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Prospective Associations between Cannabis Use, Abuse, and Dependence and Panic Attacks and Disorder

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Abstract

The present study prospectively evaluated cannabis use, abuse, and dependence in relation to the development of panic attacks and panic disorder. Participants at the start of the study were adolescents (n = 1,709) with a mean age of 16.6 years (SD = 1.2; time 1) and were re-assessed 1 year later (time 2) and then again as young adults (time 3; Mean age = 24.2 years, SD = 0.6). Results indicated that cannabis use and dependence were significantly prospectively associated with an increased odds for the development of panic attacks and panic disorder. However, cannabis was not *incrementally* associated with the development of panic after controlling for daily cigarette smoking. The theoretical and clinical implications of these findings are discussed.

Keywords

Cannabis; Marijuana; Panic Attacks; Panic Disorder; Smoking; Drug Dependence

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Cannabis is one of the most commonly used recreational drugs in the United States (U.S.) and beyond (Office of Applied Studies [SAMHSA], 2002; Rey et al., 2002). For example, approximately 4% of adults have used cannabis in the past year and rates of cannabis abuse and dependence are on the rise (Compton et al., 2004). These data are noteworthy given empirical evidence that cannabis use, aside from being linked to negative physical health and social outcomes (Ameri, 1999; Cohen, 1981; Lehman & Simpson, 1992; Lynskey & Hall, 2000), may be related to certain psychological symptoms and conditions (Green & Ritter, 2000; Tunving, 1985).

Panic attacks have historically been linked to cannabis use (Gale & Guenther, 1971). This work was initially stimulated by the observation that cannabis use may acutely promote heightened levels of anxiety symptoms and elicit panic attacks under certain conditions or among certain individuals (Hall et al., 1994; Hollister, 1986; Szuster et al., 1988; Thomas, 1996, Tunving, 1985). Subsequent studies have strengthened confidence that (a) more frequent cannabis use and/or (b) more severe cannabis problems may be related to an increased risk of panic attacks. For example, Hathaway (2003) found that among weekly users of cannabis (n = 104), approximately 40% reported having had at least one panic attack related to such use. These prevalence rates are noteworthy in light of lifetime rates of panic attacks among the general population of approximately 5%–8% (Katerndahl & Realini, 1993). Although methodological differences in the assessment of panic attacks may hinder comparisons across studies, the limited study suggests the possibility of a cannabis-panic linkage. Increasing confidence in the generalizability of these types of associations, a recent study involving a representative sample (n = 4,745) found that a lifetime history of cannabis dependence, but not use or abuse, was related to an increased risk of panic attacks after covarying the effects of polysubstance use, alcohol abuse, and demographic variables (Zvolensky, Bernstein et al., 2006). This work also is supported by other investigations showing that daily or weekly users of cannabis report greater levels of symptoms of somatic tension and arousal such as feeling dizzy compared to nonusers (Bonn-Miller et al., 2005; Milich et al., 2000; Pickard et al., 2000; Thomas, 1996) and cognitive dyscontrol symptoms (e.g., depersonalization; Dannon et al., 2004; Mathew et al., 1993; Troisi et al., 1998; Zvolensky, Bonn-Miller et al., 2006). The nature of the direction (s) of the putative cannabis-panic association is as of yet unknown from the extant empirical research.

Overall, a growing corpus of evidence collectively suggests that heavier patterns of use or more severe forms of cannabis use problems (e.g., dependence) are related to increased risk of panic attacks. Yet, existing studies on this topic are limited in a number of key respects. Perhaps most notably, the vast majority of investigations evaluating cannabis-panic associations utilized cross-sectional designs. These cross-sectional studies, by definition, cannot explicate temporal order of onset between cannabis and panic attacks, and by extension, it is not possible to differentiate possible risk factor effects from concomitants or consequences. Only prospective studies that test whether putative risk factors predict the subsequent onset of panic attacks can achieve this aim. Unfortunately, of the limited number of prospective tests involving evaluations of cannabis in terms of psychiatric conditions, panic attacks have not been assessed (Block et al., 1991; Fergusson et al., 1996). A second key limitation of past work is that none of the previous studies have tested whether the association between cannabis and panic is not simply attributable to cigarette smoking. This limitation is unfortunate, as there is a strong association between cannabis use and its disorders and cigarette smoking (Degenhardt et al., 2001, 2003). Moreover, daily cigarette smoking is related to an increased risk of panic attacks (Zvolensky & Bernstein 2005; Zvolensky et al., 2005). A final limitation of past work is that the previously reported association between cannabis dependence and panic attacks may simply be accounted for by a comorbid pattern of more severe non-cannabis drug dependence (Ross et al., 1988). Specifically, because individuals with more severe cannabis use problems (i.e., abuse and dependence) are more likely to have problems with other substances as well

as be more prone to psychological problems (Kessler et al., 1997; Regier et al., 1990), it is possible non-cannabis drug dependence could account for the documented association between cannabis dependence and panic attack.

The aim of the present study was to provide a prospective test evaluating cannabis use, abuse, and dependence in relation to the development of panic attacks and panic disorder. Participants at the start of the study were adolescents (n = 1,709) with a mean age of 16.6 years (SD = 1.2; time 1 [TI]) and were re-assessed 1 year later (time 2; [T2]) and then again as young adults (time 3 [T3], Mean age = 24.2 years, SD = 0.6; see Method section for details). It was hypothesized that cannabis dependence, but not use or abuse, would prospectively predict an increased risk for panic attacks and panic disorder after controlling for non-cannabis drug dependence and daily cigarette smoking. These hypotheses were collectively driven by past work indicating that more severe or problematic forms of cannabis use are particularly associated with panic symptoms and psychopathology (Zvolensky, Bernstein et al., 2006).

Method

Participants

Participants were a subset of individuals from the Oregon Adolescent Depression Project. Participants were originally randomly selected from nine senior high schools in western Oregon. All participants consented to participate in the investigation. A total of 1,709 adolescents (ages 14–18; mean age at initial assessment = 16.6 years, SD = 1.2) completed the initial assessment (T1), which consisted of an interview and questionnaires, between 1987 and 1989. Approximately 1 year later (T2), 1,507 participants (88.2%) participated in a reassessment that used the same interview questions and questionnaires (mean interval between T1 and T2 = 13.8 months, SD = 2.3) (additional details are provided elsewhere; Lewinsohn et al., 1997).

As probands from the Oregon Adolescent Depression Project reached their 24th birthday, participants with a history of MDD at T2 (n = 360), those with a history of other Axis I disorders at T2 (n = 284), and a subset of those with no history of mental disorder at T2 (n = 457) were invited to participate in a T3 interview. This sampling strategy was intentional due to the expense of running this longitudinal investigation. The no-disorder comparison group was representative of the entire group of participants with no mental disorder at T2 (n = 863) in age and gender within age; all racial or ethnic minority participants were invited to participate in the T3 assessment.

Of the 1,101 young adults selected for T3 interview, 941 participated (85.4%). Of those participants, 57.2% were female, 89.0% were white, 34.1% were married, 96.8% had graduated from high school, and 31.4% had a bachelor's degree or a higher educational level. Their average age at T3 was 24.2 years (SD = 0.6). Women were more likely than men to complete the T3 assessments (88.9% versus 81.0%) (F = 13.55, df = 1, n = 1,101, $p < .001$). Differences in T3 participation as a function of other demographic characteristics or T2 diagnostic status were not statistically significant.

Measures

Participants were interviewed at T1 with a version of the Schedule for Affective Disorders and Schizophrenia for School-Age Children (K-SADS) that combined features of the epidemiologic version (Orvaschel et al., 1982) and the present episode version (K-SADS-P) and included additional items to derive DSM-III-R diagnoses. At T2 and T3, participants were interviewed with the Longitudinal Interval Follow-Up Evaluation (LIFE; Keller et al., 1987), which elicited detailed information about the course of psychiatric symptoms and disorders

since the previous interview. T3 interviews were conducted by telephone, and T3 diagnoses were made using DSM-IV criteria.

Most diagnostic interviewers had an advanced degree in clinical or counseling psychology or social work, and all were extensively trained before data collection. Interrater reliability for lifetime diagnoses in a randomly selected sub-sample at T1 (n = 233) and T2 were excellent and have been reported previously (please see Lewinsohn et al. 1997).

Cannabis use, abuse, and dependence—Information on cannabis use, abuse, and dependence was measured at T1 and T2. Specifically, cannabis use was operationalized as lifetime consumption, at or prior to, T2. Cannabis use was coded categorically (0 or 1), reflecting non-use versus use. Lifetime caseness at, or prior to, T2 of cannabis abuse and dependence were coded categorically (0 or 1), reflecting non-abuse versus abuse and non-dependence versus dependence, respectively. It is important to note that in keeping with contemporary diagnostic standards, participants could *not*, by definition, be classified as abusing and dependent on cannabis concurrently. Instead, if participants met criteria for cannabis dependence, they were not classified as abusing cannabis (i.e., they were *mutually exclusive* cases).

Daily cigarette smoking—Information on daily cigarette smoking was measured at T1 and T2. Daily cigarette smoking status was operationalized as self-reported lifetime daily use of cigarettes at or prior to T2. Daily cigarette smoking status was coded categorically (0 or 1), reflecting negative versus positive daily cigarette smoking status. Daily smoking was used, as opposed to other smoking variables, as this factor has been previously reported to be related to increased risk for panic problems (Johnson et al., 2000).

Non-cannabis drug dependence—Information on non-cannabis drug dependence was measured at T1 and T2. Defined in terms of DSM-IV diagnostic standards, non-cannabis drug dependence was operationalized as self-reported lifetime dependency at or prior to T2, on one or more the following substance classes: (a) cocaine, (b) inhalants/solvents, (c) amphetamines, and (d) opiates. Non-cannabis drug dependence was coded categorically (0 or 1) reflecting non-dependence versus dependence on one or more of these substances.

Panic attacks and panic disorder—Information on panic attacks and panic disorder was measured at T1, T2 and T3. Following the most recent recommendations for the accurate classification of panic attack history (Norton et al., 1999), we employed an operational definition in line with DSM-IV diagnostic criteria. Specifically, panic attacks were defined by (1) having an attack when all of sudden you felt frightened, anxious or very uneasy in a situation when most people wouldn't feel afraid; and (2) *four* or more of the following symptoms: (a) shortness of breath; (b) felt like you were choking or smothering; (c) heart palpitations; (d) chest pains/discomfort; (e) sweating; (f) felt faint/dizzy/unsteady (g) nausea/abdominal distress; (h) depersonalization/derealization; (i) numbness/tingling; (j) felt hot flashes or cold chills; (k) trembling/shaking; (l) fear that you might die; (m) fear of going crazy/losing control. Panic attack history was coded categorically (0 or 1), reflecting a negative versus positive lifetime history of panic attacks at or prior to T3.

Defined in terms of DSM-IV diagnostic standards, panic disorder was coded categorically (0 or 1), reflecting a negative versus positive history of panic disorder caseness at or prior to T3. Although also a feature of panic-spectrum psychopathology, agoraphobia was not included in the current report because the base-rate of agoraphobia in the present sample did not permit its meaningful study (i.e., n = 5 cases of agoraphobia). Similarly, the study of panic disorder with and without agoraphobia was not possible because of the expectedly low base-rate of panic disorder; dividing the already small number of cases with a positive history of panic disorder

into these agoraphobic and non-agoraphobic sub-groups would thus result in logistic regression analyses with insufficient power to yield interpretable results.

Data Analytic Strategy

Primary dependent measures included caseness of panic attacks and panic disorder at T3 since T2 – in other words, incidence of new cases or the development of panic attacks and disorder some time following T2 and prior to T3. In testing the prospective associations between cannabis and the development of panic, all cases with a lifetime history of panic attacks or disorder at T2 ($n = 52$) were excluded from the logistic regression analyses. Thus, all analyses evaluated the association(s) between cannabis use, abuse, and dependence in relation to the *development* of panic attacks and panic disorder prospectively.

Two sets of logistic regression analyses were performed, one for each primary dependent measure. To evaluate the putative prospective association(s) between cannabis and the development of or incidence of panic prospectively, lifetime history at T2 of cannabis use, abuse, dependence were entered separately in independent regression analyses in relation to each of the dependent variables. No covariates were included in this first set of logistic regression equations. Thus, in this model, any observed effects for cannabis variables in relation to panic cannot be attributed to cannabis-panic comorbidity in which panic temporally precedes the development of cannabis use, abuse, or dependence.

In the second set of logistic regression equations, lifetime history at T2 of non-cannabis drug dependence and daily cigarette smoking status were entered as covariates at level 1 of the model to ensure that any observed effects were not due to these factors. At level 2 in the model, lifetime history at T2 of cannabis use, abuse, or dependence were entered separately in independent regression analyses in relation to each of the dependent variables. In this model, any observed effects for cannabis variables at level 2 in the model cannot be attributed to pre-existing panic or to shared variance with factors in level 1 (Cohen & Cohen, 1983).

Results

See Table 1 for prospectively measured lifetime base-rates of the studied predictor and dependent variables. In terms of the covariates, 20.9% ($n = 315$) of the sample met criteria for lifetime history of daily cigarette smoking at T2. 1.9% ($n = 29$) of the sample met criteria for a lifetime history of non-cannabis drug dependency on one or more substances at T2. In terms of the cannabis use variables, 43.4% ($n = 695$) met criteria for a positive history of cannabis use, 2.3% ($n = 34$) of the sample met criteria for a positive history of cannabis abuse, and 4.8% ($n = 72$) met criteria for a positive history of cannabis dependence at T2. Finally, in terms of the dependent variables, 9.3% ($n = 89$) of the sample met criteria for a positive lifetime history of panic attacks, and 3.7% ($n = 35$) met criteria for a positive lifetime history of panic disorder at T3. After excluding cases with a history of panic attacks at T2 or T1 ($n = 52$), 3.6% ($n = 32$) of the sample met criteria for new caseness of panic attacks and 2.3% ($n = 21$) for new caseness of panic disorder at T3 since T2.

See Table 2 for odds ratios between the predictors and lifetime history of panic attacks and panic disorder. In the logistic regression equation *without covariates*, cannabis use was prospectively associated with an increased odds for the development of panic attacks and panic disorder (OR = 2.5, $p < .05$, 95% CI = 1.2-5.3, and OR = 2.6, $p < .05$, 95% CI = 1.04-6.5, respectively). Cannabis abuse was not associated with a change in the odds of panic attacks or panic disorder incidence. Finally, cannabis dependence was significantly associated with an increased odds for the development of panic attacks and panic disorder (OR = 3.7, $p < .01$, 95% CI = 1.4-9.3, OR = 4.9, $p < .01$, 95% CI = 1.7-14.0).

In the logistic regression equations including covariates, at step one of the model, daily cigarette smoking status was significantly associated with an increased odds for the development of panic attacks and panic disorder (OR = 3.4, p = .01, 95% CI = .1.6-7.2, and OR = 4.8, 95% CI = 1.9-12.2, respectively; see Table 3). Non-cannabis drug dependence status was not associated with a change in the odds of panic attacks or panic disorder incidence. Finally, cannabis use, abuse, and dependence were not associated with a unique change in the odds of panic attacks or disorder above and beyond daily smoking status and non-cannabis drug dependence status.[1]

Discussion

Based on cross-sectional studies, research has suggested an association between cannabis and panic attacks (Thomas, 1996; Zvolensky et al., 2006). The present study builds from such work and offers novel empirical insight into the nature of cannabis-panic associations from a longitudinal perspective.

Partially consistent with expectation, cannabis use and dependence were prospectively associated with increased odds for the development of panic attacks and panic disorder. These findings are broadly in accord with past research that has found cannabis use may trigger heightened levels of anxiety symptoms and panic attacks (Hall et al., 1994; Hollister, 1986; Szuster et al., 1988; Thomas, 1996, Tunving, 1985) and extends it to include panic disorder. Although we hypothesized such an effect would be evident primarily for cannabis dependent persons, the current findings indicate it also is evident for individuals using, but not abusing, cannabis. Thus, despite the relatively larger effect for cannabis dependence compared to use for both panic dependent measures (see Table 2), the results suggest a prospective association between both cannabis use and dependence for the development of panic psychopathology. It is noteworthy that these analyses focused on new cases of panic attack and panic disorder development and thereby provide prospective empirical evidence of a cannabis use and dependence link to panic attack and panic disorder. These findings collectively suggest that cannabis use and dependence may be a risk factor for panic psychopathology. Although the present study was not focused on the mechanisms underlying the observed associations, future research may explore moderating or mediating variables, including certain types of physical illness (e.g., lung disease) and cannabis-based withdrawal symptoms.

A second set of analyses evaluated the incremental association between cannabis and the development of panic problems after covarying for the effects of non-cannabis drug dependence and daily cigarette smoking status. Daily cigarette smoking status was significantly associated with an increased odds for the development of panic attacks and panic disorder. No such effects were evident for non-cannabis drug dependence. This finding replicates past work that daily cigarette smoking is prospectively related to the development of panic psychopathology (see Zvolensky & Bernstein 2005; Zvolensky et al., 2005, for reviews). However, in contrast to associations observed between cannabis and panic at the zero-order level (see Table 2), there was no evidence that cannabis use, abuse, or dependence were associated with a unique change in the odds of panic attacks or disorder above and beyond the covariates. These results illustrate the robust clinically-meaningful effects of daily smoking for the development of panic attacks and panic disorder. Moreover, these findings suggest that

[1]An additional set of analyses was conducted to test whether the observed effects between predictors at T2 in relation to panic outcomes at T3 were replicable between T1 and T3. The results of these additional analyses were identical. The only exception was that in these T1 to T3 analyses, the effect size for smoking in relation to panic attacks and panic disorder was even larger than observed between T2 and T3. Analyses using T1 data predicting T2 were not conducted because of the very low incidence of problems of (new) panic caseness at T2 since T1 (n = 13); and because unlike T1 to T3 and T2 to T3, the period of time between T1 to T2 was too short to meaningfully test possible relations between cannabis and panic and as a result could inflate the likelihood of censored observation in the outcome variables (i.e., cases that would develop panic problems if the cases were observed over a longer period of time).

the observed associations between cannabis and the development of panic psychopathology are not independent of the effects of daily cigarette smoking. Careful consideration of these results, however, is warranted. First, to the extent that there are large numbers of individuals that are both daily cigarette smokers who use, abuse, or are dependent on cannabis (Degenhardt et al., 2001, 2003), future research should further delineate the nature of associations between cannabis (whether use, abuse, or dependence) and the development of panic psychopathology in the context of cigarette smoking rather than by statistically controlling for this variable. Indeed although testing the relative (incremental) validity of cannabis in terms of panic etiology beyond smoking was the aim of one set of the planned study objectives, the present results suggest that next steps in this line of research may benefit from more fine-grained study of cannabis and smoking in relation to panic. For example, in terms of explicating panic etiology, future study could evaluate the role(s) of cannabis among non-smokers, smoking among those who do not use cannabis, and comorbid smoking and cannabis use. In total, the present data suggest that while cannabis use and dependence are related to the development of panic attacks and panic disorder, daily cigarette smoking is a key risk factor that appears to co-occur with such cannabis use and dependence factors and should be evaluated in the context of future study on cannabis-panic relations.

The present data highlight a clinically-relevant association between cannabis use and dependence and panic attacks and panic disorder over time. Building from the present investigation, one important avenue to pursue in future work is to replicate such findings using alternative methodologies such as laboratory-based challenge procedures (Zvolensky & Eifert, 2000). These types of non-field studies are rare in the extant cannabis-panic research literature. A second line of inquiry that would be useful and timely is to begin to understand the nature of the association by evaluating individual difference parameters (e.g., anxiety sensitivity) as well as theoretical mechanisms linking cannabis use and dependence and panic psychopathology. Conducting these types of tests will be critical in refining theoretical models of cannabis-panic associations, and ultimately, advancing intervention-oriented work.

The present study has a number of other limitations that deserve comment. First, although a well-established and accepted interview was employed to index psychological and drug histories, no cross-validation of study measures was completed. Future work could address this limitation by incorporating other assessment technologies, including cross-informant reports or laboratory assessments. Second, the present study was oriented principally to examine associations between cannabis use, abuse, and dependence in relation to the development of panic attacks and panic disorder. This approach is supported by extant work, but it also may be useful to examine the role of panic attacks and disorder and perhaps other affective vulnerabilities in relation to the maintenance of cannabis use problems. Third, although we employed diagnostic assessment of cannabis use, abuse, and dependence, the frequency of use and amount of use per occasion was not obtained. Future work could usefully include such assessments to begin to more comprehensively contextualize diagnostic information with actual levels of cannabis use. In addition, observed associations between cannabis use and dependence, but not abuse, in relation to panic in the present investigation may have been related to the study-specific (lower) rate of identified abuse relative to use and dependence at the second assessment point. Therefore, future study could evaluate the replicability of the observed specificity between use and dependence, but not abuse, in relation to panic. Finally, the current sample involved in over-selection of high-risk individuals at the final phase of recruitment. This over-sampling of emotionally at-risk persons may place explanatory limits on the nature of the observed findings. Therefore, replication and extension of the current findings is a necessary next step.

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Table 1

Descriptive Data: Lifetime Base Rates of Variables as a Function of Assessment Time Point

	T1 N=1709	T2 N=1507	T3 N=941
Daily Smoking	329/1709 (19.3%)	315/1507 (20.9%)	369/941 (39.2%)
Non-Cannabis Drug Dependence	25/1709 (1.5%)	29/1507 (1.9%)	73/941 (7.8%)
Cannabis Use	654/1709 (38.3%)	602/1507 (39.9%)	585/941 (62.2%)
Cannabis Abuse	31/1709 (1.8%)	34/1507 (2.3%)	100/941 (10.6%)
Cannabis Dependence	61/1709 (3.6%)	72 /1507 (4.8%)	109/941 (11.6%)
Panic Attacks	43/1709 (2.5%)	52/1507 (3.5%)	74/941 (7.9%)
Panic Disorder	12/1709 (.7%)	16/1507 (1.1%)	35/941 (3.7%)
Unimorbid Cannabis Use (No PA/PD)	629/654 (96.2%)	575/602 (95.5%)	531/585 (90.8%)
Unimorbid Cannabis Abuse (No PA/PD)	30/31 (96.8)	33/34 (97%)	95/100 (95%)
Unimorbid Cannabis Dependence No PA/PD)	54/61 (88.5%)	63/72 (87.5%)	91/109 (83.5%)
Unimorbid Panic Attacks (No Use)	18/43 (41.9%)	25/52 (48.1%)	20/74 (27%)
Unimorbid Panic Disorder (No Use)	4/12 (33.3%)	7/16 (43.8%)	7/35 (20%)
Comorbid Use/PA	25/654 (3.8%)	27/602 (4.5%)	54/585 (9.2%)
	25/43 (58.1%)	27/52 (51.9%)	54/74 (73%)
Comorbid Abuse/PA	1/31 (3.2%)	1/34 (2.9%)	5/100 (5%)
	1/43 (2.5%)	1/52 (1.9%)	5/74 (6.8%)
Comorbid Dependence/PA	7/61 (11.5%)	9/72 (12.5%)	18/109 (16.5%)
	7/43 (16.3%)	9/52 (17.3)	18/74 (24.3%)
Comorbid Use/PD	8/654 (1.2%)	9/602 (1.5%)	28/585 (4.8%)
	8/12 (66.7%)	9/16 (56.3%)	28/35 (80%)
Comrobid Abuse/PD	1/31 (3.2%)	1/34 (2.9%)	4/100 (4%)
	1/12 (8.3%)	1/16 (6.3%)	4/35 (11.4%)
Comorbid Dependence/PD	1/61 (1.6%)	1/72 (1.4%)	10/109 (9.2%)
	1/12 (8.3%)	1/16 (6.3%)	10/35 (28.6%)

Table 2

Lifetime History at T2 of Cannabis Use, Abuse and Dependence in relation to Incidence or Development of Panic Attacks and Panic Disorder at T3 since T2 (no covariates).

	Panic Attacks	Panic Disorder
Step 1		
Cannabis Use	**2.5 (1.2–5.3)***	**2.6 (1.04–6.5)***
Cannabis Abuse	.00	.00
Cannabis Dependence	**3.7 (1.4–9.3)****	**4.9 (1.7–14.0)****

Note. Significant ORs are in bold type

* $p < .05$

** $p < .01$

Table 3

Lifetime History at T2 of Cannabis Use, Abuse and Dependence in relation to Incidence or Development of Panic Attacks and Panic Disorder at T3 since T2 Above and Beyond Non-Cannabis Drug Dependence and Daily Cigarette Smoking Status.

	Panic Attacks	Panic Disorder
Step 1		
Smoking	**3.4 (1.6–7.2)****	**4.8 (1.9–12.2)****
Non-Cannabis Drug Dependence Comorbidity	2.9 (.89–9.4)	2.9 (.75–10.97)
Step 2		
Cannabis Use	1.3 (.55–3.2)	1.0 (.34–3.2)
Cannabis Abuse	.00	.00
Cannabis Dependence	1.3 (.43–4.2)	1.6 (.45–5.8)

Note. Significant ORs are in bold type

* $p < .05$

** $p < .01$