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PTSD symptom clusters and craving differs by primary drug of choice

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Abstract

Objective: Research has demonstrated a cyclical relationship between posttraumatic stress disorder (PTSD) and substance use disorder (SUD). Identifying factors that link PTSD symptom clusters and SUD may illuminate mechanisms underlying the PTSD-SUD relationship, better informing interventions that target this comorbidity. The current study of individuals enrolled in an outpatient aftercare chemical dependency program in King County, Washington assessed whether overall PTSD symptoms, and specific PTSD symptom clusters predicted craving depending on individuals identified primary drug of choice (DOC).

Methods: Participants eligible for the parent study were at least 18 years of age, fluent in English, medically cleared from substance withdrawal, able to participate in treatment sessions, and agreed to random assignment. Random assignment to either a mindfulness-based relapse prevention group, a standard relapse prevention group, or a treatment as usual group was conducted on a computer randomization program. A secondary analysis of baseline data was employed in the current study to determine which of the PTSD symptom clusters (avoidance, hyperarousal, and intrusion) predicted substance craving.

Results: Co-varying for severity of dependence, results suggest that overall PTSD scores predicted craving in participants who identified alcohol, stimulants, and opiates as their primary DOC. Further, avoidance-related PTSD symptoms alone predicted a significant proportion of the variability in craving in stimulant users, and hyperarousal symptoms alone predicted a significant proportion of the variability in craving in alcohol users. No specific PTSD cluster significantly predicted a proportion of the variability in craving in marijuana or opiates users.

Conclusions: Findings suggest craving may play a role in maintaining the relationship between specific PTSD symptom clusters and SUD, and the nature of this relationship may differ by primary DOC. The clinical trial on which this secondary analysis of data was conducted is registered as , at www.clinicaltrials.gov.

Keywords

Substance Use Disorder; Posttraumatic Stress Disorder; PTSD symptom clusters; craving; dual diagnosis; self-medication hypothesis

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Disclosures

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Introduction

Posttraumatic stress disorder (PTSD) is characterized by symptoms appearing after exposure to actual or threatened death, traumatic injury, or sexual assault, with symptoms categorized into four distinct clusters: Intrusion, Avoidance, Negative Alterations in Cognition and Mood, and Alterations in Arousal and Reactivity (American Psychiatric Association, 2013). In the general population, past-year prevalence rate of PTSD is approximately 4.7%, while lifetime prevalence rate is 6.1% (Goldstein et al., 2016). However, prevalence rates vary by population, type of trauma, and gender (Kilpatrick et al., 2013).

PTSD frequently co-occurs with substance use disorder (SUD; Grant et al., 2016). For example, among persons enrolled in inpatient SUD treatment, approximately 14% had a current diagnosis of PTSD (Chen et al., 2011). Similarly, a Dutch study of 423 patients in SUD treatment found that almost 37% met diagnostic criteria for current PTSD (Gielen, Havermans, Tekelenburg, & Jansen, 2012). The National Epidemiologic Survey on Alcohol and Related Conditions (N = 34,653) estimated 47% of persons with PTSD in the U.S. met criteria for SUD (Pietrzak, Goldstein, Southwick, & Grant, 2011) , and a Australian National Survey of Mental Health indicated nearly 35% of persons who met criteria for PTSD also met criteria for SUD (Mill, Teeson, Ross, & Peters, 2006). Rates of comorbid PTSD and SUD have been reported higher in some populations, such as combat veterans (Seal, Cohen, Waldrop, Cohen, Maguen, & Ren, 2011) and incarcerated women (Wolff, Frueh, Shi, Gerardi, Fabrikant, & Schumann, 2011).

A significant body of research supports the relationship between PTSD symptom severity and SUD severity (see reviews by Leeies, Pagura, Sareen, & Bolton, 2010; Conrad & Stewart, 2003; Jacobsen et al., 2001). It is essential to improve our understanding of this relationship, as persons with both disorders present with worse psychological functioning and greater psychiatric problems than persons with SUD alone (Flanagan, Korte, Killeen, & Back, 2016). Patients in residential SUD treatment with PTSD report greater overall emotion dysregulation and difficulty controlling impulsive behavior when distressed compared to patients without PTSD (Weiss, Tull, Anestis, & Gratz, 2012). Similarly, patients in SUD treatment who report higher PTSD scores also report higher scores on craving, depression, anxiety and stress (Wieferink, de Haan, Dijkstra, Fledderus, & Kok, 2017). Further, persons in SUD treatment with co-occurring PTSD have worse treatment outcomes than those with SUD only, including treatment noncompliance and shorter time to relapse (see review by Najt, Fusar-Poli, & Brambilla, 2011). Remission of PTSD symptoms, however, is predictive of abstinence from substance use up to five years following SUD treatment (Coffey, Schumacher, Brady, & Dansky Cotton, 2007; Hein et al., 2010), pointing to the interconnected nature of these disorders.

The relationship between PTSD and SUD is important to understand as persons who have experienced trauma, but do not meet diagnostic criteria for PTSD, still are at elevated risk for negative mental health outcomes. For example, persons with subthreshold PTSD symptoms report greater psychological impairment compared to trauma exposed persons without PTSD (Mylle & Maes, 2004), and subthreshold PTSD symptoms are consistently

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related to elevated risk for psychiatric conditions (Grubaugh, et al., 2005; Mota, et al., 2016). In addition, trauma exposed persons with subthreshold PTSD have greater risk for binge drinking compared to trauma exposed persons without PTSD (Boscarino, & Adams, 2009). There seem to be population-specific patterns, such as subthreshold PTSD as a risk factor for substance misuse in college students (Borsari, Read, & Campbell, 2008, 2009), and association of lifetime subthreshold PTSD with greater likelihood of lifetime SUD in military veterans (Mota, Tsai, Sareen, Marx, Wiscco, et al., 2016).

Although several models have been offered to explain co-occurring PTSD-SUD, the self-medication hypothesis (Khantzian, 1985) stands as the most accepted (Kramer, Polusny, Arbisi, & Krueger, 2014). This hypothesis posits that individuals use, and eventually become addicted to, substances as a way to cope with distressing PTSD symptoms acquired prior to use. This has been supported by numerous studies (Dworkin, Wanklyn, Stasjewicz, & Coffey, 2018; Ertl, Saile, Neuner, & Catani, 2016; Garland, Pettus-Davis, & Howard, 2013; Shadur, Hussong, & Haroon, 2015; Simpson et al., 2012), including studies showing that fluctuations in PTSD and SUD symptoms occur simultaneously. One study found fluctuations in weekly PTSD symptom severity were associated with concurrent fluctuations in alcohol and cocaine dependence severity, and opiate dependence severity the following week (Ouimette, Read, Wade, & Tirone, 2010). Similarly, Kaysen and colleagues (2014) found greater daily fluctuations in PTSD symptom severity predicted greater urges to drink in a sample of college women who experienced sexual assault, specifically when they experienced intrusion and avoidance symptoms. Additionally, using ecological momentary assessment, Possemato et al. (2015) found a higher incidence of PTSD symptoms was associated with heavier drinking within a 3-hour block of time, but no increases in alcohol use were observed in subsequent time blocks.

Alongside studies of co-occurring PTSD-SUD are investigations of specific PTSD symptom clusters and substance use (Debell et al, 2015; Dworkin et al, 2017, 2018). Results from these studies vary, however. Increased hyperarousal and avoidance/numbing symptoms have been found in persons with cocaine, alcohol, opioid, cannabis, and amphetamine use disorders (Dworkin et al., 2018; Khoury, Tang, Bradley, Cubells, & Ressler, 2010; Afful, Strickland, Cottler, & Bierut, 2010; Smith et al., 2010; Smith et al., 2016), and increased intrusion symptoms have been found in persons with cocaine, alcohol, cannabis, and sedative use disorders (Avant, Davis, & Cranston, 2011; Khoury et al., 2010). Inconsistent findings highlight the need for further investigations to clarify these relationships.

The link between affective discomfort, craving and substance use may shed light on the substrates of the PTSD-SUD relationship. While there are multiple predictors of substance use, (e.g., negative affect; Slofstra et al., 2018; Witkiewitz & Bowen, 2010) and impulsivity (see review by Loree, Lundahl, & Ledgerwood, 2015), craving has consistently been shown to be a primary predictor in SUD populations (Weiss, 2005). The aversive and seemingly uncontrollable nature of PTSD symptoms may lead to craving in individuals with SUD, as research indicates negative affect and craving are positively correlated (Schlauch, Gwynn-Shapiro, Stasiewicz, Molnar, & Lang, 2013). Saladin et al. (2003) found that, among individuals with co-occurring PTSD-SUD, PTSD symptom severity, elicited by reading a trauma script, predicted substance craving regardless of whether substance cues were

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present. Further, substance cues only elicited craving when the trauma script preceded substance cues, suggesting that trauma cues elicit substance craving over substance use cues alone (Saladin et al., 2003). Moreover, management of craving appears to have a positive effect on substance use severity following treatment (Mo & Deane, 2016).

Isolating predictors of relapse, such as craving, could help identify specific factors underlying use in relation to PTSD (Bradizza, Stasiewicz, & Paas, 2006), and further enhance treatments for individuals with SUDs experiencing trauma-related symptoms. Interventions that target craving in individuals experiencing specific PTSD symptoms may decrease the likelihood of relapse. Further, investigating PTSD cluster-craving relationships within various drugs of choice (DOCs) may inform more individualized treatment approaches, and may enhance treatment gains.

While studies have demonstrated relationships between PTSD severity, craving, and substance use, there is limited understanding of links within these relationships for different DOCs. Studies demonstrate associations between specific SUDs and PTSD clusters (Dworkin, Wanklyn, Stasiewicz, & Coffey, 2018; Khoury et al., 2010; Afful et al., 2010; Smith et al., 2010, 2016; Avant, Davis, & Cranston, 2011; Khoury, Tang, Bradley, Cubells, & Ressler, 2010); however, no locatable literature examines whether specific PTSD symptom clusters differentially predict craving depending on DOC. Such an understanding may reveal patterns of attempting to self-medicate certain symptoms through a substance that provides specific effects. As craving is a primary predictor of relapse (Fatseas, Serre, Alexandre, Debrabant, Auriacombe, & Swendsen, 2015; Serre, Fatseas, Swendsen, & Auriacombe, 2015), it may provide a more nuanced understanding of which PTSD clusters predict craving for different DOCs.

The current study was a secondary analysis of baseline data collected from adults in SUD treatment (Bowen et al., 2014), and assessed which specific PTSD symptom clusters predicted craving among individuals stratified by primary DOC. Specifically, we hypothesized that overall PTSD symptom severity would predict craving for all primary DOC categories, and that specific symptom clusters would differentially predict craving for each DOC. In line with the self-medication hypothesis (Khantzian, 1985), we expected high levels of hyperarousal and/or avoidance symptoms would be associated with craving for substances such as marijuana, alcohol or opiates, versus stimulants. We also hypothesized that high levels of re-experiencing symptoms would predict craving for stimulants, as stimulant use often results in physical activity (NIDA, 2013), which may serve as a distraction from intrusive thoughts.

Methods

2.1 Participants

Data for the current study are drawn from the baseline assessment ($N = 257$) of an IRB approved, randomized-controlled trial conducted in a community-based outpatient aftercare program (Bowen, et al., 2014). Prior to aftercare, participants in the parent study had attended either inpatient or intensive outpatient SUD treatment. Oral and written informed consent was obtained. Eligible participants were fluent in English, medically cleared to

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attend aftercare, able to participate in treatment sessions, and agreed to random assignment. Participants were not required to meet criteria for a dual diagnosis in order to be included. Individuals were excluded from the original study if they had a current diagnosis of dementia or a psychotic disorder, were experiencing suicidality, were an imminent danger to others, or had participated in previous MBRP trials. Information regarding co-occurring mental health diagnoses was not reported. See Table 1 for participant demographics.

2.2 Measures

Paper-and-pencil self-report questionnaires were administered in the original study (Bowen et al., 2014), including information regarding SUD diagnosis and drug of choice. Measures included in the current study are listed below.

PTSD Checklist – Civilian version (PCL-C; Weathers et al., 1994).—The PCL-C is a 17-item Likert scale assessing Diagnostic and Statistical Manual for Mental Health Disorders-IV-Text Revision (DSM-IV-TR; APA, 2000) symptoms for PTSD over the past 30 days. Items can be totaled for overall symptom severity, or calculated for symptom cluster subscale scores. The PCL-C has shown good test–retest reliability and internal consistency (Blanchard et al., 1996; Ruggiero et al., 2003). Items range from 1 (*not at all*) to 5 (*extremely*). In the current study, internal consistency of total PCL-C was excellent (α = .93). Internal consistency for the re-experiencing and avoidance clusters were good (α = .86 and α = .85, respectively), and poor for hyperarousal (α = .67). The PCL-C, versus the PCL-5, was used because at the time of the original trial, the DSM-5 (APA, 2013), and thus PCL-5, was not yet available.

Penn Alcohol Craving Scale (PACS; Flannery, Volpicelli, & Pettinati, 1999).—The PACS is a 5-item Likert scale assessing intensity, duration, and frequency of substance cravings (Flannery, Volpicelli, & Pettinati, 1999). Items range from 0 (*never/least severe*) to 6 (*always/most severe*). The PACS was adapted to include craving for any substance of choice versus only alcohol (Bowen, Chawla, Collins, Witkiewitz, Hsu, Grow, et al. 2009). In the current study, internal consistency was good (α = .88).

Severity of Dependence Scale (SDS; Gossop et al., 1995).—The SDS is a 5-item Likert-type scale assessing degree of substance dependence. Items are rated from 0 (*never/almost never*) to 3 (*always/nearly always*). In the current study, internal consistency was good (α = .86).

2.3 Procedure

For the current study, data were split into four primary DOC categories: alcohol, stimulants (cocaine, and methamphetamine), opioids (heroin and prescription opioids) and marijuana. SPSS V.25 was used for all analyses.

2.4 Statistical Analyses

Data were checked for normality, and assumptions were met for multiple regression analyses. Two multiple regression analyses were run for each primary DOC group to determine whether PCL-C overall scores and which PCL-C subscale scores significantly

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predicted substance craving. Since severity of substance dependence is correlated with craving in the current study (r = .228; p <.000), SDS scores were added as covariates in the model. Gender (r = −.007, p = .458) and medication assisted treatment (r = −.057, p = .181) were not correlated with substance craving, and were not included in the model. Mean imputation was used for item-level missing data for participants with > 80% complete data, and excluded from analyses if they had < 80% missing item-level data (n = 8). For the first regression, SDS total was entered into step 1, and PCL-C overall score was entered in step 2. In the second regression, SDS total was entered into step 1, and PCL-C subscale scores were entered simultaneously into step 2. Clusters were entered simultaneously to assess which of the three clusters predicted substance craving when assessed together.

Results

Descriptive statistics and correlations between SDS, PCL-C and PACS can be found in Table 2.

For participants who identified alcohol as primary DOC (n = 131), PCL-C overall score predicted 18% of the variability in craving, $F(2, 128) = 14.061$, $p < .001$, $R^2 = .180$. When mean PCL-C cluster scores were entered as separate variables, the model was still significant, $F(4, 126) = 7.958$, $p < .001$, $R^2 = .202$; PTSD-related hyperarousal predicted a significant portion of variance in alcohol craving, $t(126) = 2.283$, $p = .024$, $R^2 = .033$.

For participants identifying stimulants as primary DOC (n = 66), PCL-C overall score predicted 15.7% of the variability in craving, $F(2, 63) = 5.886$, $p = .005$, $R^2 = .157$. When mean PCL-C cluster scores were entered as separate variables, the model was still significant, $F(4, 61) = 4.334$, $p = .004$, $R^2 = .221$; PTSD-related avoidance predicted a significant portion of variance in stimulant craving, $t(61) = 2.445$, $p = .017$, $R^2 = .076$.

For participants who identified opiates as primary DOC (n = 36), mean PCL-C overall score predicted 18.5% of the variability in craving, $F(2, 33) = 3.751$, $p = .034$, $R^2 = .185$. When PCL-C cluster scores were entered into the model as separate variables, no one cluster alone significantly contributed to variance accounted for in opiate craving, $F(4, 31) = 1.732$, $p = .168$, $R^2 = .183$.

For participants who identified marijuana as primary DOC (n = 24), PCL-C overall score did not significantly predict craving, $F(2, 21) = 2.290$, $p = .126$ $R^2 = .179$. When PCL-C cluster scores were entered into the model separately, no one cluster significantly predicted craving $F(4, 18) = 2.014$, $p = .113$, $R^2 = .298$; however, trauma-related hyperarousal accounted for 12.4% of the variability in craving, $t(19) = 1.834$, $p = .080$, $R^2 = .124$. (see Table 3).

Discussion

The current study examined relationships between PTSD clusters, SUD craving, and DOC among substance users attending SUD outpatient aftercare programs. Specifically, we assessed which PTSD symptom clusters predicted craving for different primary DOC. It was hypothesized that overall PTSD symptom severity would predict craving for all DOCs, and only specific PTSD clusters would predict craving based on identified DOC. Results

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suggested that, after controlling for severity of dependence, overall PTSD symptom severity significantly predicted craving for those who identified alcohol, stimulants, and opiates as primary DOC; however, it did not predict craving for those who identified marijuana as primary DOC. Thus, our first hypothesis, that overall PTSD symptom severity would predict craving for all primary DOC categories, was only partially confirmed. Results suggest use of alcohol, stimulants, and opiates may have been negatively reinforced, (i.e., individuals repeatedly used a specific substance to alleviate PTSD-related distress), which then increased the probability of future use and/or craving. This supports the self-medication hypothesis (Khantzian, 1985), and aligns with previous studies in which PTSD symptom severity significantly predicted craving among persons with co-occurring PTSD-SUD (Simpson et al., 2012).

Our second hypothesis posited that only specific PTSD symptom clusters would predict craving depending on primary DOC. Indeed, individuals endorsing marijuana as primary did not show a significant relationship between overall levels of PTSD, symptom clusters, and craving. To check whether non-significant results were due to lack of statistical power, a post hoc power analyses was conducted using G*Power (Faul, Erdfelder, Lang, & Buchner, 2007) with power $(1 - \beta)$ set at 0.80 and $\alpha = .05$, two-tailed. The power analysis indicated the sample size of $n = 77$ would be needed to detect a medium sized effect (0.15). The power of the current study sample size to detect medium effect size in those who identify marijuana as primary was 0.42. Thus, lack of significant findings may be due to limited number of participants identifying marijuana as primary DOC. Additionally, 91.0% of the sample who identified marijuana as primary DOC identified alcohol (58.3%), stimulants (20.8%) and opiates (4.2%) as secondary DOC; thus, marijuana may be used to counterbalance effects of the secondary DOC (e.g., alcohol withdrawal), and may therefore be unrelated to PTSD symptoms.

Results supporting our second hypothesis demonstrated that hyperarousal significantly predicted craving for those who identified alcohol as the primary DOC. This finding aligns with results from a study by Simpson, Stappenbeck, Varra, Moore, and Kaysen (2012), in which symptoms of hyperarousal, particularly startle response, anger, and irritability, predicted same-day craving for alcohol; the effect was stronger for women versus men. They also found symptoms of sleep disturbance and hypervigilance predicted next-day craving for alcohol, with no difference between genders. Given that alcohol is a CNS depressant, it may alleviate arousal and reactivity in individuals experiencing PTSD-related hyperarousal symptoms. Consequently, the anxiolytic properties of alcohol may negatively reinforce continued use. This is supported by studies suggesting alcohol use, AUD diagnosis, and longitudinal consequences of alcohol use are predicted by hyperarousal symptoms (Read, Colder, Merrill, Ouimette, White, & Swartout, 2012).

While our hypothesis that re-experiencing-related PTSD symptoms would predict craving for stimulants was not supported, results indicated avoidance-related PTSD symptoms significantly predicted craving for self-identified stimulant users, suggesting stimulant drugs may be used to avoid/escape PTSD-related distress. Previous studies found that PTSD symptom severity predicted craving among cocaine-dependent individuals in response to trauma cues (Khantzian, 1985; Saladin et al., 2003). Stimulant use increases motor activity

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for long periods of time (SAMHSA, 1999), and therefore may be used to initiate engagement in activities that distract from trauma cues. Additionally, initial euphoric effects of stimulants may shift focus from aversive PTSD symptoms to hedonic stimuli, providing temporary escape from distress.

No one specific PTSD symptom cluster predicted craving for persons who identified opioids as their primary DOC. Total PCL-C score predicted craving in the first regression analysis. However, when symptom clusters were entered as individual predictors, results were non-significant, indicating no one specific PTSD symptom cluster predicted craving. To assess whether non-significant results were due to lack of statistical power, a post hoc power analyses was set at 0.80 and $\alpha = .05$, two-tailed. Results indicated sample size would have to increase up to $n = 85$ to detect a significant medium sized effect. Power in the current study to detect medium effect size in those who identify opiates as primary DOC was 0.37.

Limitations of this study should be noted. First, a large proportion (81.3%) of participants endorsed polysubstance use, and identified alcohol as secondary DOC (21.8%); therefore, there may be confounding effects of use of multiple substances on craving. While this is a common challenge in substance use research, literature suggests polysubstance use profiles can vary based on primary substance of use (Connor, Gullo, White, & Kelly, 2014). Thus, examining whether PTSD symptoms predict craving for the primary substance in polysubstance users is warranted. Second, only 24 participants identified marijuana as primary DOC, and 36 endorsed opiates, which limited power to detect which individual clusters predicted craving in this subset. Third, the PCL-C was used to measure these data because, during the time of the original study, the PCL-5 was yet not available. The DSM-5, and subsequently the PCL-5, includes a fourth PTSD symptom cluster of negative alteration in mood and cognition. Variability in craving due to this cluster is thus not represented the PCL-C three-cluster model. Fifth, variables such as co-morbid psychiatric diagnoses and amount of time in treatment prior to aftercare are factors that impact craving, and were not assessed in the parent study. Finally, assessment of trauma exposure was not collected during the parent trial, thus elevated ratings of trauma symptom severity via the PCL-C may actually reflect symptoms of general distress or negative affect. While the PCL-C demonstrates good discriminant validity between related constructs (i.e., depression, anxiety, hostility, physical and emotional functioning; see review by Wilkins, Lang, & Norman, 2011), future studies should include a standardized assessment of trauma exposure in addition to PCL-C scores to better distinguish trauma symptoms from other psychological symptoms.

Findings from the current study further inform our understanding of the PTSD-SUD relationship. Specifically, craving elicited by PTSD symptoms may differ between individuals with different DOCs, shaped by a history of attempts to alleviate specific symptoms with specific effects of different substances. A more nuanced understanding of factors that underlie and perpetuate PTSD-SUD comorbidity can offer specificity to current treatments targeting dually-diagnosed individuals.

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References

Afful SE, Strickland JR, Cottler L, & Bierut LJ (2010). Exposure to trauma: compare son of cocaine-dependent cases and a community-matched sample. Drug and Alcohol Dependence, 112(1-2), 46–53. doi:10.1016/j.drugalcdep.2010.05.012 [PubMed: 20599330]

American Psychiatric Association. (2013). Diagnostic and statistical manual of mental disorders (5th ed.). Arlington, VA: American Psychiatric Publishing.

American Psychiatric Association (2000). Diagnostic and statistical manual of mental disorders (4th ed., Text Revision). Washington, DC: Author.

Avant EM, Davis JL, & Cranston CC (2011). Posttraumatic stress symptom clusters, trauma history, and substance use among college students. Journal of Aggression, Maltreatment and Trauma, 20, 539–555. doi:10.1080/10926771.2011.588153

Bonn-Miller MO, Vujanovic AA, & Drescher KD (2011). Cannabis use among military veterans after residential treatment for posttraumatic stress disorder. Psychology of Addictive Behaviors, 25(3), 485. doi:10.1037/a0021945 [PubMed: 21261407]

Bonn-Miller MO, Babson KA, & Vandrey R (2014). Using cannabis to help you sleep: Heightened frequency of medical cannabis use among those with PTSD. Drug and Alcohol Dependence, 136, 162–165. doi:10.1016/j.drugalcdep.2013.12.008 [PubMed: 24412475]

Borsari B, Read JP, & Campbell JF (2008). Posttraumatic stress disorder and substance use disorders in college students. Journal of College Student Psychotherapy, 22(3), 61–85. doi: 10.1080/87568220801960720 [PubMed: 19834572]

Boscarino JA, & Adams RE (2009). PTSD onset and course following the World Trade Center disaster: Findings and implications for future research. Social Psychiatry and Psychiatric Epidemiology, 44(10), 887–898.doi: 10.1007/s00127-009-0011-y [PubMed: 19277439]

Bowen S, Chawla N, Collins SE, Witkiewitz K, Hsu S, Grow J, … Marlatt A (2009). Mindfulness-based relapse prevention for substance use disorders: A pilot efficacy trial. Substance Abuse, 30(4), 295–305. doi:10.1080/08897070903250084 [PubMed: 19904665]

Bowen S, Witkiewitz K, Clifasefi SL, Grow J, Chawla N, Hsu SH, … Larimer ME (2014). Relative efficacy of mindfulness-based relapse prevention, standard relapse prevention, and treatment as usual for substance use disorders: A randomized clinical trial. JAMA Psychiatry, 71, 547–556. doi: 10.1001/jamapsychiatry.2013.4546 [PubMed: 24647726]

Bradizza CM, Stasiewicz PR, & Paas ND (2006). Relapse to alcohol and drug use among individuals diagnosed with co-occurring mental health and substance use disorders: A review. Clinical Psychology Review, 26(2), 162–178. doi:10.1016/j.cpr.2005.11.005 [PubMed: 16406196]

Buckner JD, Jeffries ER, Crosby RD, Zvolensky MJ, Cavanaugh CE, & Wonderlich SA (2018). The impact of PTSD clusters on cannabis use in a racially diverse trauma-exposed sample: An analysis from ecological momentary assessment. The American Journal of Drug and Alcohol Abuse, 44(5), 532–542. doi:10.1080/00952990.2018.1430149 [PubMed: 29442522]

Cavazos-Rehg P, Krauss M, Spitznagel E, Buckner-Petty S, Grucza R, & Bierut L (2015). Monitoring marijuana use and risk perceptions with Google Trends data. Drug & Alcohol Dependence, 146, e242–e243. doi:10.1016/j.drugalcdep.2014.09.126

Center for Substance Abuse Treatment. Treatment for Stimulant Use Disorders. Rockville (MD): Substance Abuse and Mental Health Services Administration (US); 1999 (Treatment Improvement

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Protocol (TIP) Series, No. 33.) Chapter 2—How Stimulants Affect the Brain and Behavior. Available from: https://www.ncbi.nlm.nih.gov/books/NBK64328/

Chen KW, Banducci AN, Guller L, Macatee RJ, Lavelle A, Daughters SB, & Lejuez CW (2011). An examination of psychiatric comorbidities as a function of gender and substance type within an inpatient substance use treatment program. Drug and Alcohol Dependence, 118(2-3), 92–99. doi: 10.1016/j.drugalcdep.2011.03.003 [PubMed: 21514751]

Coffey SF, Schumacher JA, Brady KT, & Cotton BD (2007). Changes in PTSD symptomatology during acute and protracted alcohol and cocaine abstinence. Drug and Alcohol Dependence, 87(2), 241–248. doi:10.1016/j.drugalcdep.2006.08.025 [PubMed: 17008029]

Coffey SF, Schumacher JA, Stasiewicz PR, Henslee AM, Baillie LE, & Landy N (2010). Craving and physiological reactivity to trauma and alcohol cues in posttraumatic stress disorder and alcohol dependence. Experimental and Clinical Psychopharmacology, 18(4), 340. doi:10.1037/a0019790 [PubMed: 20695690]

Connor JP, Gullo MJ, White A, & Kelly AB (2014). Polysubstance use: Diagnostic challenges, patterns of use and health. Current Opinion in Psychiatry, 27(4), 269–275. doi: 10.1097/YCO.0000000000000069. [PubMed: 24852056]

Conrad PJ, & Stewart SH (2003). Experimental studies exploring functional relations between posttraumatic stress disorder and substance use disorder In Ouimette P, & Brown PJ (Eds.). Trauma and substance abuse: Causes, consequences, and treatment of comorbid disorders (pp. 57–72). Washington, DC: American Psychological Association.

Cougle JR, Bonn-Miller MO, Vujanovic AA, Zvolensky MJ, & Hawkins KA (2011). Posttraumatic stress disorder and cannabis use in a nationally representative sample. Psychology of Addictive Behaviors, 25(3), 554. doi:10.1037/a0023076 [PubMed: 21480682]

Debell F, Fear NT, Head M, Batt-Rawden S, Greenberg N, Wessely S, & Goodwin L (2014). A systematic review of the comorbidity between PTSD and alcohol misuse. Social Psychiatry and Psychiatric Epidemiology, 49(9), 1401–1425. doi:10.1007/s00127-014-0855-7 [PubMed: 24643298]

Dworkin ER, Mota NP, Schumacher JA, Vinci C, & Coffey SF (2017). The unique associations of sexual assault and intimate partner violence with PTSD symptom clusters in a traumatized substance-abusing sample. Psychological Trauma: Theory, Research, Practice, and Policy, 9(4), 500. doi: 10.1037/tra0000212

Dworkin ER, Wanklyn S, Stasiewicz PR, & Coffey SF (2018). PTSD symptom presentation among people with alcohol and drug use disorders: Comparisons by substance of abuse. Addictive Behaviors, 76, 188–194. doi: 10.1016/j.addbeh.2017.08.019 [PubMed: 28846939]

Ertl V, Saile R, Neuner F, & Catani C (2016). Drinking to ease the burden: A cross-sectional study on trauma, alcohol abuse and psychopathology in a post-conflict context. BioMed Central Psychiatry, 16(1), 1–13. doi:10.1186/s12888-016-0905-7 [PubMed: 26739960]

Fatseas M, Serre F, Alexandre JM, Debrabant R, Auriacombe M, & Swendsen J (2015). Craving and substance use among patients with alcohol, tobacco, cannabis or heroin addiction: A comparison of substance- and person-specific cues. Addiction, 110(6), 1035–1042. doi:10.1111/add.12882 [PubMed: 25688760]

Faul F, Erdfelder E, Lang AG, & Buchner A (2007). G* Power 3: A flexible statistical power analysis program for the social, behavioral, and biomedical sciences. Behavior Research Methods, 39(2), 175–191. [PubMed: 17695343]

Flanagan JC, Korte KJ, Killeen TK, & Back SE (2016). Concurrent treatment of substance use and PTSD. Current Psychiatry Reports, 18(8), 70. doi:10.1007/s11920-016-0709-y [PubMed: 27278509]

Flannery BA, Volpicelli JR & Pettinati HM (1999). Psychometric properties of the Penn Alcohol Craving Scale. Alcoholism: Clinical and Experimental Research, 23(8), 1289–1295.doi:10.1111/j.1530-0277.1999.tb04349.x

Garland EL, Pettus-Davis C, & Howard MO (2013). Self-medication among traumatized youth: Structural equation modeling of pathways between trauma history, substance misuse, and psychological distress. Journal of Behavioral Medicine, 36(2), 175–185. doi:10.1007/s10865-012-9413-5 [PubMed: 22454227]

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

Gielen N, Havermans R, Tekelenburg M, & Jansen A (2012). Prevalence of post-traumatic stress disorder among patients with substance use disorder: It is higher than clinicians think it is. European Journal of Psychotraumatology, 3(1), 17734. doi:10.3402/ejpt.v3i0.17734

Goldstein RB, Smith SM, Chou SP, Saha TD, Jung J, Zhang H, … & Grant BF (2016). The epidemiology of DSM-5 posttraumatic stress disorder in the United States: Results from the National Epidemiologic Survey on Alcohol and Related Conditions-III. Social Psychiatry and Psychiatric Epidemiology, 51(8), 1137–1148. doi:10.1007/s00127-016-1208-5 [PubMed: 27106853]

Grant BF, Saha TD, Ruan WJ, Goldstein RB, Chou SP, Jung J, … & Hasin DS (2016). Epidemiology of DSM-5 drug use disorder: Results from the National Epidemiologic Survey on Alcohol and Related Conditions-III. JAMA Psychiatry, 73(1), 39–47. doi:10.1001/jamapsychiatry.2015.2132 [PubMed: 26580136]

Grubaugh AL, Magruder KM, Waldrop AE, Elhai JD, Knapp RG, & Frueh BC (2005). Subthreshold PTSD in primary care: Prevalence, psychiatric disorders, healthcare use, and functional status. The Journal of Nervous and Mental Disease, 193(10), 658–664. [PubMed: 16208161]

Gossop M, Darke S, Griffiths P, Hando J, Powis B, Hall W, & Strang J (1995). The Severity of Dependence Scale (SDS): Psychometric properties of the SDS in English and Australian samples of heroin, cocaine and amphetamine users. Addiction, 90(5), 607–614. [PubMed: 7795497]

Hien DA, Jiang H, Campbell AN, Hu MC, Miele GM, Cohen LR, … & Suarez-Morales L (2010). Do treatment improvements in PTSD severity affect substance use outcomes? A secondary analysis from a randomized clinical trial in NIDA's Clinical Trials Network. American Journal of Psychiatry, 167(1), 95–101. doi:10.1176/appi.ajp.2009.09091261 [PubMed: 19917596]

Hill MN, Bierer LM, Makotkine I, Golier JA, Galea S, Mcewen BS, Hillard CJ, & Yehuda R (2013). Reductions in circulating endocannabinoid levels in individuals with post-traumatic stress disorder following exposure to the world trade center attacks. Psychoneuroendocrinology, 38, 2952–61. doi: 10.1016/j.psyneuen.2013.08.004 [PubMed: 24035186]

Jacobsen LK, Southwick SM, & Kosten TR (2001). Substance use disorders in patients with posttraumatic stress disorder: A review of the literature. American Journal of Psychiatry, 158(8), 1184–1190. doi: 10.1176/appi.ajp.158.8.1184 [PubMed: 11481147]

Kaysen D, Atkins DC, Simpson TL, Stappenbeck CA, Blayney JA, Lee CM, & Larimer ME (2014). Proximal relationships between PTSD symptoms and drinking among female college students: Results from a daily monitoring study. Psychology of Addictive Behaviors: Journal of the Society of Psychologists in Addictive Behaviors, 28(1), 62–73. doi:10.1037/a0033588 [PubMed: 23915369]

Khoury L, Tang Y, Bradley B, Cubells J, & Ressler K (2010). Substance use, childhood traumatic experience, and posttraumatic stress disorder in an urban civilian population. Depression and Anxiety, 27(12), 1077–1086. doi:10.1002/da.20751 [PubMed: 21049532]

Khantzian EJ (1985). The self-medication hypothesis of addictive disorders: Focus on heroin and cocaine dependence. American Journal of Psychiatry, 142, 1259–1264. [PubMed: 3904487]

Kilpatrick DG, Resnick HS, Milanak ME, Miller MW, Keyes KM, & Friedman MJ (2013). National estimates of exposure to traumatic events and PTSD prevalence using DSM-IV and DSM-5 criteria. Journal of Traumatic Stress, 26(5), 537–547. doi:10.1002/jts.21848 [PubMed: 24151000]

Kramer MD, Polusny MA, Arbisi PA, & Krueger RF (2014). Comorbidity of PTSD and SUDs: Toward an etiologic understanding In Ouimette P & Read JP (Eds.), Trauma and substance abuse: Causes, consequences, and treatment of comorbid disorders (pp. 53–75). Washington, DC, US: American Psychological Association.

Leeies M, Pagura J, Sareen J, & Bolton J (2010). The use of alcohol and drugs to self-medicate symptoms of posttraumatic stress disorder. Depression and Anxiety, 27(8), 731–736. doi: 10.1002/da.20677 [PubMed: 20186981]

Loree AM, Lundahl LH, & Ledgerwood DM (2015). Impulsivity as a predictor of treatment outcome in substance use disorders: Review and synthesis. Drug and Alcohol Review, 34(2), 119–134. doi: 10.1111/dar.12132 [PubMed: 24684591]

Mo C & Deane F (2016). Reductions in craving and negative affect predict 3-month post-discharge alcohol use following residential treatment. International Journal of Mental Health Addiction, 14, 761–774. doi:10.1007/s11469-015-9626-2

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

Mota NP, Tsai J, Sareen J, Marx BP, Wisco BE, Harpaz-Rotem I, … & Pietrzak RH (2016). High burden of subthreshold DSM-5 post-traumatic stress disorder in US military veterans. World Psychiatry, 15(2), 185. doi: 10.1002/wps.20313 [PubMed: 27265715]

Mylle J, & Maes M (2004). Partial posttraumatic stress disorder revisited. Journal of Affective Disorders, 78(1), 37–48.doi: 10.1016/S0165-0327(02)00218-5 [PubMed: 14672795]

Naj P, Fusar-Pol P, & Brambill P (2011). Co-occurring mental and substance abuse disorders: A review on the potential predictors and clinical outcomes. Psychiatry Research, 186(2-3), 159–164. doi: 10.1016/j.psychres.2010.07.042 [PubMed: 20728943]

National Institute on Drug Abuse (NIDA; 2013). Methamphetamine. Retrieved on 02/05/2019 from https://www.drugabuse.gov/publications/research-reports/methamphetamine/what-are-immediate-short-term-effects-methamphetamine-abuse

Neumeister A (2013). The endocannabinoid system provides an avenue for evidence-based treatment development for PTSD. Depression and Anxiety, 30, 93–6. doi:10.1002/da.22031 [PubMed: 23225490]

Neumeister A, Seidel J Ragen BJ, & Pietrzak RH (2015). Translational evidence for a role of endocannabinoids in the etiology and treatment of posttraumatic stress disorder. Psychoneuroendocrinology, 51, 577–584. doi: 10.1016/j.psyneuen.2014.10.012 [PubMed: 25456347]

Ouimette PC, Read JP, Wade M, & Tirone V (2010). Modeling associations between posttraumatic stress symptoms and substance use. Addictive Behaviors, 35, 64–67. doi: 10.1016/j.addbeh.2009.08.009 [PubMed: 19729250]

Pietrzak RH, Goldstein RB, Southwick SM, & Grant BF (2011). Prevalence and axis I comorbidity of full and partial posttraumatic stress disorder in the United States: Resultsfrom wave 2 of the National Epidemiologic Survey on Alcohol and Related Conditions. Journal of Anxiety Disorders, 25(3), 456–465. doi: 10.1016/j.janxdis.2010.11.010 [PubMed: 21168991]

Possemato K, Maisto SA, Wade M, Barrie K, McKenzie S, Lantinga LJ, & Ouimette P (2015). Ecological momentary assessment of PTSD symptoms and alcohol use in combat veterans. Psychology of Addictive Behaviors, 29(4), 894. doi:10.1037/adb0000129. [PubMed: 26727007]

Read JP, Colder CR, Merrill JE, Ouimette P, White J, & Swartout A (2012). Trauma and posttraumatic stress symptoms predict alcohol and other drug consequence trajectories in the first year of college. Journal of Consulting and Clinical Psychology, 80(3), 426. doi: 10.1037/a0028210. [PubMed: 22545739]

Ruggiero KJ, Del Ben K, Scotti JR, & Rabalais AE (2003). Psychometric properties of the PTSD Checklist—Civilian Version. Journal of Traumatic Stress, 16(5), 495. doi:10.1023/A:1025714729117 [PubMed: 14584634]

Saladin ME, Drobes DJ, Coffey SF, Dansky BS, Brady KT, & Kilpatrick DG (2003). PTSD symptom severity as a predictor of cue-elicited drug craving in victims of violent crime. Addictive Behaviors, 28(9), 1611–1629. [PubMed: 14656549]

Schlauch RC, Gwynn-Shapiro D, Stasiewicz PR, Molnar DS, & Lang AR (2013). Affect and craving: Positive and negative affect are differentially associated with approach and avoidance inclinations. Addictive Behaviors, 38(4), 1970–1979. doi:10.1016/j.addbeh.2012.12.003. [PubMed: 23380493]

Seal KH, Cohen G, Waldrop A, Cohen BE, Maguen S, & Ren L (2011). Substance use disorders in Iraq and Afghanistan veterans in VA healthcare, 2001–2010: Implications for screening, diagnosis and treatment. Drug and Alcohol Dependence, 116(1-3), 93–101. doi: 10.1016/j.drugalcdep.2010.11.027 [PubMed: 21277712]

Serre F, Fatseas M, Swendsen J, & Auriacombe M (2015). Ecological momentary assessment in the investigation of craving and substance use in daily life: A systematic review. Drug & Alcohol Dependence, 148, 1–20. doi: 10.1016/j.drugalcdep.2014.12.024

Shadur JM, Hussong AM, & Haroon M (2015). Negative affect variability and adolescent self-medication: The role of the peer context. Drug and Alcohol Review, 34, 571–580. doi: 10.1111/dar.12260 [PubMed: 25867550]

Simpson TL, Stappenbeck CA, Varra AA, Moore SA, & Kaysen D (2012). Symptoms of posttraumatic stress predict craving among alcohol treatment seekers: Results of a daily monitoring study. Psychology of Addictive Behaviors, 26(4), 724–33. doi: 10.1037/a0027169 [PubMed: 22369221]

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Slofstra C, Nauta MH, Bringmann LF, Klein NS, Albers CJ, Batalas N, … Bockting CL (2018). Individual negative affective trajectories can be detected during different depressive relapse prevention strategies. Psychotherapy and Psychosomatics, 87(4)1–3. doi:10.1159/000489044 [PubMed: 29306948]

Smith KZ, Smith PH, Cercone SA, McKee SA, & Homish GG (2016). Past year non-medical opioid use and abuse and PTSD diagnosis: Interactions with sex and associations with symptom clusters. Addictive Behaviors, 58, 167–174. doi: 10.1016/j.addbeh.2016.02.019 [PubMed: 26946448]

Smith RC, Blumenthal H, Badour C, & Feldner MT (2010). An investigation of relations between crystal methamphetamine use and posttraumatic stress disorder. Addictive Behaviors, 35, 625–627. doi: 10.1016/j.addbeh.2010.01.010 [PubMed: 20153121]

Tul MT, Berghof CR, Wheeles LE, Cohe RT, & Grat KL (2018). PTSD symptom severity and emotion regulation strategy use during trauma cue exposure among patients with substance use disorders: Associations with negative affect, craving, and cortisol reactivity. Behavior Therapy, 49(1), 57–70. doi: 10.1016/j.beth.2017.05.005. [PubMed: 29405922]

Weathers FW, Litz BT, Herman DS, Huska JA, & Keane TM (1993). The PTSD Checklist (PCL): Reliability, validity, and diagnostic utility Paper presented at the meeting of the International Society of Traumatic Stress Studies, San Antonio, TX.

Weiss F (2005). Neurobiology of craving, conditioned reward and relapse. Current Opinion in Pharmacology, 5(1), 9–19. doi:10.1016/j.coph.2004.11.001 [PubMed: 15661620]

Weiss NH, Tull MT, Anestis MD, & Gratz KL (2013). The relative and unique contributions of emotion dysregulation and impulsivity to posttraumatic stress disorder among substance dependent inpatients. Drug and Alcohol Dependence, 128(1-2), 45–51.doi: 10.1016/j.drugalcdep.2012.07.017 [PubMed: 22917752]

Wieferink CE, de Haan HA, Dijkstra BA, Fledderus M, & Kok T (2017). Treatment of substance use disorders: Effects on patients with higher or lower levels of PTSD symptoms. Addictive Behaviors, 74, 122–126. doi:10.1016/j.addbeh.2017.06.005 [PubMed: 28622616]

Wilkins KC, Lang AJ, & Norman SB (2011). Synthesis of the psychometric properties of the PTSD checklist (PCL) military, civilian, and specific versions. Depression & Anxiety, 28(7), 596–606. https://doi-org.proxy.lib.pacificu.edu:2443/10.1002/da.20837 [PubMed: 21681864]

Witkiewitz K, & Bowen S (2010). Depression, craving, and substance use following a randomized trial of Mindfulness-Based Relapse Prevention. Journal of Consulting and Clinical Psychology, 78(3), 362.doi: 10.1037/a0019172 [PubMed: 20515211]

Wolff N, Frueh B, Shi J, Gerardi D, Fabrikant N, & Schumann B (2011). Trauma exposure and mental health characteristics of incarcerated females self-referred to specialty PTSD treatment. Psychiatric Services, 62(8), 954–8.doi:10.1176/ps.62.8.pss6208_0954 [PubMed: 21807837]

Yarnell S (2015). The use of medicinal marijuana for posttraumatic stress disorder: A review of the current literature. The Primary Care Companion for CNS Disorders, 17(3), 10.4088/PCC. 15r01786. doi:10.4088/PCC.15r01786. doi: 10.4088/PCC.15r01786

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Table 1:

Sample Demographic Characteristics

Demographic Factors	**(*N* = 257)**
Age	***M* (*SD*)**
	38.51 (11.03)
Gender	***n* (%)**
Male	182 (70.8)
Female	70 (27.2)
Other	1 (.4)
Race/Ethnicity	
White	128 (49.8)
Black	53 (20.2)
Hispanic	22 (8.6)
Asian	2 (.8)
Native American	16 (6.2)
Mixed	26 (10.3)
Other	9 (3.5)
Education	
≤ GED	82 (31.6)
≥ High School	174 (68.0)
Household Annual Income	
0-$4,999	151 (58.8)
$5,000-$9,999	25 (9.7)
> $10,000	43 (16.7)
Unknown	34 (13.2)
Primary drug of choice	
Alcohol	131 (51.0)
Stimulants	66 (25.7)
Opiates	36 (14.0)
Marijuana	24 (9.3)
Prescribed medication-assisted treatment	
	36 (14.2)

Note. GED = general education development.

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Table 2.

Means and Correlations of PTSD Symptom Severity, Craving, and Severity of Dependence.

Primary DOC	*M* (*SD*)	1	2	3	4	5	6
Alcohol *n* = 131							
1. PTSD Symptom Severity	35.83 (12.93)	-					
2. Hyperarousal	9.40 (3.40)	.869**	-				
3. Avoidance	2.28 (0.85)	.942**	.731**	-			
4. Re-experiencing	2.19 (0.80)	.874**	.660**	.717**	-		
5. Craving	6.56 (5.10)	.424**	.429**	.396**	.359**	-	
6. Severity of Dependence	8.97 (4.11)	.319**	.328**	.309**	.273**	.142*	-
Stimulants *n* = 66							
1. PTSD Symptom Score	34.48 (12.06)	-					
2. Hyperarousal	8.67 (3.44)	.869**	-				
3. Avoidance	2.23 (0.82)	.953**	.793**	-			
4. Re-experiencing	2.11 (0.80)	.874**	.660**	.772**	-		
5. Craving	5.23 (4.17)	.363**	.334**	.405**	.233*	-	
6. Severity of Dependence	9.61 (3.86)	.248*	.206*	.201*	.300**	.245*	-
Opiates *n* = 36							
1. PTSD Symptom Score	34.08 (10.28)	-					
2. Hyperarousal	8.92 (2.98)	.830**	-				
3. Avoidance	2.18 (0.79)	.934**	.686**	-			
4. Re-experiencing	2.01 (0.66)	.799**	.544**	.607**	-		
5. Craving	9.05 (5.47)	.350*	.287*	.305*	.322*	-	
6. Severity of Dependence	12.28 (2.68)	−.207	−.244*	−.272	.003	.163	-
Marijuana *n* = 24							
1. PTSD Symptom Score	33.62 (11.49)	-					
2. Hyperarousal	9.00 (3.32)	.844**	-				
3. Avoidance	2.14 (0.86)	.869**	.582**	-			
4. Re-experiencing	2.00 (0.85)	.844**	.636**	.596**	-		
5. Craving	6.16 (4.98)	.356*	.446**	.229	.180	-	
6. Severity of Dependence	5.46 (3.89)	.424*	.390*	.241	.474**	.379*	-

Note. PTSD = posttraumatic stress disorder; DOC = drug of choice.

* $p < .05$.

** $p < .001$.

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Table 3.

Multiple Regression Analyses Predicting Substance Craving by PTSD Symptom Severity and by Trauma Symptom Clusters while Covarying Severity of Dependence.

	Primary Drug of Choice																			
Predictor	**Alcohol**					**Stimulants**					**Opiates**					**Marijuana**				
	F	R^2	ΔR^2	β	***t***	***F***	R^2	ΔR^2	β	***t***	***F***	R^2	ΔR^2	β	***t***	***F***	R^2	ΔR^2	β	***t***
PTSD Overall Severity	14.016***	.180	.160	.422	4.997***	5.886**	.157	.097	.322	2.695**	3.751*	.185	.159	.407	2.536*	2.290	.179	.035	.207	.948
Clusters	7.958***	.202	.161	-	-	4.334**	.221	.161	-	-	1.732	.183	.156	-	-	2.014	.298	.154	-	-
Avoidance			.007	.141	1.071			.076	.542	2.445*			.017	.200	.804			.002	.055	.218
Hyperarousal			.033	.281	2.283*			.001	.044	.237			.011	.147	.634			.124	.490	1.834
Re-experiencing			.003	.076	.640			.029	−.278	−1.512			.008	.120	.556			.047	−.320	−1.129

Note. Craving as measured by the Penn Alcohol Craving Scale (PACS), modified for substance craving. PTSD = posttraumatic stress disorder.

* $p < .05$.

** $p < .01$.

*** $p < .001$.

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