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The Impact of PTSD Clusters on Cannabis Use in a Racially Diverse Trauma-Exposed Sample: An Analysis from Ecological Momentary Assessment

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Abstract

Background: Accumulating evidence indicates a link between posttraumatic stress disorder (PTSD) and cannabis use and suggests that this link may vary as a function of PTSD symptom cluster type. Consistent with negative reinforcement models of substance use, individuals with elevated Cluster D (Hyperarousal) symptoms may be more likely to use cannabis in response to elevated state anxiety and experience decreases in state anxiety after using cannabis.

Objectives: We aimed to test hypotheses that the interaction of Cluster D and state anxiety would be related to subsequent cannabis use and that those with elevated Cluster D symptoms who used cannabis would report the greatest decreases in state anxiety. To test specificity, we tested whether Clusters B (re-experiencing) and C (avoidance) showed similar relationships.

Methods: The present study used ecological momentary assessment to examine cannabis use among 87 cannabis-using individuals with PTSD symptoms (64.4% male, 56.3% non-Hispanic Caucasian). State anxiety and cannabis use were assessed over the two-week period via signal contingent (six random prompts per day), interval contingent (each bedtime), and event contingent (cannabis use episodes) assessments.

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Results: Consistent with negative reinforcement models, participants with clinically significant Cluster D symptoms with elevated state anxiety had a greater likelihood of subsequent cannabis use and cannabis use resulted in less subsequent anxiety. The negative reinforcement hypothesis was only partially supported for those with Cluster B and C symptoms.

Conclusions: Results suggest that negative reinforcement models may be especially relevant to understanding cannabis use among those with clinically elevated Cluster D symptoms.

Keywords

cannabis; marijuana; posttraumatic stress; ecological momentary assessment; negative reinforcement

Introduction

Posttraumatic stress disorder (PTSD) is associated with cannabis use and cannabis-related problems. Data from three large national epidemiological studies demonstrate a link between PTSD and cannabis use and cannabis use disorder (CUD).¹⁻³ To illustrate, the National Comorbidity Study Replication found that 65% of those with a lifetime diagnosis of PTSD reported lifetime cannabis use and rates of current cannabis use were higher among those with PTSD (14%) compared to individuals without a lifetime diagnosis of PTSD (9.2%).³ Further, the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC) found that a lifetime PTSD diagnosis was significantly associated with both CUD ($OR = 1.217$) and lifetime cannabis use ($OR = 0.992$).¹ Additionally, the NESARC-III found that individuals with a past 12-month diagnosis of CUD had significantly greater odds of having a comorbid PTSD diagnosis ($OR = 4.3$) than those without CUD.²

Among veterans, those with a CUD had significantly higher rates of PTSD than those with other substance use disorders⁴ and PTSD symptom severity was incrementally positively associated with cannabis-related problems and withdrawal severity during a previous quit attempt, after controlling for other substance use and co-occurring anxiety and mood disorders.⁵ Additionally, 14.6% of veterans seeking treatment at a Veterans Affairs PTSD specialty clinic reported past six-month cannabis use and after controlling for a wide range of relevant variables (e.g., combat exposure, age, race), past six-month cannabis use was significantly associated with PTSD symptom severity.⁶ Similarly, veterans who screened positive for PTSD were significantly more likely to have used cannabis in their lifetime and within the past month than veterans who did not screen positive for PTSD (68.3% vs. 54.5% and 27.9% vs. 12%, respectively).⁷ Taken together, there appears to be a consistent link between PTSD and cannabis/CUD among representative United States populations, as well as veterans.

Negative reinforcement models of substance use propose that substances may be used in an attempt to relieve unpleasant physical and/or emotional states such as withdrawal, craving, and negative affect.^{8,9} Consistent with these models, substance use should be maintained if the desired effect is achieved (i.e., substance produces alleviation of negative state). More recent models highlight the importance of desire to escape from *and* avoid aversive internal states, such as negative affect, in motivation to use substances.⁸ This model also highlights

the importance of considering level of negative affect, as the model proposes that individuals are more likely to use substances at higher (versus more moderate) levels of negative affect.⁸ In line with these models, persons with post-traumatic symptomatology (which includes elevated negative affect) may be particularly likely to use cannabis during periods of elevated negative affect and may also experience greater effects of these substances. In support of this hypothesis, PTSD symptom severity is related to more coping-motivated cannabis use (i.e., using cannabis to cope with negative affective states) among general cannabis-using adult populations^{10, 11} and veterans.⁵ Further, cannabis-using veterans expected cannabis to relieve symptoms related to PTSD, particularly symptoms associated with arousal and intrusion, and these expectancies mediated the relationship between these PTSD symptoms and quantity of cannabis used per month.¹² Similarly, among young adult veterans, expectancies related to tension-reduction and relaxation moderated the relationship between PTSD and past-month cannabis use, such that the participants who screened positive for PTSD and endorsed higher levels of these expectancies were most likely to report past-month cannabis use.⁷

Emerging data suggest that the PTSD clusters are differentially related to cannabis use, indicating a need to examine the relation of PTSD symptomatology separately by cluster. To illustrate, re-experiencing symptoms (Cluster B) and hyperarousal symptoms (Cluster D) were positively associated with severity of cannabis use among methadone maintenance patients.¹³ Greater frequency of cannabis use following residential PTSD treatment was found among military veterans with less change in PTSD avoidance/numbing symptoms (Cluster C) and Cluster D symptoms between treatment intake and discharge.¹⁴ Further, a diagnosis of CUD was predictive of lower levels of change in Cluster C and Cluster D symptoms between treatment intake and discharge among veterans.¹⁵ Moreover, veterans with PTSD reported that using cannabis helped to alleviate sleep disturbance (i.e., a Cluster D symptom)¹⁶ and medical-cannabis users with probable PTSD were more likely to use cannabis to improve sleep than those without PTSD.¹⁷ Additionally, among veterans, coping motives related to sleep mediated the relationship between PTSD and cannabis use frequency, cannabis-related problems, and CUD.¹⁸ Taken together, and consistent with negative reinforcement models of substance use⁸, cannabis appears to be most consistently related to Cluster D, which largely consists of aversive internal states (in contrast to Cluster C, which largely consists of attempts to avoid experiencing aversive internal states).¹⁹

Consistent with negative reinforcement models of substance use,⁸ cannabidiol (CBD), one of the two main constituents of cannabis, exhibits anxiolytic effects (i.e., reduces a negative affective state).²⁰ The other main constituent of cannabis, Δ^9 -tetrahydrocannabinol (Δ^9 -THC) can produce anxiogenic effects, especially when taken in high doses or among cannabis-naïve individuals.^{21, 22} Yet, Δ^9 -THC taken in low doses has been found to produce anxiolytic effects among laboratory rodents²³ and sleep-inducing effects among humans.²⁴ In two pilot studies of PTSD patients, both Δ^9 -THC and nabilone (a synthetic cannabinoid) reduced nightmares and Δ^9 -THC improved sleep quality and Cluster D symptoms.^{25, 26} A recent positron emission tomography study found that individuals with PTSD evidenced greater CB₁ receptor availability than both healthy controls and trauma-exposed controls,²⁷ thereby pointing to greater CB₁ receptor availability as a potential mechanism by which cannabis alleviates PTSD symptoms. Taken together, the pharmacological effects of

cannabis are relevant to the negative reinforcement model of substance use broadly and PTSD symptomatology specifically. Consistent with negative reinforcement hypotheses regarding likelihood of substance use in a state of heightened negative affect,⁸ Cluster D may be especially related to cannabis use if individuals with elevated Cluster D symptoms are vulnerable to using cannabis during periods of elevated state anxiety in an attempt to manage this arousal. Yet, no known studies have tested this hypothesis.

The present study used ecological momentary assessment (EMA) to collect real-world data about cannabis use episodes over a two-week period to test the utility of the negative reinforcement⁸ hypothesis in regards to cannabis use among those with and without elevated PTSD symptoms in a racially diverse sample of community-recruited adult cannabis users. Benefits of EMA include: collection of data in real-world environments; minimization of retrospective recall bias; and aggregation of observations over multiple assessments facilitating within-subject assessments across time and context, permitting the examination of both antecedents and consequences of use.²⁸ It was predicted that the interaction of Cluster D and state anxiety would be prospectively related to cannabis use such that those with elevated Cluster D symptoms and elevated state anxiety would be the most likely to use cannabis. It was also predicted that those with elevated Cluster D symptoms who used cannabis would report the greatest decreases in state anxiety. Specifically, for the within-person effect, we hypothesized that on those occasions in which an individual experienced greater state anxiety, that individual would be more likely to use cannabis at the next study assessment. To test the specificity of these relationships, we also tested whether Clusters B and C evinced similar relationships to state anxiety and cannabis use during the monitoring period.

Material and methods

Participants and Procedures

Participants were recruited via community advertisements (e.g., flyers, newspaper ads) for a study of the relation between anxiety and cannabis use.²⁹ The current study is based on secondary analyses of those data. Interested participants completed a screening (on-line or telephone) and baseline appointment to determine eligibility. Participants were asked to refrain from cannabis use the day of their appointment. Eligibility criteria included being between 18–45 years old, current cannabis use (confirmed via urine sample using a 50 ng/ml positive cutoff), cannabis as drug of choice, and no interest in, or current receipt of, substance use disorder treatment. Of the 125 people who attended a baseline appointment, 1 refused to participate and 14 were excluded due to: negative biological verification of cannabis use ($n=6$), being under the influence of cannabis during assessment ($n=1$), meeting DSM criteria for primary substance dependence other than cannabis dependence ($n=3$), and meeting criteria for other diagnoses (e.g., psychosis) that would preclude participation ($n=4$). Of the 110 participants enrolled, 8 dropped out during the monitoring period and 9 were excluded due to: equipment malfunction ($n=7$), non-compliance with protocol during check-in appointments ($n=1$), and non-compliance with EMA data collection ($n=1$; described below). An additional 6 were excluded from the current study due to denial of PTSD symptoms.

The final sample was composed of 87 cannabis users (35.6% female) aged 18–36 years ($M=21.0$, $SD=2.7$). The racial composition was: 56.3% non-Hispanic Caucasian, 24.1% African American or Black, 3.4% Hispanic Caucasian, 1.1% American Indian, 1.1% Asian, 10.3% mixed, and 3.4% other. The majority (79.3%) were college students from a variety of local colleges (i.e., the state's Flagship University, a Historically Black College, a community college, a regional university) with 14.9% employed full-time and 39.1% employed part-time. Mean age of first cannabis use was 15.9 ($SD=2.1$; range=11–19). At baseline, participants reported using cannabis 7–90 ($M=70.1$, $SD=20.5$) days in the past 90 days. All participants endorsed at least weekly use in the past month (with 81.6% endorsing daily use).

Baseline Measures

The *Posttraumatic Stress Disorder Checklist-Civilian* (PCL-C) version 4 was used to assess PTSD symptoms at baseline.³⁰ Instructions asked participants to rate the degree to which they have experienced 17 symptoms of posttraumatic stress during the past month in response to an extremely stressful event such as being in combat, being attacked, being sexually assaulted, being physically or sexually abused, seeing someone killed or injured, or being in a fire, flood, or natural disaster. Items are assessed on a five point Likert scale (ranging from 1 [*not at all*] to 5 [*extremely*]) and correspond to the symptoms for PTSD in the *Diagnostic and Statistical Manual for Mental Disorders-IV*.¹⁹ Symptoms rated as 3 (*moderately*) or higher are considered clinically significant. Cluster B (re-experiencing) is clinically significant if at least one re-experiencing symptom is rated as 3 or higher. Cluster C (avoidance) is clinically significant if three or more avoidance symptoms are rated as 3 or higher. Cluster D (hyperarousal) is clinically significant if two or more hyperarousal symptoms are rated as 3 or higher. We categorized participants into PTSD symptom+ (any clinically significant PTSD symptom cluster) and PTSD symptom– (no clinically significant PTSD symptom clusters). The PCL-C items may be summed to yield a total symptoms severity score and/or the items assessing specific PTSD clusters (i.e., re-experiencing, avoidance, and hyperarousal) may be summed to derive a cluster specific symptom severity score. The PCL has been used to assess PTSD symptoms in numerous samples.^{31–33} Internal consistency in the current sample was excellent ($\alpha=.90$).

Cannabis-related problems were assessed using the *Marijuana Problems Scale* (MPS; Stephens et al., 2000), a list of 19 cannabis-related problems experienced in the past 90 days. Endorsed items were summed. The MPS has demonstrated good internal consistency in prior research (Buckner et al., 2010b).

Frequency of cannabis use during the 90 days prior to baseline was assessed with the *Timeline Follow Back*.³⁴ Participants were asked to report for each day how many cigarette-sized joints of cannabis they used. Days on which cannabis was used were summed to determine frequency of cannabis use. This measure has demonstrated good test-retest reliability.³⁵

EMA Measures

EMA assessments were completed on a personal digital assistant (PDA) using Satellite Forms 5.2 by Pumatech. Three types of assessments were collected several times per day over the two-week period:³⁶ *signal contingent* (completed in response to a signal from the PDA at six semi-random times within 20 minutes of the following anchor times: 9:20am, 11:40am, 1:00pm, 3:20pm, 5:40pm, and 7:20pm), *interval contingent* (completed at bedtime), and *event contingent* (completed immediately prior to using cannabis). Participants were presented with the below questions regardless of assessment type.

State anxiety.—*Subjective Units of Distress* (SUDS)³⁷ were used to assess state anxiety. Participants were asked to “Please indicate your current level of anxiety by choosing the number that best corresponds with the way you are feeling RIGHT NOW” from 0 (*Totally relaxed, on the verge of sleep*) to 10 (*The highest anxiety you have ever experienced*). SUDS ratings during EMA monitoring have been found to correlate with longer measures of anxiety.³⁸

Cannabis use.—Because participants were instructed to complete an EMA assessment immediately prior to cannabis use, participants indicated whether they were about to use cannabis (yes or no). “Yes” responses were considered cannabis use episodes. In prior work, this EMA measure was related to baseline self-report accounts of cannabis use.³⁸

Procedures

Study procedures were approved by the University’s Institutional Review Board and informed consent was obtained prior to data collection. Participants were trained on PDA use and given printed PDA instructions. Participants were instructed to not complete assessments when it was inconvenient (e.g., in class) or unsafe (e.g., driving) and asked to respond to any PDA signals within one hour if possible.

Consistent with other EMA protocols,³⁹ participants completed two days of practice data (not used for analyses) then returned to the lab to receive feedback on compliance. Participants then completed EMA assessments for two weeks, given that this timeframe appears sufficient to monitor substance use.^{38, 40, 41} Participants were paid \$25 for completing the baseline assessment and \$100 for each week of EMA data completed. A \$25 bonus was given for completing at least 85% of the random prompts during the monitoring period.

Data Analyses

Analyses were conducted using mixed effects functions in SPSS version 22.0. Models were random intercept, random slope designs. Pseudo R-squared values were calculated using error terms from the unrestricted and restricted models as described by Kreft and de Leeuw.⁴² The relationships of antecedents (baseline PTSD symptom cluster status [clinically elevated: positive vs negative] X EMA-assessed state anxiety measured continuously with the SUDS) to cannabis use were evaluated in two ways. First, generalized linear models (GLM) with a logistic response function (0=no cannabis use, 1=cannabis use) were used to conduct cross-lagged analyses to test whether PTSD cluster status interacted with state

anxiety at one time point to predict cannabis use at the next time point that day. Second, GLM was used to conduct cross-lagged analyses to test whether PTSD cluster status interacted with cannabis use at one time point to predict anxiety at the next time point. The SUDS and lagged SUDS variables were person-mean centered.

Compliance with the EMA protocol was assessed by determining mean percentage of random prompts, of end of day assessments, and of both random and end of day assessments completed per participant. Consistent with prior work,^{38, 41, 43} one participant was excluded for completing less than 20% of assessments. Remaining participants completed a mean of 68.5% ($SD=18.5\%$; range=23%–98%) of random signals, 60.7% ($SD=23.6\%$; range=7%–100%) of end of day assessments, and 67.4% ($SD=18.0\%$; range=26%–95%) of both random and end of day assessments, with compliance rates slightly higher on cannabis use days (68.8%) than non-use days (63.8%). Compliance was evenly distributed among hours of the day and days of the week. These rates are comparable to other EMA studies of cannabis users.^{38, 41} Participants completed 4,828 signal contingent ($M=55.9$, $SD=15.2$ per participant), 726 interval contingent ($M=8.5$, $SD=3.3$ per participant), and 1,011 event contingent ($M=13.1$, $SD=11.5$ per participant) assessments. Signal contingent assessments were completed on average 29.1 ($SD=55.4$) minutes after the signal occurred and there was an average of 122.1 ($SD=110.9$) minutes between same-day assessments. Participants recorded 1,812 cannabis use entries ($M=22.2$, $SD=14.2$ per participant), suggesting some cannabis use was recorded during signal and interval contingent assessments. Participants reported an average of 1.8 ($SD=2.3$) cannabis use episodes per day. Possible reactivity to EMA recording was assessed by ratings during the first and last four days of the monitoring period. Mean ratings of affect, $F(1, 84) = 0.54$, $p = .465$, $d = .11$, and number of cannabis use episodes, $F(1, 86) = 0.25$, $p = .618$, $d = .08$, did not significantly differ. In other words, there was no evidence of reactivity in the current study.

Results

Baseline Sample Descriptives

Based on PCL scores, 41.4% reported clinically significant Cluster B (Re-experiencing) symptoms ($n = 36$), 31.0% reported clinically significant Cluster C (Avoidance) symptoms ($n = 27$), and 43.7% reported clinically significant Cluster D (Hyperarousal) symptoms ($n = 38$). The PTSD groups did not differ on any demographic variable other than anxiety treatment history (Table 1), such that more participants with clinically significant PTSD cluster symptoms (PTSD symptom+) reported more anxiety treatment than those without clinically significant PTSD symptoms (PTSD symptom–). PTSD symptom+ participants reported more cannabis-related problems, although they did not differ from PTSD symptom– participants on frequency of cannabis use in the two weeks prior to the baseline appointment. Baseline cannabis use frequency was positively correlated with use during the monitoring period, $r = .38$, $p = .001$.

Baseline PTSD Clusters' Relationships with State Anxiety and Cannabis Use during the Monitoring Period

For descriptive purposes, EMA data were aggregated by participant and analyses of variance conducted. During the monitoring period, participants with clinically elevated Cluster B symptoms (Cluster B+; $M = 2.6$, $SD = 1.3$) reported significantly greater mean state anxiety than participants without clinically elevated Cluster B symptoms (Cluster B-; $M = 1.7$, $SD = 1.3$), $F(1, 85) = 10.04$, $p = .002$, $d = .69$. Cluster C+ participants ($M = 2.6$, $SD = 1.4$) reported significantly greater mean state anxiety than Cluster C- participants ($M = 1.9$, $SD = 1.3$), $F(1, 85) = 5.30$, $p = .024$, $d = .52$ and Cluster D+ participants ($M = 2.7$, $SD = 1.4$) reported significantly greater mean state anxiety than Cluster D- participants ($M = 1.6$, $SD = 1.2$), $F(1, 85) = 14.08$, $p < .001$, $d = .86$.

During the monitoring period, Cluster B+ participants ($M = 22.8$, $SD = 14.8$) did not differ from Cluster B- participants ($M = 19.5$, $SD = 12.4$) on frequency of cannabis use (i.e., number of times cannabis was used during the monitoring period), $F(1, 85) = 1.29$, $p = .259$, $d = .25$. Cluster C+ participants ($M = 21.6$, $SD = 12.5$) did not differ from Cluster C- participants ($M = 20.5$, $SD = 14.0$) on cannabis use frequency, $F(1, 85) = 0.11$, $p = .738$, $d = .08$. Cluster D+ participants ($M = 20.7$, $SD = 11.9$) did not differ from Cluster D- participants ($M = 20.9$, $SD = 14.7$) on cannabis use frequency, $F(1, 85) = 0.00$, $p = .956$, $d = .01$.

PTSD Clusters X State Anxiety in the Prediction of Subsequent Cannabis Use

Cross-lagged analyses tested whether state anxiety at one assessment point interacted with PTSD Cluster to predict cannabis use at the subsequent assessment (Table 2). The within-subjects interaction was significant for Cluster D such that subsequent cannabis use was greatest among Cluster D+ participants with higher SUDS ratings (Figure 1A). The within-subjects interaction was also significant for Cluster C but was not significant for Cluster B.

PTSD Clusters X Cannabis Use in the Prediction of Subsequent State Anxiety

Next, cross-lagged analyses tested whether Cluster D status interacted with cannabis use at one assessment point to predict state anxiety at the subsequent assessment (Table 3). Cluster D+ participants reported significantly greater state anxiety, although this significant main effect is qualified by the significant interaction indicating that subsequent state anxiety decreased among those who used cannabis and the rate of this decline was greatest among Cluster D+ participants (Figure 1B). Cluster B+ and Cluster C+ participants reported significantly greater state anxiety than B- and C- participants. The interaction was also significant for Cluster B, but was not for Cluster C.

Discussion

The current study tested negative reinforcement-based models of cannabis use⁸ in a racially diverse sample of cannabis-using individuals with elevated symptoms of specific PTSD clusters. We simultaneously examined antecedents and consequences of cannabis use among cannabis-using individuals with and without elevated PTSD symptom clusters. Findings contribute to our understanding of cannabis use in several substantial ways. First, in line

with negative reinforcement hypotheses, participants with clinically elevated PTSD symptoms should be more likely to use cannabis after experiencing elevated state anxiety and they should experience decreases in anxiety following cannabis use. This pattern was observed for participants with clinically significant Cluster D (Hyperarousal) symptoms, as cannabis use was especially likely among participants with elevated Cluster D symptoms and state anxiety and cannabis use resulted in less subsequent anxiety. This is consistent with previous findings that cannabis-using veterans expect that cannabis will alleviate arousal symptoms associated with PTSD¹² and that veterans reported that using cannabis alleviates sleep disturbance.¹⁶

Second, there was some evidence of the specificity of the utility of negative reinforcement models in understanding cannabis use amongst cannabis users with elevated Cluster D symptoms, in that the negative reinforcement hypothesis was only partially supported for those with Cluster B (Re-experiencing) and C (Avoidance) symptoms. Specifically, among Cluster C+ participants, elevated anxiety predicted greater likelihood of subsequent cannabis use but cannabis use did not result in less subsequent anxiety. On the other hand, among Cluster B+ participants, cannabis use was not associated with state anxiety. Although not fully in line with negative reinforcement models of substance use, these results are partially consistent with prior findings that cannabis-using veterans expect cannabis to relieve PTSD symptoms related to arousal and intrusion.¹² Further, these findings suggest cannabis may reduce anxiety among individuals with elevated Cluster B and D symptoms, which is consistent with prior work finding that cannabis exhibits anxiolytic effects.²⁰

Results of the current study suggest that negative reinforcement models of substance use may apply differentially to PTSD symptom clusters. This model posits that substance use tends to be reinitiated during times of negative affect and stress and that withdrawal symptoms can contribute.⁸ Consistent with this, our results suggest that experiencing Cluster D symptomatology combined with elevated state anxiety, potentially from withdrawal, may place individuals at a high risk for using and that using cannabis decreases these symptoms, thereby providing negative reinforcement. Given that individuals with elevated Cluster C symptoms used cannabis after experiencing elevated anxiety but their anxiety did not subsequently decrease, there may be other factors driving their cannabis use besides negative reinforcement.

Of note, we found that PTSD symptom+ participants and PTSD symptom– participants did not differ significantly in terms of their use of cannabis during the two weeks prior to the monitoring period, although the magnitude of the effect was nearly in the small range, suggesting this finding may be partially explained by our small sample size. Yet, PTSD symptom+ participants endorsed significantly more cannabis-related problems than PTSD symptom– participants, suggesting something about their use makes them more vulnerable to cannabis-related impairment. These results are consistent with prior work finding a stronger relation between PTSD and cannabis-related problems than PTSD and cannabis use.¹

Results have important clinical implications. For example, data suggest that patients with PTSD symptoms may benefit from tailored interventions based on which PTSD symptom

clusters they are currently experiencing. To illustrate, given that participants with clinically elevated Cluster D symptoms were more likely to use cannabis after experiencing elevated state anxiety and experience decreases in anxiety following cannabis use, these patients may especially benefit from learning more adaptive means to manage elevated anxiety. Given that discontinuation of cannabis use is associated with a number of symptoms synonymous with Cluster D symptoms (i.e., sleep disturbance, irritability, anxiety),^{44, 45} it may be especially difficult for individuals who experience elevated hyperarousal symptoms to quit using cannabis without more adaptive skills to manage increases in negative affectivity such as anxiety. On the other hand, individuals with elevated Cluster C+ symptoms may benefit from psychoeducation that although they may use cannabis in response to elevations in anxiety, cannabis use may not result in less subsequent anxiety.

Results of the present study should be considered in light of limitations that point to additional avenues of work in this area. First, we did not collect data on type of trauma and future work could benefit from testing whether negative reinforcement hypotheses are more relevant to specific types of trauma. Second, data were collected via self-report and future work could benefit from biological verification of cannabis use during the monitoring period. Relatedly, although we cannot know compliance rates for event-contingent assessments, rates for signal- and interval-contingent assessments were approximately 67%. Thus, biological verification of use could help inform compliance of event-contingent assessment and it is recommended that future work strive to improve overall compliance. Third, although recruited from the community, the present sample comprised undergraduate and graduate students from several local colleges (e.g., community college, Historically Black University). Thus, although our data are thereby generalizable to groups particularly vulnerable to cannabis use and cannabis-related impairment (i.e., young adults, college students),^{46, 47} future study is needed to examine whether the observed relations generalize to other cannabis-using populations. Similarly, our sample consisted of frequent cannabis users (e.g., over 80% of the sample reported daily use) and it is unknown whether these results would generalize to infrequent cannabis users or cannabis users using other substances concurrently. Additionally, much of the extant research that informed our hypotheses was conducted among veterans; thus, an important next step would be to examine whether our findings generalize to cannabis-using veteran populations. Fourth, participants were not treatment-seeking, permitting the examination of factors that maintain cannabis use among non-treatment seekers with and without clinically elevated PTSD symptom clusters. Given that anxiety is related to poorer cannabis cessation outcomes,^{47, 48} an important next step will be to identify factors related to lapse and/or relapse vulnerability among cannabis users in cannabis cessation treatment. Fifth, although compliance rates were comparable to other EMA studies with cannabis users,^{38, 41, 49} these rates are lower than those obtained with other populations and future work could benefit from testing ways to increase EMA protocol compliance among cannabis users. Sixth, we did not collect data on the use of other substances during the monitoring period. Given that PTSD is related to problems related to use of other substances,^{50, 51} future work could benefit from investigation of the role of other substances in cannabis use among those with PTSD. Seventh, future work could benefit from larger samples with greater representation of female participants to test whether gender moderates observed effects. Eighth, we categorized

PTSD clusters dichotomously based on PCL cut scores and future work would benefit from examining whether utilizing severity of PTSD clusters or number of PTSD symptoms per cluster evince similar relations.

Despite these limitations, these findings contribute to our understanding of co-occurring PTSD symptomatology and cannabis use. Individuals with elevated Cluster D symptoms may be especially vulnerable to the reinforcing effects of cannabis in response to elevated anxiety given that their anxiety subsequently reduces after using cannabis. Thus, these individuals in particular may benefit from interventions that teach them more adaptive strategies to manage elevated negative affective states.

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Figure 1.
The interaction of PTSD Cluster D status with (A) state anxiety to predict subsequent cannabis use and (B) cannabis use to predict subsequent state anxiety.

Table 1

Demographic and baseline cannabis variables by those with and without clinically elevated PTSD cluster symptoms.

	PTSD symptom– (<i>n</i> = 33)	PTSD symptom+ (<i>n</i> = 54)	<i>F</i> or χ^2	<i>p</i>	<i>d</i> or Cramer's <i>V</i>
Age	20.6 (1.9)	21.2 (3.0)	0.99	.322	0.23
Gender (% male)	72.7	59.3	1.62	.203	0.14
Ethnicity (% non-Hispanic/Latino)	84.8	92.6	1.32	.250	0.12
Race (% Caucasian)	60.6	59.3	1.42	.922	0.13
Estimated family income	152,911 (369,329)	113,647 (107,747)	0.47	.496	0.14
Employment status (% unemployed)	48.5	42.6	0.87	.832	0.10
Class (% second year)	30.3	25.9	2.54	.771	0.17
Marital status (% single)	93.9	90.7	3.17	.531	0.19
Anxiety treatment history (%)	6.1	25.9	5.39	.020	0.25
Substance treatment history (%)	6.1	11.1	0.63	.429	0.09
Cannabis problems	3.2 (2.2)	6.9 (3.7)	27.15	<.001	1.22
Past 2-week cannabis use (days)	11.1 (3.1)	11.7 (3.1)	0.71	.402	0.19

Note. PTSD symptom– = no clinically significant PTSD symptom cluster. PTSD symptom+ = at least one clinically significant PTSD cluster. PTSD symptom cluster status based on PCL-C³⁰.

Table 2

Separate Generalized Estimating Equations Analyses of the Lagged Effects of State Anxiety on Cannabis Use.

Parameter Estimates	B	SE	Wald χ^2	df	p
For the model involving the predictor variable of Cluster D status					
Cluster D status	0.77	0.31	6.03	1	.014
Between-person mean lagged state anxiety	0.02	0.03	0.41	1	.523
Within-person mean lagged state anxiety	0.22	0.07	10.36	1	.001
Cluster D status * Between-person mean lagged state anxiety	-0.05	0.05	1.01	1	.315
Cluster D status * Within-person mean lagged state anxiety	-0.41	0.13	9.82	1	.002
For the model involving the predictor variable of Cluster B status					
Cluster B status	0.54	0.41	1.69	1	.194
Between-person mean lagged state anxiety	-0.00	0.03	0.01	1	.929
Within-person mean lagged state anxiety	0.19	0.11	2.93	1	.087
Cluster B status * Between-person mean lagged state anxiety	0.01	0.04	0.03	1	.858
Cluster B status * Within-person mean lagged state anxiety	-0.26	0.14	3.57	1	.059
For the model involving the predictor variable of Cluster C status					
Cluster C status	1.01	0.28	12.64	1	<.001
Between-person mean lagged state anxiety	-0.01	0.04	0.05	1	.832
Within-person mean lagged state anxiety	0.28	0.06	19.76	1	<.001
Cluster C status * Between-person mean lagged state anxiety	0.02	0.05	0.11	1	.743
Cluster C status * Within-person mean lagged state anxiety	-0.36	0.10	13.85	1	<.001

Table 3

Separate Generalized Linear Modeling Analyses of the Lagged Effects of Cannabis use on State Anxiety.

Parameter Estimates	<i>B</i>	<i>SE</i>	<i>df</i>	<i>t</i>	<i>p</i>
For the model involving the predictor variable of Cluster D status					
Intercept	2.71				
Cluster D status	1.11	0.27	82.62	-4.14	<.001
Between-Person mean lagged cannabis use	-6.38	2.09	82.57	3.05	.003
Within-Person mean lagged cannabis use	0.56	0.07	6700.74	-8.46	<.001
Cluster D status * Between-Person mean lagged cannabis use	8.43	2.67	82.51	-3.16	.002
Cluster D status * Within-Person mean lagged cannabis use	-0.35	0.09	6700.97	3.94	<.001
For the model involving the predictor variable of Cluster B status					
Intercept	2.60				
Cluster B status	0.93	0.29	82.83	-3.25	.002
Between-Person mean lagged cannabis use	-3.72	1.98	82.76	1.88	.064
Within-Person mean lagged cannabis use	0.57	0.07	6701.78	-8.51	<.001
Cluster B status * Between-Person mean lagged cannabis use	5.65	2.77	82.69	-2.04	.044
Cluster B status * Within-Person mean lagged cannabis use	-0.36	0.09	6701.22	4.02	<.001
For the model involving the predictor variable of Cluster C status					
Intercept	2.59				
Cluster C status	0.74	0.30	82.69	-2.44	.017
Between-Person mean lagged cannabis use	-7.68	2.63	82.23	2.93	.004
Within-Person mean lagged cannabis use	0.32	0.08	6700.72	-4.15	<.001
Cluster C status * Between-Person mean lagged cannabis use	8.90	3.08	82.35	-2.89	.005
Cluster C status * Within-Person mean lagged cannabis use	0.06	0.09	6700.81	-0.68	.496