The Effects of Extended Cannabis Abstinence in Comorbid Posttraumatic Stress Disorder and Cannabis Use Disorder

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Abstract

Objective: This preliminary open-label study examined whether 12 weeks of cannabis abstinence was associated with posttraumatic stress disorder (PTSD) symptom improvement in people with comorbid PTSD and cannabis use disorder (CUD) (N=21).

Methods: Participants received progressive contingency reinforcement payments for successful abstinence at weeks 4, 8, and 12. Abstinence was defined as a 11-nor-9-carboxy-tetrahydrocannabinol (THC-COOH) level ≤50 ng/mL with no self-reported cannabis use. PTSD symptoms were evaluated using the Clinician-Administered PTSD Scale for *DSM-5* (CAPS-5), capturing total severity, symptom count, and cluster scores. Data were collected from January 2022 to April 2025.

Results: Participants who achieved abstinence (n = 11) reported significantly greater reductions in total PTSD symptom severity and symptom count relative to those who did not (n = 10). CAPS-5 total scores decreased from 36.2 to 10.5 among abstainers versus 34.6 to 21.8 among nonabstainers ($P = .001$). Time-by-group interactions revealed more pronounced improvements in avoidance, negative mood and cognition, and hyperarousal among abstainers. Reexperiencing symptoms improved across both groups over time, with no significant difference by abstinence status.

Conclusions: Sustained cannabis abstinence was associated with significant reductions in PTSD symptom severity and frequency over 12 weeks. While not definitive, the results raise questions about the assumption that long-term cannabis use improves symptoms or functioning in PTSD. The data instead suggest that continued cannabis use could limit recovery in some domains. This underscores the need to routinely assess cannabis use during PTSD treatment and to educate patients on the potential consequences of continued use. Larger randomized trials are warranted to replicate and extend these findings and to investigate mechanisms through which abstinence may relate to symptom changes in PTSD with CUD.

Trial Registration: ClinicalTrials.gov identifier: NCT05162651.

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Posttraumatic stress disorder (PTSD) is a debilitating psychiatric condition that can develop following exposure to trauma, characterized by intrusive memories, avoidance, negative alterations in cognitions and mood, and hyperarousal.[1] With lifetime prevalence estimated between 6.1% and 9.2%[1] and 1-year prevalence ranging from 3.5% to 4.7%,[1] PTSD imposes a considerable burden on individuals and healthcare systems alike. It also contributes to elevated risks for suicide,[2] medical comorbidities,[3] and concurrent substance use disorders (SUDs).[4]

Consistent with the self-medication and negative reinforcement models of substance use,[5] individuals with PTSD may be particularly inclined to use cannabis to alleviate trauma-related distress. Approximately 28% of individuals with PTSD report past-year cannabis use,[6] and 9.4% meet criteria for cannabis use disorder (CUD)[7]—a maladaptive pattern of cannabis use associated with tolerance, withdrawal, and continued use despite harm. CUD has been linked to greater PTSD symptom severity[8] and poorer treatment outcomes,[9] suggesting that PTSD with CUD may represent a clinically distinct subgroup compared to PTSD alone. While some studies report symptom reductions with cannabis or cannabinoid-based treatments (eg, improved sleep[10] and mood regulation[11] or benefits with synthetic nabilone for nightmares[12]), others identify potential risks, including disrupted fear-extinction learning[13] and worse clinical[14] and treatment[15] outcomes with chronic or heavy use. A recent systematic review[16] found mixed evidence, with 6 studies suggesting benefits,

Clinical Points

- The therapeutic effects of cannabis use for posttraumatic stress disorder (PTSD) remain uncertain, particularly among individuals with comorbid cannabis use disorder, who often use cannabis to self-manage distress.
- Over 12 weeks, this study found that sustained, biochemically verified cannabis abstinence was associated with greater reductions in PTSD symptom severity and total symptom count than nonabstinence.
- The observed association between cannabis abstinence and lower PTSD symptoms suggests that discussing the potential benefits of abstinence within PTSD care may be clinically valuable, alongside assessment of cannabis use and symptoms.

5 reporting worsened symptoms, and 3 showing no significant effects. Importantly, evidence also differs by use type: medicinal or prescribed cannabinoid products may have different effects than recreational or nonmedicinal use. This uncertainty leaves clinicians and patients weighing anecdotal benefits against inconsistent empirical findings, underscoring the need to clarify whether extended abstinence is associated with symptom improvement.

Beyond the scientific debate, advocacy groups, including veteran organizations, continue to promote cannabis as a therapeutic option for PTSD.[17] Veterans Affairs Canada's expenditure on cannabis for medical purposes has increased dramatically from $5.16 million in 2014 to $74.6 million in 2019 and has increased to over $200 million by 2024.[18,19] These advocacy efforts lean heavily on lived experience and anecdotal benefit, yet the empirical evidence remains limited and inconsistent.[20,21] Despite these concerns, few studies have examined the effects of cannabis abstinence on PTSD symptomatology. Given the rise of cannabis use in Canada[22] and the United States,[23] clearer data on abstinence effects are needed. Accordingly, we examined associations between 12-week cannabis abstinence and PTSD symptoms in individuals with comorbid PTSD and CUD. Secondary aims determined whether abstinence was associated with changes in both the number and severity of symptoms across 4 *Diagnostic and Statistical Manual of Mental Disorders,* Fifth Edition *(DSM-5)*–defined clusters: Cluster B (intrusion, such as flashbacks and nightmares), Cluster C (avoidance of trauma-related stimuli), Cluster D (negative alterations in cognition and mood, including guilt, detachment, or anhedonia), and Cluster E (hyperarousal symptoms like irritability, insomnia, and exaggerated startle response).[24] We hypothesized that 12 weeks of abstinence would be associated with greater reductions in total symptoms as well as cluster-specific improvements compared to nonabstinence.

METHODS

Participants

Participants with PTSD and co-occurring CUD, aged 18–65 years, were recruited through the Centre for Addiction and Mental Health (Toronto, Canada) via clinician referrals, study flyers, and online advertisements. Eligibility was assessed through an initial standardized phone interview, followed by an in-depth eligibility screening after informed consent. This study is registered at ClinicalTrials.gov (identifier: NCT05162651).

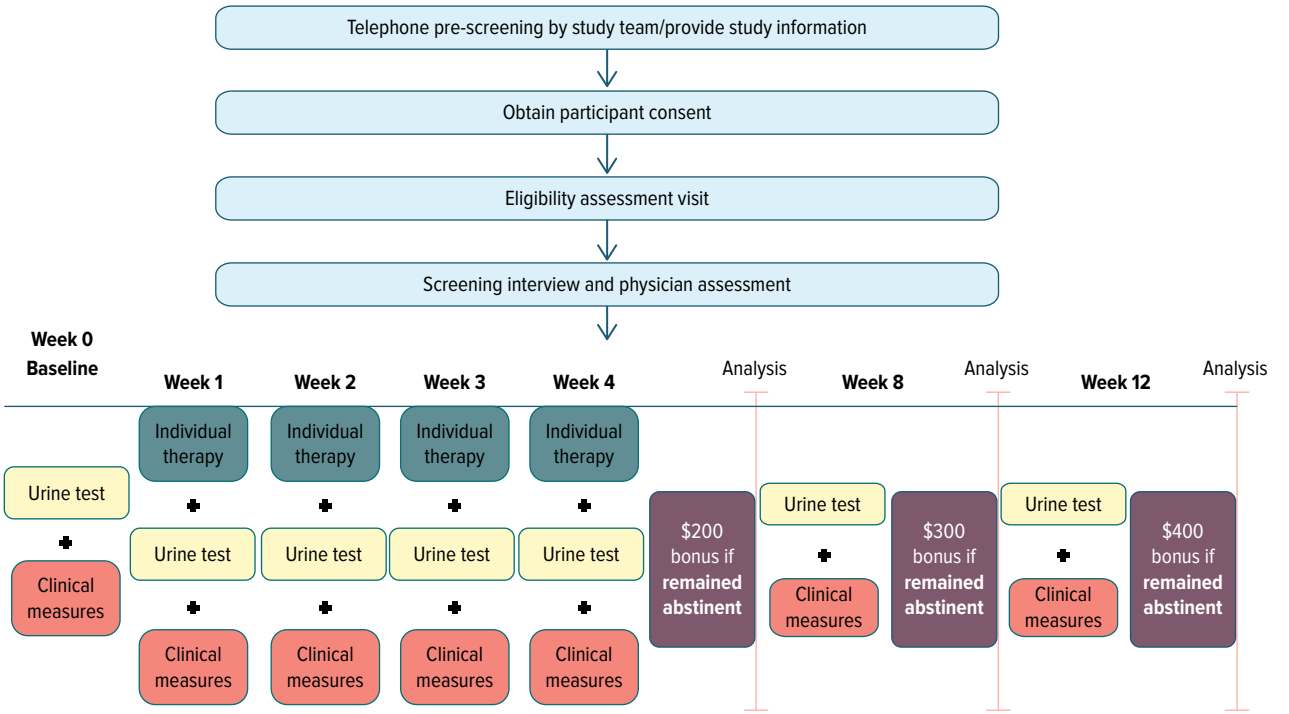
All participants met *DSM-5* criteria for current CUD and PTSD using the Structured Clinical Interview for *DSM-5*[25] and Clinician-Administered PTSD Scale for *DSM-5* (CAPS-5),[26] were on a stable antidepressant dose for at least 1 month, and had a positive urine screen (≥150 ng/mL 11-nor-9-carboxy-tetrahydrocannabinol [THC-COOH], NarcoCheck) confirming recent or regular cannabis use. This level of THC-COOH concentration in urine reveals regular consumption, a recent episode of consumption (in the last 48 hours), or resumption of cannabis use.[27]

Exclusion criteria included severe or unstable medical conditions, other SUDs in the past 3 months (excluding nicotine/caffeine), psychotic or bipolar disorders, medical cannabis prescriptions, active suicidal ideation measured using the Columbia-Suicide Severity Rating Scale,[28] neurological or medical conditions affecting cognition (eg, concussion), or moderate/severe pain on the Short Form Survey.[29] Comorbid depression, anxiety, and personality disorders were allowed to reflect real-world PTSD populations.

Study Design

Participants were asked to discontinue cannabis use for 12 weeks. Abstinence was assessed through self-report using the Timeline Followback (TLFB)[30] and confirmed by semi-quantitative urine carboxy-THC analysis (NarcoCheck) at baseline and at weeks 1, 2, 3, 4, 8, and 12. Due to the heterogeneity of cannabis administration methods (eg, smoked, vaped, edible, oil) and variability in potency, quantitative TLFB data were not included in the present analyses. Instead, TLFB responses were used to derive a binary abstinence variable (yes/no), which was verified by urine toxicology. Upon successful abstinence at weeks 4, 8, and 12, participants were eligible to receive an increasing contingent cash bonus of $200, $300, and $400, respectively. Participants attended weekly study visits, including urinalysis, clinical measures, and motivational interviewing sessions (see Figure 1 for study design). The PTSD interview was administered at baseline, week 4, week 8, and week 12. To support attendance and abstinence attempts, participants received brief (20-minute), weekly individual therapy sessions that combined psychoeducation,

Figure 1.
Overview of Study Design



motivational interviewing, and coping skills therapy between weeks 1 and 4 led by trained clinical staff. Early sessions (weeks 1 and 2) emphasized rapport and education about cannabis effects on psychiatric symptoms, while later sessions (weeks 2–4) addressed coping strategies, relapse prevention, high-risk situations, cravings, and potential lapses.

Clinical Measures

Clinical measures of PTSD were administered at baseline and weeks 4, 8, and 12. The primary outcome was PTSD severity via CAPS-5.[26] Anxiety was indexed using the self-report Beck Anxiety Inventory,[31] while depression was measured using the self-reported Beck Depression Inventory.[32]

Substance Use Measures

CUD severity was evaluated using the Cannabis Use Disorders Identification Test–Revised[33] and the *DSM-5*.[24] A 16-item version of the Marijuana Withdrawal Checklist[34] was also administered, capturing cannabis withdrawal symptoms (eg, craving, headache, restlessness).

Cannabis Screen

Biochemical verification of cannabis use (semiquantitative carboxy-THC levels) was conducted using a NarcoCheck Extended Version PreDosage Cannabis kit. This test measures specific levels of the THC metabolite THC-COOH and has 6 detection levels: negative (0 ng/mL), very low (18 ng/mL), low (50 ng/mL), medium (150 ng/mL), high (300 ng/mL), and very high (600 ng/mL). We selected the low (50 ng/mL) threshold for THC-COOH in urine as the cutoff for abstinence based on prior literature employing that level to differentiate residual excretion from new use under continuously supervised abstinence.[35,36]

Data Analysis

The primary outcome was change from baseline to week 12 in total PTSD symptom severity assessed by the CAPS-5 total score. Secondary outcomes included total number of PTSD symptoms endorsed, symptom severity, and count across each of the 4 *DSM-5* clusters: intrusion (B), avoidance (C), negative alterations in cognition and mood (D), and hyperarousal (E). Linear mixed-effects models (LMMs) were used to analyze outcomes with statistical significance defined as $P < .05$; all tests were 2-tailed. Fixed effects included time (treated as a categorical variable: baseline and weeks 4, 8, and 12), group (continuous abstinence vs nonabstinence), and their interaction. Participants were classified as continuously abstinent if they self-reported no cannabis use between baseline and week 12 and demonstrated THC-COOH levels ≤50 ng/mL at weeks 4, 8, and 12. Participants reporting cannabis use

or evinced THC-COOH levels >50 ng/mL at any of these timepoints were otherwise classified as being nonabstinent. Analyses were restricted to completers, as abstinence status required biochemical verification across all timepoints and could not be determined for dropouts.

Prespecified covariates included age, gender, education, race, income, baseline cannabis withdrawal severity, baseline anxiety severity, and severity of cannabis and tobacco use disorders. These were selected based on prior evidence linking these factors to PTSD symptom trajectories and substance use outcomes,[8,15] as well as their potential to introduce sociodemographic confounding. Baseline Beck Anxiety Inventory and Marijuana Withdrawal Checklist scores were also included given group differences observed at baseline. All models included a random intercept for participant ID to account for repeated measurements and were estimated using restricted maximum likelihood.

Post hoc pairwise comparisons between timepoints were conducted using estimated marginal means (EMMs), with Bonferroni adjustments for multiple comparisons. Within group effect sizes for changes in our primary and secondary outcomes from baseline to weeks 4, 8, and 12 were calculated using Cohen *d*. All analyses were conducted in R v.4.1.4, using the lme4, lmerTest, and emmeans packages.

RESULTS

Participants were prescreened by phone interview. Of the 101 individuals screened, 52 were invited to complete a full eligibility assessment, conducted in person or online.

Following consent, 13 were excluded based on eligibility criteria, and 11 withdrew voluntarily. Reasons included not meeting diagnostic criteria for CUD (n = 5), meeting criteria for another SUD (n = 3), a negative or subthreshold urine screen for cannabis (n = 2), and not meeting PTSD criteria according to the CAPS-5 (n = 3).

A total of 28 participants completed the baseline session (see Table 1 for sample characteristics). Of these, 9 later discontinued, leaving 21 participants with complete data for analysis; details are presented in the CONSORT diagram (Supplementary Figure 1). Completers (n = 21) and dropouts (n = 8) did not differ significantly in demographic or substance use characteristics (Supplementary Table 1). However, completers exhibited slightly higher baseline PTSD severity and symptom severity in total CAPS-5 scores and avoidance and intrusion clusters. These differences were modest and not indicative of systematic attrition bias.

At baseline, those who achieved abstinence had significantly lower THC-COOH levels (P =.04), higher withdrawal severity (P =.03), and elevated anxiety symptoms, which were then controlled for in subsequent analyses. Baseline THC-COOH levels were not significantly correlated with CAPS-5 total severity (r = 0.10, P = .63) or symptom count (r = 0.10, P = .60) across all participants.

Changes in THC-COOH Levels

Among the 21 participants who completed all 12 study visits, 11 (52.4%) achieved 12 weeks of biochemically verified cannabis abstinence. The LMMs revealed no main effect of time (β = −.25, P = .95) or abstinence status (β = −122.46, P = .10) on THC-COOH levels. However, a significant time × group interaction (β = −19.44, SE = 5.33, $t_{116.1}$ = −3.64, P < .001) indicated distinct patterns across those who achieved 12 weeks of continued abstinence and those who did not (Figure 2).

Among abstinent participants, estimated THC-COOH levels decreased from 333.3 ng/mL at baseline (95% CI: 244.43 to 422.15) to 9.3 ng/mL at week 4 (95% CI: −79.57 to 98.15), 10.9 ng/mL at week 8 (95% CI: −77.93 to 99.79), and 15.5 ng/mL at week 12 (95% CI: −73.38 to 104.33). In contrast, those who did not maintain abstinence showed less dramatic reductions: with estimated means from 417.1 ng/mL at baseline (95% CI: 332.92 to 501.24) to 254.6 ng/mL at week 4 (95% CI: 170.42 to 338.74), 318.4 ng/mL at week 8 (95% CI: 227.56 to 409.18), and 345.7 ng/mL at week 12 (95% CI: 251.54 to 439.91).

Changes in Total PTSD Symptom Severity

LMMs revealed a significant time × group interaction (β = −12.83, 95% CI: −19.98 to −5.67, SE = 3.65, P = .001), indicating that the trajectory of PTSD symptom severity differed by abstinence status (Table 2; Figure 3A). A significant main effect of time was also observed (β = −12.81, SE = 2.71, P < .001), while the main effect of abstinence status was not significant (β = 1.58, SE = 4.51, P = .73). To assess whether this effect was attributable to THC exposure, THC-COOH levels were added as a covariate in exploratory mixed-effects models (Supplementary Table 2). THC concentration was not a significant predictor of PTSD severity, and the time × abstinence group interaction remained significant after adjustment (P = .03).

EMMs indicated that participants who maintained cannabis abstinence across the 12-week period showed greater reductions in PTSD symptom severity, with CAPS-5 total scores decreasing from 36.2 at baseline (95% CI: 29.72 to 42.62) to 10.5 at week 12 (95% CI: 4.09 to 16.98). In contrast, participants who did not maintain 12 weeks of abstinence experienced less symptom reduction, from 34.6 at baseline (95% CI: 28.51 to 40.67) to 21.8 at week 12 (95% CI: 15.11 to 28.45).

Table 1.

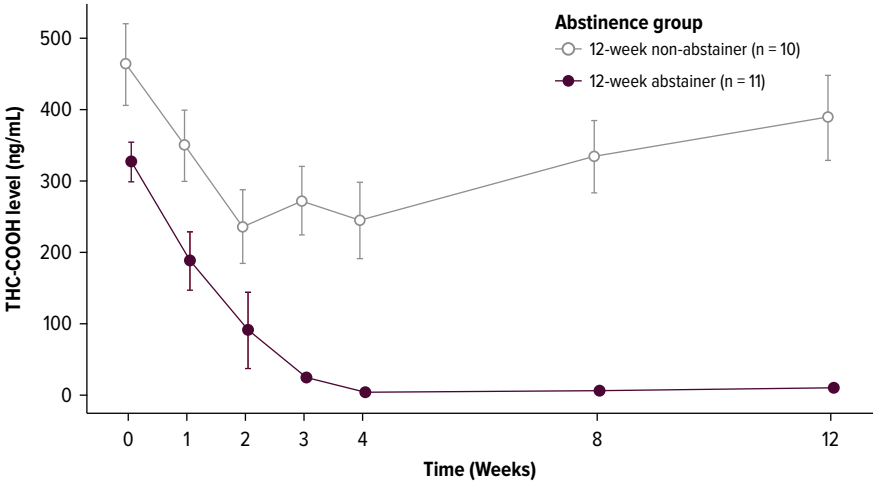
Baseline Demographic and Clinical Characteristics of Sample

Variable	Maintained abstinence (n = 11)[a]	Did not maintain abstinence (n = 10)[a]	Significance level
Age, y	29.91 (11.22)	34.80 (10.52)	.32
Education, y	15.18 (3.84)	14.95 (2.47)	.87
Sex at birth (F/M/no disclosure), n	8/2/1	6/4/0	.38
Race (White/Black/Asian/Mixed/Other), n	5/0/1/4/1	5/1/2/2/0	.06
FTND score	1.82 (2.56)	1.10 (1.72)	.47
BDI score	29.73 (11.48)	23.50 (12.86)	.26
BAI score	33.64 (8.74)	20.70 (10.64)	.007[b]
CAPS-5 total severity	36.82 (7.94)	34.50 (6.55)	.48
CAPS-5 total symptoms	14.64 (2.97)	13.40 (2.01)	.29
MWC score	24.45 (9.33)	15.56 (6.21)	.03[c]
CUDIT-R score	22.09 (4.43)	22.10 (6.26)	.99
THC-COOH levels	327.27 (90.45)	465.00 (19.60)	.04[c]

[a]Continuous variables are presented as mean (SD). Categorical variables are presented as number of participants. All measures were collected at baseline.
[b]$p < .01$.
[c]$p < .05$.
Abbreviations: BAI = Beck Anxiety Inventory, BDI = Beck Depression Inventory, CAPS-5 = Clinician-Administered PTSD Scale for *DSM-5*, CUDIT-R = Cannabis Use Disorder Identification Test–Revised, F = female, FTND = Fagerström Test for Nicotine Dependence, M = male, MWC = Marijuana Withdrawal Checklist, THC-COOH = 11-nor-9-carboxy-Δ^9-tetrahydrocannabinol.

Figure 2.

THC-COOH Levels (ng/mL) Over 12 Weeks by Cannabis Abstinence Status[a]



[a]Analyses were restricted to participants who completed the 12-week trial (n = 21) and had verified abstinence status at weeks 4, 8, and 12. Mean THC-COOH levels (ng/mL) are shown for participants who maintained cannabis abstinence (●, n = 11) versus those who did not (○, n = 10). The time × abstinence group interaction was significant ($P < .001$), with abstainers showing a decline in THC-COOH levels (−317.82 ng/mL, $P < .0001$) and nonabstainers showing no meaningful change. Error bars represent SEs of the mean.
Abbreviation: THC-COOH=11-nor-9-carboxy-Δ^9-tetrahydrocannabinol.

Changes in Total Number of PTSD Symptoms

In addition to total severity, we examined symptom counts, defined as the number of PTSD symptoms meeting threshold on the CAPS-5. LMMs revealed a significant time × group interaction ($\beta = -5.59$, 95% CI: −8.79 to −2.40, SE = 1.63, $P = .001$), indicating that patterns of PTSD symptom

Table 2.

Changes in CAPS-5 PTSD Symptom Severity and Symptom Counts by Cluster and Abstinence Status Over 12 Weeks[a]

Cluster	Abstinence status	Week 0, mean (SD)	Week 4, mean (SD)	Week 8, mean (SD)	Week 12, mean (SD)	Cohen *d* 0–4	Cohen *d* 0–8	Cohen *d* 0–12	*P* (time)	*P* (abstinence status)	*P* (time × abstinence status)
B severity	Maintained 12 weeks of continued abstinence	8.43 (0.98)	4.88 (0.98)	2.70 (0.98)	2.61 (0.98)	−1.14	−2.06	1.84	<.001	.841	.102
	Noncontinued abstinence	8.71 (0.92)	6.13 (0.92)	6.48 (0.99)	5.07 (1.06)	−0.977	−0.964	1.24	<.001	.841	.102
B symptoms	Maintained 12 weeks of continued abstinence	3.30 (0.43)	2.12 (0.43)	1.03 (0.43)	1.03 (0.43)	−0.977	−1.959	1.67	.002	.639	.228
	Noncontinued abstinence	3.59 (0.41)	2.43 (0.41)	2.78 (0.44)	2.10 (0.48)	−1.011	−0.786	1.19	.002	.639	.228
C severity	Maintained 12 weeks of continued abstinence	4.89 (0.59)	2.53 (0.59)	2.25 (0.59)	1.16 (0.59)	−1.679	−1.508	2.62	.021	.127	.004
	Noncontinued abstinence	3.54 (0.56)	3.04 (0.56)	2.86 (0.59)	2.15 (0.64)	−0.274	−0.273	0.63	.021	.127	.004
C symptoms	Maintained 12 weeks of continued abstinence	1.91 (0.24)	1.00 (0.24)	0.91 (0.24)	0.46 (0.24)	−1.547	−1.427	2.35	.034	.100	.018
	Noncontinued abstinence	1.33 (0.22)	1.33 (0.22)	1.15 (0.24)	0.75 (0.27)	0	−0.138	0.63	.034	.100	.018
D severity	Maintained 12 weeks of continued abstinence	12.66 (1.44)	8.66 (1.44)	7.48 (1.44)	4.39 (1.44)	−0.852	−1.139	1.05	<.001	.753	.120
	Noncontinued abstinence	13.33 (1.36)	10.08 (1.36)	7.82 (1.45)	8.04 (1.55)	−0.816	−1.177	0.93	<.001	.753	.120
D symptoms	Maintained 12 weeks of continued abstinence	5.86 (0.66)	3.13 (0.66)	2.86 (0.66)	1.58 (0.66)	−0.981	−1.048	1.67	.015	.678	.030
	Noncontinued abstinence	5.46 (0.63)	3.96 (0.63)	3.14 (0.68)	3.52 (0.75)	−0.968	−1.287	1.05	.015	.678	.030
E severity	Maintained 12 weeks of continued abstinence	10.14 (1.04)	6.23 (1.04)	4.14 (1.04)	2.32 (1.04)	−1.161	−1.648	2.87	.065	.459	.001
	Noncontinued abstinence	9.01 (0.99)	6.76 (0.99)	6.38 (1.07)	6.77 (1.17)	−0.828	−1.107	0.93	.065	.459	.001
E symptoms	Maintained 12 weeks of continued abstinence	4.05 (0.40)	2.68 (0.40)	1.68 (0.40)	0.96 (0.40)	−1.027	−1.687	2.68	.220	.289	<.001
	Noncontinued abstinence	3.43 (0.38)	2.93 (0.38)	2.65 (0.41)	2.85 (0.45)	−0.461	−0.97	0.52	.220	.289	<.001
Total severity	Maintained 12 weeks of continued abstinence	36.17 (3.10)	22.63 (3.10)	16.71 (3.10)	10.53 (3.10)	−1.372	−2.031	3.28	<.001	.730	.001
	Noncontinued abstinence	34.58 (2.93)	25.67 (2.93)	23.39 (3.08)	21.78 (3.27)	−1.151	−1.401	1.48	<.001	.730	.001
Total symptoms	Maintained 12 weeks of continued abstinence	14.32 (1.26)	9.05 (1.26)	6.51 (1.26)	4.14 (1.26)	−1.354	−1.998	3.09	<.001	.660	.001
	Noncontinued abstinence	13.50 (1.19)	10.59 (1.19)	9.47 (1.26)	8.92 (1.35)	−0.984	−1.372	1.30	<.001	.660	.001

[a]Symptom severity and count scores are presented for each CAPS-5 symptom cluster: B, intrusion; C, avoidance; D, negative alterations in cognition and mood; and E, hyperarousal. Values represent estimated marginal means (SDs) at weeks 0, 4, 8, and 12 for participants who maintained 12 weeks of cannabis abstinence and those who did not. *P* values reflect fixed effects for time, abstinence status, and their interaction (time × abstinence status) from linear mixed-effects models. Analyses were restricted to participants who completed the 12-week trial (n = 21) and had verified abstinence status at all follow-up visits.

Abbreviations: CAPS-5 = Clinician-Administered PTSD Scale for *DSM-5*, PTSD = posttraumatic stress disorder.

count change differed by abstinence status (Table 2; Figure 3B).

A significant main effect of time was also observed (β = −4.59, SE = 1.21, $P < .001$), whereas the main effect of abstinence status was not statistically significant (β = 0.82, SE = 1.82, $P = .66$). Similarly, after controlling for THC-COOH, our results did not change, with the time × abstinence group interaction remaining significant ($P = .026$).

EMMs revealed that participants who remained abstinent across all 12 weeks had larger decreases in PTSD symptom count, with total number of symptoms dropping from 14.3 at baseline (95% CI: 11.73 to 16.92) to 4.1 at week 12 (95% CI: 1.55 to 6.74); lesser reductions were observed in nonabstainers: 13.5 at baseline (95% CI: 11.05 to 15.95) to 8.9 at week 12 (95% CI: 6.17 to 11.66).

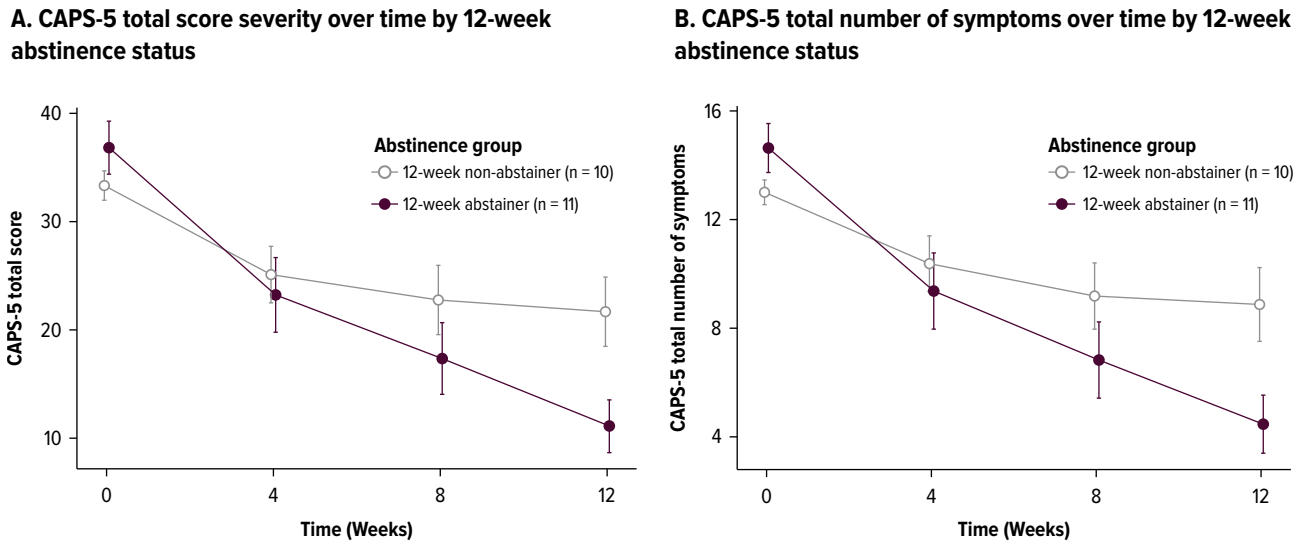
Changes in CAPS-5 Cluster-Based Symptom Severity

Analyses of CAPS-5 cluster-based severity scores for clusters B through E revealed differential patterns of symptom change depending on cannabis abstinence (Table 2).

A significant time × group interaction emerged for Cluster C (β = −2.34, 95% CI: −3.88 to −0.80, SE = 0.79, $P = .0043$) and Cluster E (β = −5.57, 95% CI: −8.74 to −2.41, SE = 1.61, $P = .001$). The interaction for Cluster D trended in the same direction (β = −2.98, 95% CI: −6.69 to 0.72, SE = 1.89, $P = .12$) but did not reach significance. Main effects of time were also significant for Cluster B (β = −1.50, SE = 0.47, $P = .0024$), Cluster C (β = −0.58, SE = 0.27, $P = .034$), and Cluster D (β = −1.94, SE = 0.78, $P = .015$), reflecting overall symptom reduction regardless of abstinence status. No

Figure 3.

Changes in PTSD Symptom Severity (A) and Symptom Count (B) Over 12 Weeks by Cannabis Abstinence Status[a]



[a]Analyses were limited to participants who completed the 12-week trial (n = 21) and had verified abstinence status at all follow-up visits. Panels depict mean CAPS-5 total severity (A) and total symptom count (B) for abstainers (●, n = 11) and nonabstainers (○, n = 10). Significant time × abstinence group interactions ($P < .05$) reflect greater reductions in PTSD symptom severity and symptom count among abstainers. Error bars indicate SEs of the mean.

Abbreviations: CAPS-5=Clinician-Administered PTSD Scale for *DSM-5*, PTSD=posttraumatic stress disorder.

significant main effects of group status alone were observed for any cluster ($Ps > .10$).

EMMs indicated that participants who maintained cannabis abstinence for 12 weeks showed greater reductions in symptom severity across Clusters B–E, whereas nonabstainers exhibited more modest improvements. Refer to Table 2 for EMMs and effect sizes.

Changes in CAPS-5 Cluster-Based Symptom Count

Analyses of CAPS-5 cluster-based symptom counts for Clusters B through E revealed distinct patterns of symptom change based on abstinence status. Significant time × group interactions were observed for Cluster C (β = −0.88, 95% CI: −1.58 to −0.17, SE = 0.36, $P = .018$), Cluster D (β = −2.33, 95% CI: −4.39 to −0.28, SE = 1.05, $P = .03$), and Cluster E (β = −2.51, 95% CI: −3.75 to −1.26, SE = 0.64, $P < .001$), indicating that symptom trajectories differed between those who sustained abstinence and those who did not. The interaction for Cluster B (β = −0.78, 95% CI: −2.02 to 0.47, SE = 0.64, $P = .23$) was not significant. Main effects of time reached significance for Cluster B (β = −1.50, SE = 0.47, $P = .002$), Cluster C (β = −0.58, SE = 0.27, $P = .034$), and Cluster D (β = −1.94, SE = 0.78, $P = .015$), reflecting overall symptom reductions over time across groups. No significant main effects of abstinence status alone were detected for any cluster ($Ps > .10$).

As shown in Table 2, participants who maintained 12 weeks of cannabis abstinence demonstrated greater reductions in CAPS-5 symptom counts across Clusters B–E compared to nonabstainers. Improvements were most pronounced in Clusters D and E, with effect sizes increasing over time. In contrast, reductions among nonabstinent participants were smaller and less consistent.

DISCUSSION

The present findings suggest that, among individuals with comorbid PTSD and CUD, biochemically verified sustained cannabis abstinence may be associated with greater reductions in PTSD symptom severity and frequency over a 12-week period in comparison to continued use or intermittent abstinence. The observed differences in symptom trajectories between groups are consistent with prior studies that associate continued cannabis use with poorer PTSD outcomes.[8,37,38] Notably, individuals who remained abstinent showed greater reductions in several core symptom clusters, including avoidance (C), negative alterations in mood and cognition (D), and hyperarousal (E). These domains are frequently cited as targets for cannabis-based self-medication among individuals with PTSD[39–42]; however, in this comorbid PTSD and CUD sample, sustained cannabis abstinence was associated with symptom

improvement, thereby challenging assumptions about its clinical utility in this population.

Several mechanisms may explain the link between abstinence and observed clinical improvements. Chronic cannabis use may downregulate cannabinoid type 1 (CB1) receptor expression and function,[43,44] impairing endogenous cannabinoid signaling and contributing to maladaptive stress responses. This dysregulation may also worsen PTSD-related hyperarousal and avoidance behaviors. In contrast, cannabis abstinence may be associated with facilitating the restoration of CB1 receptor functioning in frontal and limbic brain regions,[45] which could be related to better emotion regulation, cognitive flexibility, and capacity for trauma processing. Moreover, cannabis abstinence may coincide with shifts away from avoidance-based coping strategies, which may relate to observed symptom improvements. Chronic cannabis use, particularly in the context of CUD, has been associated with impaired distress tolerance,[46,47] reinforcing substance-based coping strategies that may hinder symptom recovery. Abstinence from cannabis may be linked to greater distress tolerance and engagement in adaptive coping strategies, such as mindfulness and acceptance-based coping,[48–50] all of which have demonstrated efficacy in managing PTSD symptomatology.

Interestingly, while cannabis abstinence was associated with significant reductions in several PTSD symptom clusters, there were no differential effects for Cluster B symptoms (reexperiencing), such as flashbacks, intrusive memories, and nightmares. Both abstinent and nonabstinent participants reported similar Cluster B improvements, suggesting that factors unrelated to cannabis use may have contributed to symptom change or insufficient power. Exposure to structured assessments and repeated clinical contact may have supported reflection or self-monitoring, which can modestly alleviate distressing memory intrusions. Additionally, although cannabis has been linked to impaired extinction learning,[51] its influence on spontaneous trauma-related intrusions is less clear. In contrast, symptoms within Clusters C, D, and E draw more heavily on systems involved in affect regulation, stress reactivity, and behavioral control. These functions are closely tied to endocannabinoid signaling,[52] which may help account for the observed patterns of associations across symptom domains.

Our findings contribute to a growing body of literature suggesting that perceived benefits of cannabis use for PTSD may not translate into lasting clinical improvements. While many individuals with PTSD report using cannabis to manage trauma-related symptoms, the present results suggest that abstinence may be associated with greater reductions in these same domains. Much of the symptom improvement occurred within the first 4 weeks, even as cannabis use in the nonabstaining group remained high. Early reductions may reflect nonspecific factors such as psychoeducation, therapeutic engagement, or expectancy effects during initial study visits. It is also possible that persistent PTSD symptoms made abstinence more difficult to achieve, consistent with evidence linking higher symptom burden to challenges in cessation. This aligns with recent randomized clinical trial (RCT) findings showing no significant effect of smoked cannabis on PTSD symptoms.[53] Together, these findings raise the possibility that symptom relief reported during cannabis use may be shaped more by expectancy effects, temporary anxiolytic effects, cannabis withdrawal symptom attenuation, or avoidance maintenance rather than any direct therapeutic action of cannabinoids.

Our findings contribute to ongoing efforts aiming to clarify whether cannabis use alleviates or exacerbates PTSD symptoms. This study offers a unique contribution by providing biochemically verified evidence that sustained cannabis abstinence is associated with symptom improvement in individuals with comorbid PTSD and CUD. In contrast to prior work relying primarily on self-reported use or cross-sectional data, our prospective design tracked PTSD symptom trajectories over 12 weeks and incorporated objective measures of abstinence through THC-COOH assays. By pairing biochemical verification with the widely validated CAPS-5, this study may reduce reporting bias and support confidence in the observed associations between cannabis abstinence and PTSD symptom change. Overall, these findings help refine the ongoing debate by indicating that continued cannabis use may be linked to less favorable clinical outcomes that needs to be validated in future research.

This study has several limitations. The sample size was relatively small, and a larger cohort is needed to confirm and extend these preliminary results. Relatedly, the study employed an open-label design, where participants' expectancy effects concerning cannabis abstinence may have influenced our outcomes. Additionally, while abstinence was biochemically verified, self-reported cannabis use could introduce recall, response, or social desirability biases. Moreover, our 8- and 12-week follow-up visits relied upon a binary yes/no question to evaluate cannabis abstinence alongside biochemical verification, which prevents a more nuanced analysis of gradual reductions in cannabis use among nonabstainers. Consequently, it remains unclear whether reductions in cannabis use are associated with subsequent PTSD symptom change beyond abstinence status. Another limitation is that our findings are specific to individuals with comorbid PTSD and CUD and should not be generalized to PTSD populations without CUD, as withdrawal- and dependence-related factors may

uniquely influence symptom patterns in this group. Additionally, although participants' compensation was tied to verified abstinence, contingency management may have indirectly influenced symptom trajectories by increasing engagement or perceived reward. Without a comparison group that did not receive noncontingent reinforcement, the extent to which observed improvements reflect abstinence itself versus behavioral reinforcement remains uncertain. Furthermore, our analyses were restricted to study completers, which may bias results if participants who discontinued differed systematically in symptom severity or abstinence likelihood. Finally, we did not collect information on cannabis potency or cannabinoid composition at baseline, which could play a role in PTSD symptom trajectories. Variability in THC exposure may play a role in both withdrawal experiences and clinical outcomes, while other cannabinoids, such as cannabinol and cannabigerol, and minor constituents like terpenes and flavonoids, may modulate the psychoactive and anxiolytic effects of cannabis. Future studies should use RCTs with larger samples, longer follow-ups, and detailed cannabis use assessments.

CONCLUSIONS

Our study provides preliminary evidence that, among individuals with comorbid PTSD and CUD, cannabis abstinence was associated with lower PTSD severity and fewer symptoms. These findings contrast with the commonly reported rationale for cannabis use among individuals with PTSD, where symptom relief is often cited as a primary motivator.[35,54,55] This discrepancy underscores the need for rigorous studies to clarify associations and potential mechanisms linking cannabis use, abstinence, and PTSD symptoms in populations with and without CUD.

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Supplementary Material

Article Title: The Effects of Extended Cannabis Abstinence in Comorbid Posttraumatic Stress Disorder and Cannabis Use Disorder

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LIST OF SUPPLEMENTARY MATERIAL FOR THE ARTICLE

1. **Table 1** Comparison of Baseline Characteristics Between Completers (n = 21) and Dropouts (n = 8)
2. **Table 2** Exploratory Analyses Including THC-COOH Concentration as a Covariate in Mixed-Effects Models Predicting PTSD Outcomes
3. **Figure 1** CONSORT Diagram

DISCLAIMER

This Supplementary Material has been provided by the authors as an enhancement to the published article. It has been approved by peer review; however, it has undergone neither editing nor formatting by in-house editorial staff. The material is presented in the manner supplied by the author.

Supplementary Table 1. Comparison of Baseline Characteristics Between Completers (n = 21) and Dropouts (n = 8)

Variable	Completers (n = 21) Mean (SD)	Dropouts (n = 8) Mean (SD)	p-value
Demographic Characteristics			
Age (years)	32.35 (11.19)	32.43 (10.63)	.987
Education (years)	15.18 (3.24)	16.50 (3.62)	.413
Income (category)	5.10 (3.52)	5.71 (4.50)	.751
Sex at birth (F/M ratio)	0.80 (0.70)	0.71 (0.49)	.727
Race (categorical score)	9.75 (4.58)	9.29 (4.39)	.816
Employment status	0.85 (0.93)	1.00 (0.82)	.694
Marital status	1.30 (2.43)	0.57 (1.51)	.368
Substance-Use and Withdrawal Measures			
Urinary THC-COOH (ng/mL)	382.50 (149.80)	343.75 (227.47)	.666
CUDIT-R score	22.10 (5.38)	21.14 (2.19)	.519
Marijuana Withdrawal Checklist (MWC) total	20.21 (9.28)	24.20 (7.85)	.363
Fagerström Test for Nicotine Dependence (FTND)	1.82 (2.56)	1.10 (1.72)	.471
Affective and Anxiety Symptoms			
Beck Depression Inventory (BDI)	26.90 (12.57)	25.38 (7.71)	.701
Beck Anxiety Inventory (BAI)	27.95 (11.62)	25.29 (16.26)	.700
PTSD Severity and Symptom Measures (CAPS-5)			
Total PTSD severity	36.10 (7.19)	31.12 (3.80)	.026

Variable	Completers (n = 21) Mean (SD)	Dropouts (n = 8) Mean (SD)	p-value
Total PTSD symptom count	14.15 (2.60)	12.38 (1.51)	.035
Cluster B (Intrusion) severity	2.45 (0.51)	1.38 (0.92)	.012
Cluster C (Avoidance) severity	1.70 (0.47)	1.25 (0.46)	.038
Cluster B symptom count	*0.80 (0.41)*	*1.00 (0.00)*	*.042*
Cluster D1 severity	0.95 (1.23)	0.25 (0.71)	.073
Cluster E total symptom count	3.90 (1.02)	3.12 (0.99)	.086
Cluster C2 symptom count	0.75 (0.44)	0.38 (0.52)	.098
Cluster E1 severity	1.70 (0.98)	1.00 (0.93)	.098
Cluster D total severity	13.20 (4.12)	12.00 (2.56)	.364
Cluster E total severity	9.85 (2.37)	8.12 (2.70)	.141

Supplementary Table 2. Exploratory Analyses Including THC-COOH Concentration as a Covariate in Mixed-Effects Models Predicting PTSD Outcomes

Outcome	Predictor	Estimate (β)	SE	t(df)	p-value
CAPS-5 Total Severity	Time	**-1.03**	0.21	-4.86	**<.001**
	Continued Abstinence	3.24	4.37	0.74	.468
	THC-COOH (ng/mL)	**0.015**	0.005	2.86	**.006**
	Age	0.12	0.25	0.50	.627
	Gender	-1.66	2.41	-0.69	.502
	FND_Total	-0.20	1.39	-0.15	.885
	CUDIT-R	-0.12	0.51	-0.24	.811
	Education (years)	-0.59	0.82	-0.72	.483
	Baseline MWC	0.24	0.30	0.80	.438
	Race	-0.28	0.43	-0.64	.532
	Income	-0.45	0.81	-0.55	.593
	Time × Continued Abstinence	**-0.68**	0.31	-2.21	**.031**
CAPS-5 Total Symptom Count	Time	**-0.37**	0.09	-4.04	**<.001**
	Continued Abstinence	1.36	1.73	0.79	.441
	THC-COOH (ng/mL)	**0.006**	0.002	2.78	**.007**
	Age	0.02	0.10	0.26	.800
	Gender	-0.64	0.94	-0.68	.507
	FND_Total	0.04	0.54	0.07	.949
	CUDIT-R	-0.05	0.20	-0.26	.801
	Education (years)	-0.23	0.32	-0.71	.492
	Baseline MWC	0.09	0.12	0.76	.461

Outcome	Predictor	Estimate (β)	SE	t(df)	p-value
	Race	-0.08	0.17	-0.47	.650
	Income	-0.14	0.32	-0.45	.663
	Time × Continued Abstinence	**-0.30**	0.13	-2.28	**.026**

Supplementary Figure 1. CONSORT Diagram