p > 0.1$) with the exception of the CP and Positive Symptoms subscales. When monthly cannabis use was included as a covariate in CP symptoms, $F(1, 367) = 7.72$, $p < 0.01$, CPD group members ($M = 10.4$, $SD = 7.6$) still had significantly higher scores on the CP subscale of the SPQ than CLD group members ($M = 8.1$, $SD = 6.0$), $F(1, 367) = 4.64$, $p < 0.05$. When monthly cannabis use was included as a covariate in the analysis of Positive symptoms $F(1, 367) = 9.44$, $p < 0.01$, CPD group members ($M = 11.4$, $SD = 8.6$) still had significantly higher scores on the Positive Symptoms subscale of the SPQ than CLD group members ($M = 8.6$, $SD = 6.6$), $F(1, 367) = 4.98$, $p < 0.05$. In short, polydrug users differed from both other groups even when controlling for monthly cannabis use.

DISCUSSION

Cannabis groups and schizotypy

In a sample larger than all previous studies on the topic, never, former, and current cannabis users reported use of other drugs and completed the SPQ to assess psychosis-like symptoms. Cannabis groups differed significantly on SPQ total score, as well as CP, Positive Symptom, and D subscales. The effects of cannabis group on total SPQ scores disappeared once we included the significant contribution of psychomotor stimulants. While psychomotor stimulant use was a significant covariate with cannabis group on the CP, Positive Symptom, and both D subscales, it did not eliminate the significant effect of cannabis group. The use of psychomotor stimulants plus ecstasy, however, did eliminate the significant effect of cannabis group on all subscales except the Raine D subscale. The lifetime use of all illicit substances did eliminate the significant effect of cannabis group on the Raine D subscale.

Out of the six potential subscales, four were significantly different between cannabis groups, and the differences on three of these scales were accounted for by the covariance of lifetime use of psychomotor stimulants plus ecstasy, and the last was accounted for by the covariance of lifetime use of all illicit drugs. Lifetime psychomotor stimulant plus ecstasy use eliminated the cannabis effect on the Vollema and Hoijtink D subscale, but not the Raine D subscale. The difference between these two scales is only computational; they are both comprised of the same exact items but have different weightings. The D subscale of Vollema and Hoijtink (2000) is based upon multidimensional Rasch modeling, an advanced item scoring technique where individual items were given weights based on their representativeness of the construct. Item 16—"I sometimes jump quickly from one topic to another when speaking," Item 34—"I often ramble on too much when speaking," and Item 72—"People occasionally comment that my conversation is confusing" are given two points instead of one when endorsed. These items were especially representative of the D subscale in a sample where psychiatrically diagnosed SPD was represented (Vollema and Hoijtink, 2000). Furthermore, the effect size for differences across cannabis group on the Vollema and Hoijtink D subscale (Cohen's $d = 0.41$) was comparable to that of the Raine D subscale (Cohen's $d = 0.44$). Given the greater accuracy of the Vollema and Hoijtink scale, negligible differences between the two effect sizes, and the lack of representation of psychosis-like symptoms by this subscale (Raine, 2006; Siever, 1995; Vollema *et al*., 2002), this finding is not especially germane to the idea that drug use covaries with genuine psychosis.

Even when other drug use was included in analyses, *p*-values could still be considered "statistical trends." The decision that $p < 0.05$ be used as a cut-score is an arbitrary decision (Cohen, 1994), so examinations of the size of effects can prove more informative. Measures of the use of other drugs consistently decreased the size of the effect of cannabis on schizotypy scores. The results suggest that links between cannabis and schizotypy require cautious interpretations that consider, as best is possible, other contributing factors to the relationship, especially polydrug use.

It is interesting to note that psychomotor stimulant use alone eliminated the cannabis effect in total SPQ scores, and psychomotor stimulant plus ecstasy use eliminated the cannabis effect in the CP, Positive Symptom, and Vollema and Hoijtink D subscales. When other illicit drugs that increase dopamine levels at least in the ventral tegmental area (Gardner, 2005; Robbins and Everitt, 1999) were included in analyses, the cannabis effect on the Raine D subscale also disappeared. Testing hypotheses of a potentially causal or contributing role of cannabis in the development of schizotypy or related psychotic disorders must include extensive assessments of the use of other drugs under circumstances that can keep response biases to a minimum.

There were no significant differences between the non-users group and former users on any of the

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measures of schizotypy. This result may imply that even if cannabis use increases the risk for some aspects of schizotypy or psychosis, that risk can be diminished through a reduction or cessation in use. This result seems to be congruent with the evidence of an acute toxic psychosis that resolves with abstinence (Johns, 2001), rather than the singular etiological induction of a long-term psychosis. Monthly and weekly users also did not significantly differ in any measure of schizotypy, indicating the possibility that frequency of use is not associated with psychosis-like symptoms. (See Tables 1and 2).

Drug use groups and schizotypy

Schizotypy scores did not differ between participants who used cannabis but no other illicit drugs (CLD) and users of LD. Both groups, however, scored significantly lower than the CPD group, suggesting that polydrug use is a better predictor of schizotypy than cannabis use alone. To offer further support for the import of polydrug use, monthly cannabis use served as a significant covariate in the CP and Positive Symptom subscales (the two most relevant subscales in psychosis-like symptoms), but it did not eliminate the significant effect of drug group. Thus, even when controlling for monthly cannabis use, there is still a difference between CLD and CPD group members with regards to psychosis-like symptoms.

Comparisons with other studies

Previous studies have shown a link between LD use and schizotypy (Larrison *et al*., 1999, Nunn *et al*., 2001). These studies however, have not controlled for a potential relation between LDs and illicit drugs (with the exception of cannabis, which interacted with alcohol, Nunn *et al*., 2001). Previous studies have also used different measures of schizotypal symptoms. Another major departure from previous studies (Williams *et al*., 1996) is the fact that illicit drug use serves as a confound in schizotypy scores across cannabis use. We attribute this result to our use of a newer measure of schizotypy, which has been shown to relate well to dopaminergic function (Siever, 1995) and genetic contributions to psychosis (Vollema *et al*., 2002). Additionally, our sample was nearly twice the size of the previous work.

Differences in effect sizes are also noticeable. Previous studies (Bailey and Swallow, 2004; Mass *et al*., 2001; Skosnik *et al*., 2001) found large effects, while the current study found small to medium ($d = 0.3$–0.6) effects. This result is likely due to the large sample size of the present study allowing greater power to detect smaller effects. Small sample studies finding significant effects are likely to inflate effect size estimates relative to larger studies of the same phenomena. The other two studies that had the largest sample sizes using the SPQ to date (Dumas *et al*., 2002; Schiffman *et al*., 2005) reported similar effect sizes ($d = 0.4$–0.6) but failed to account for the contribution of other illicit drug use.

Limitations

Some of the methodological limitations of the present study warrant comment. A major limitation of the study is that it was cross-sectional. The intriguing question is whether or not cannabis plays a causal role in psychosis, and longitudinal studies are better able to address this question, but are not necessarily more accurate at causal statements given that the prerequisites for causality are difficult to meet in the realm of drug use (Moore *et al*., 2007; Zullino *et al*., 2007). Despite this limitation, we believe that cross-sectional studies can still provide important contributions to the debate.

Because the survey was administered through the internet, participants were not randomly selected, which may have lead to response bias. Eighty-one per cent of participants in this study reported current or former use of cannabis. Previous studies of cannabis and schizotypy, that have samples sizes that are somewhat comparable, report percentages of cannabis use that are smaller (72% Mass *et al*., 2001; 66% Williams *et al*., 1996), but still comparable. Additionally, the method of administration has its own limitations. We did not choose to block based on IP address, which increases the potential for duplicate data, but no duplicate data sets were detected. While we acknowledge the possibility that a given individual could have responded more than once, it seems unlikely that an individual would go through the trouble of re-taking the entire questionnaire and responding differently, when s/he could have completed a portion of the questions and then skipped to the end to enter an additional e-mail address.

We also acknowledge the possibility of fraudulent data entry. While we did our best to screen for this problem, some individuals could have entered inauthentic responses. These responses would have increased within-group variance, making a significant effect more difficult to detect. Although we asked participants to report potential psychotic symptoms independently of substance use, some participants might have been experiencing substance-induced

DOI: 10.1002/hup

psychosis. The overall rates of illicit substance use in the last month were very low and there were no significant differences in monthly use of illicit substances between individuals scoring in the top 10% on the SPQ to the rest of the group. This result indicates that those individuals who were most likely to be experiencing what might be considered a psychotic episode did not have a significantly different rate of illicit substance use in the last month. This result does not rule out the possibility of acute substance-induced psychosis, but it is unlikely.

Additionally, internet access might require some basic computer skills and financial resources that prevent severely impaired or individuals of low socioeconomic status from participating. Our results may not apply to the drug-using population as a whole. Despite these limitations, an internet sample may provide more diverse results than other common methods of collecting data about substance use (Reips, 2002). In addition, internet sampling allows access to cannabis users who may be severely impaired or unwilling to attend a laboratory session. Participants may show similar reluctance to admit to more deviant psychosis-like symptoms in a less anonymous setting. Participants are more likely to respond truthfully over the internet because of belief in heightened anonymity (Rhodes *et al*., 2003). At least one study has shown that respondents tend to report more drug use on an internet survey than on an identical paper-and-pencil questionnaire (Wang *et al*., 2005).

CONCLUSION

Using different methods of data analysis, the picture remains the same; polydrug use is more strongly related to symptoms of schizotypy than cannabis use alone. The generalizability of these findings may not apply to all drug users given the limitations of the study methodology. Despite any limitations, lifetime illicit drug use, however crude an indicator, does at least signal a warning that other drug use is a likely confounding factor between cannabis and psychosis-like symptoms, at least as measured by the SPQ. Analyses based on users of different drugs confirmed these results. The polydrug use group had significantly more psychosis-like symptoms (notably in the subscales of CP and Positive Symptoms, which have empirically supported and facile relations to psychosis) than members of either the CLD group or the LD group. The latter two were not statistically different. This difference was still evident even after controlling for monthly cannabis use. The data indicate that other drug use is an important covariate in the link between cannabis and schizotypy. While the current study does not eliminate cannabis as a potential cause of schizotypy or psychosis, it does cast doubt on its being a sole cause. Future studies that measure psychosis-like symptoms prior to the use of any drugs could further elucidate the role of cannabis and other drugs in the development of schizotypy and psychosis.

ACKNOWLEDGEMENTS

The authors thank Hélène Verdoux, Gillinder Bedi, and two anonymous reviewers for comments on earlier drafts. All authors have contributed to the study and read the manuscript. Author M. E. declares involvement with a number of cannabis law reform groups. Authors N. V. D. and G. D. declare no conflict of interest. Data collection was supported by a grant from the Marijuana Policy Project. Marijuana Policy Project had no input on the design, analysis, written manuscript, or decision to submit for publication.

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DOI: 10.1002/hup