HHS Public Access

Author manuscript

Addict Behav. Author manuscript; available in PMC 2019 January 01.

Published in final edited form as:

Addict Behav. 2018 January ; 76: 188–194. doi:10.1016/j.addbeh.2017.08.019.

PTSD symptom presentation among people with alcohol and drug use disorders: Comparisons by substance of abuse

Emily R. Dworkin[*,1,2], **Sonya Wanklyn**[1,2], **Paul R. Stasiewicz**[3], and **Scott F. Coffey**[2]

[1]G.V. (Sonny) Montgomery Veteran Affairs Medical Center, 1500 E Woodrow Wilson Ave, Jackson, MS 39216, USA

[2]University of Mississippi Medical Center, 2500 North State Street, Jackson, MS 39216, USA

[3]Research Institute on Addictions, University at Buffalo, 1021 Main Street, Buffalo, NY 14203-1016, USA

Abstract

Posttraumatic stress disorder (PTSD) and substance use disorders (SUDs) commonly co-occur, and there is some evidence to suggest that PTSD symptom clusters are differentially related to various substances of abuse. However, few studies to date have compared PTSD symptom patterns across people with different types of SUDs, and fewer still have accounted for the presence of comorbidity across types of SUDs in understanding symptom patterns. Thus, in the current study, we use a treatment-seeking sample of people with elevated symptoms of PTSD and problem alcohol use to explore differential associations between past-year SUDs with active use and PTSD symptoms, while accounting for the presence of multiple SUDs. When comparing alcohol and drug use disorders, avoidance symptoms were elevated in those with alcohol use disorder, and hyperarousal symptoms were elevated in those who had a drug use disorder. In the subsample with alcohol use disorder, hyperarousal symptoms were elevated in people with co-occurring cocaine use disorders and numbing symptoms were elevated in people with co-occurring sedative/hypnotic/anxiolytic use disorder. These findings provide evidence for different symptom cluster patterns between PTSD and various types of SUD and highlight the importance of examining the functional relationship between specific substances of abuse when understanding the interplay between PTSD and SUD.

[*]Corresponding author.

Contributors

Author SC designed the study and collected data. Author ED conceptualized the research question with assistance from the other authors and conducted data analyses in consultation with PS. Authors ED and SW wrote the first draft of the manuscript, and all authors contributed to and approved the final manuscript.

Conflict of Interest

All authors declare that they have no conflicts of interest.

Keywords

posttraumatic stress; substance use disorders; PTSD symptom clusters

Posttraumatic stress disorder (PTSD) is common among people seeking treatment for substance use disorders (SUDs) (Jacobsen, Southwick, & Kosten, 2001), and research has increasingly attempted to understand the mechanisms accounting for this comorbidity. However, potential differential associations between PTSD symptoms based on different substances of abuse have been underexplored. Clarifying associations between PTSD symptoms and different substances of abuse could inform theory and intervention development for co-occurring PTSD and SUD. Thus, this study used a sample of 208 men and women in residential treatment for SUD to understand how substances of abuse were associated with PTSD symptom presentations.

Several theories of the co-occurrence of SUD and PTSD have been proposed, including the shared vulnerability hypothesis, which suggests that the co-occurrence of these disorders is due to shared risk factors (Chilcoat & Breslau, 1998); the susceptibility hypothesis, which posits that substance use peripheral to trauma impedes natural recovery from PTSD symptoms; the high-risk hypothesis, that asserts that risky behaviors commonly associated with substance use increase risk for trauma exposure and, therefore, PTSD (Chilcoat & Breslau, 1998); the self-medication hypothesis, that argues that individuals with PTSD use substances to alleviate emotional distress and cope with PTSD symptoms (Khantzian, 1985; Stewart, 1996); and the mutual maintenance theory, which proposes that PTSD promotes SUD which, in turn, maintains PTSD symptoms (Kaysen et al., 2011). Support has been documented for each of these theories (Begle et al., 2011; Coffey, Schumacher, Brady, & Cotton, 2007; Kaysen et al., 2011; Read, Wardell, Colder, 2013), although research appears to support a self-medication pathway between SUD and PTSD across longitudinal (Possemato et al., 2015; Simpson, Stappenbeck, Luterek, Lehavot, & Kaysen, 2014), laboratory (Coffey et al., 2002; Murphy et al., 2012), and clinical (Back, Brady, Sonne, & Verduin, 2006; Hien et al., 2010) investigations.

Using or abusing specific substances may be associated with differential symptomatology across PTSD symptom clusters. In studies of associations between lifetime use or abuse of a single substance and current PTSD symptoms, increased intrusion symptoms have been identified for cocaine, alcohol, cannabis, and sedatives (Avant et al., 2011; Khoury et al., 2010); increased symptoms of numbing/avoidance have been found for cocaine, alcohol, cannabis, amphetamines, opioids, and sedatives (Avant et al., 2011; Jakupcak et al., 2010; Khoury et al., 2010; Smith et al., 2010; Smith et al., 2016); and increased hyperarousal symptoms have been found for cocaine, alcohol, opioids, cannabis, and amphetamines (Jakupcak et al., 2010; Khoury et al., 2010; McFall et al., 1992; Najavits et al., 2003; Smith et al., 2010; Smith et al., 2016). Comparisons of people who *currently* abuse substances have also yielded evidence that abuse of certain substances is associated with higher severity on some PTSD symptom clusters than abuse of other substances (see Table 1 for a summary of findings), although these findings are mixed. In a treatment-seeking sample of 36 people with current or lifetime PTSD and either cocaine use disorder or AUD, people with an AUD

evidenced more hyperarousal, but not avoidance or re-experiencing, than those with a cocaine use disorder (Saladin et al., 1995). Read and colleagues (2004) assessed 133 people receiving inpatient psychiatric treatment for SUD, and results suggested that AUD was associated with increased re-experiencing symptoms relative to other SUDs, but no other cluster differences were identified for alcohol, opioid, cannabis, cocaine, or sedative use disorders. Similarly, Tull and colleagues (2010) used PTSD symptom cluster scores to predict heroin, crack/cocaine, and alcohol dependence in 48 people completing a 30-day treatment for a SUD. Unlike the previous two studies, this work statistically accounted for the abuse of multiple substances. Results indicated that hyperarousal was positively associated with heroin dependence and avoidance was negatively associated with heroin dependence. Finally, Stewart and colleagues (1999) sampled 295 community women and found that, relative to other SUDs, AUD was associated with arousal symptoms, anxiolytic dependence was associated with arousal and numbing symptoms, and analgesic dependence was associated with arousal, intrusion, and numbing symptoms.

In sum, although research suggests that substances have different associations with PTSD symptom clusters, these results are equivocal, and several limitations to this work warrant additional research on this topic. First, few studies have compared people with different SUDs to each other in terms of their PTSD symptoms. This limits conclusions regarding the extent to which specific substances contribute differentially to symptom patterns beyond the general effect of substance use. Second, most studies have included participants regardless of trauma history or PTSD status, so findings of differential effects could be driven by differences in trauma exposure across substances. Third, multiple comorbidities between SUDs, although common (Stinson et al., 2005), have received little attention. Only one study in this body of literature statistically accounted for the abuse of multiple substances (Tull et al., 2010), and no studies to our knowledge have examined differences in PTSD symptom patterns as a function of multiple SUD comorbidities. Finally, only one study assessed symptom-level differences in PTSD (Saladin et al., 1995), and this study had a relatively small sample. Thus, the current study uses a treatment-seeking sample of people with SUDs who screened positive for PTSD to examine cluster-level and symptom-level differences in PTSD symptom presentation.

Method

Participants

Participants were 208 people seeking treatment at a residential SUD treatment facility who were part of a larger IRB-approved study investigating PTSD treatment effectiveness (Coffey et al., 2016). Of the original sample of 225, participants who reported no lifetime criterion A events ($n = 4$) or reported criterion A events but did not complete the clinical interview assessing PTSD ($n = 2$) were excluded, along with participants who were missing data on their baseline SUD diagnosis ($n = 4$) and/or recent substance use ($n = 6$) and one participant who had no current SUD diagnosis with recent use. In the final sample, participants were 48.08% women and had a mean age of 33.82 ($SD = 10.35$), with a mean annual household income of $33,418 ($SD = $35,822). Most (77.88%) of the sample was

White/Caucasian, with a significant minority reporting that their race was Black/African American (20.67%).

Procedures

Participants were screened for the following inclusion criteria : 18–64 years of age, English literacy, score >8 on the Alcohol Use Disorders Identification Test (AUDIT; Babor et al., 1992), and to identify individuals with likely PTSD, score >44 on the PTSD Checklist-Civilian version (PCL-C; Weathers et al., 1993). Individuals were excluded if they were at imminent risk for suicide, psychotic or manic, in an ongoing abuse relationship related to their criterion A trauma, or judged to have a medical condition that might have compromised participation (e.g., dementia). Further, because the larger clinical trial examined the effects of psychotherapy on PTSD among substance-dependent individuals, using benzodiazapines or medications to reduce substance use was an additional exclusion criterion. After obtaining informed consent, interviewers administered clinical interviews and self-report questionnaires.

Measures

National Women's Study PTSD Module (NWS-PTSD: Kilpatrick, Resnick, Saunders, & Best, 1989; Resnick, 1996)—The NWS is a structured clinical interview that assesses lifetime exposure to a variety of traumas. The current study used a modified version of the NWS-PTSD designed to explore interpersonal violence (Dansky, Bryne, & Brady, 1999). This instrument was used to calculate the number of criterion A events experienced by participants.

Clinician Administered PTSD Scale (CAPS; Blake et al., 1995)—The CAPS was used to assess PTSD symptom severity. This semi-structured interview assesses each of the 17 DSM-IV-TR symptoms of PTSD, with frequency and intensity measured for each symptom over the prior 30 days. Symptoms were considered to be present when they had a frequency of ≥1 and severity of ≥2 (Weathers, Keane, & Davidson, 2001). The CAPS has strong reliability and high concurrent validity (Weathers et al., 2001). Most participants (92.79%) received a PTSD diagnosis via the CAPS, indicating that it had concurrent validity with the PCL-C (which had been used as a screening measure). Consistent with past research (King et al., 1998; Stewart et al., 1999), we coded the items of the CAPS into four clusters reflecting summed frequency and severity items: intrusions (5 items; e.g., intrusive memories; α=0.83), avoidance (2 items; e.g., avoidance of external trauma reminders; α=0.71), numbing (5 items; e.g., feeling detached or estranged from others; α=0.73), and hyperarousal (5 items; e.g., hypervigilance; α=0.73).

Computerized Diagnostic Interview Schedule (C-DIS IV; Robins et al., 2000)—The C-DIS is a structured questionnaire that assesses psychiatric disorders consistent with the DSM-IV (Robins et al., 2000). In this study, the C-DIS was used to assess past-year cannabis use disorder, AUD, amphetamine use disorder, cocaine use disorder, sedative/hypnotic/anxiolytic use disorder, and opioid use disorder.

TimeLine Follow Back (TLFB; Sobell & Sobell, 1992)—The TLFB uses a retrospective self-report calendar method and has well-established reliability and validity for assessing substance use (Sobell & Sobell, 1992). Because we sought to understand associations between current use and PTSD, participants were considered to have a SUD with active use if they met diagnostic criteria on the C-DIS and endorsed use of that substance in the past 3 months on the TLFB.

Analyses

We conducted MANCOVA models to examine differences in PTSD symptom presentation as a function of active substance(s) of abuse. Analyses examined main effects for the presence of each specific SUD with active use on PTSD symptoms, as compared to the absence of that specific SUD; results represent the difference between average symptom scores for people with a specific SUD and people with all other types of SUDs, accounting for the variance associated with the other SUDs and covariates. Due to significant differences in PTSD symptom scores by age, income, and number of criterion A events (Table 2), we controlled for these covariates.

In the first model, to facilitate comparison with past research that has taken this approach, we compared symptom cluster severity in alcohol use disorder and all drug use disorders combined. In the second model, we examined symptom cluster severity specific to each drug use disorder. However, because this sample was selected for high levels of alcohol abuse, nearly all of the sample had an alcohol use disorder. There were also differences in likelihood of an AUD diagnosis across each drug use disorder (Table 3). Thus, to create a more homogenous sample and thereby simplify interpretations, we decided to limit the sample for these analyses to those with an AUD ($N = 192$). In this way, we were able to examine the association between specific DUDs and PTSD symptoms without the confound of differences in prevalence of comorbid AUD across DUDs. We then used the results of the second model to identify symptom clusters that evidenced differences across SUDs. For these clusters, we tested differences in expression of specific symptoms within each cluster in relation to any SUDs that evidenced significant differences in the second model.

Results

See Table 2 for descriptive statistics. All CAPS symptom cluster scores were significantly correlated with each other. Participants had between one and six past-year SUDs with active use ($M = 2.36$, $SD = 1.18$). Table 3 presents frequencies for each SUD and comorbidities with other SUDs. Most of the sample (92.3%) met criteria for AUD and/or drug use disorder (DUD) (76.4%), and 74.5% of those with an AUD also met criteria for a DUD.

Table 4 includes *t*-tests comparing CAPS cluster scores by diagnosis. In the full sample, participants with an AUD scored significantly higher on avoidance than those without an AUD, and participants with a DUD scored significantly higher on numbing and hyperarousal than those without a DUD. We then restricted the sample to only those with an AUD and explored bivariate differences for specific DUDs. Participants with cannabis use disorder scored significantly higher on hyperarousal than those without, participants with cocaine use disorder scored significantly higher on hyperarousal than those without, participants with an

opioid use disorder scored significantly higher on intrusion, numbing, and hyperarousal, and participants with a sedative/hypnotic/anxiolytic use disorder scored significantly higher on numbing.

The first model explored differences in symptom cluster scores by AUD and DUD. Significant omnibus differences were identified for DUD and AUD (Table 5), indicating that there are differences in cluster scores for people with DUD or DUD/AUD as compared to those with only AUD, as well as for those with AUD or AUD/DUD as compared to those with only DUD. Between-subjects effects revealed that differences for DUD were due to significantly higher scores in the hyperarousal cluster for those with a DUD, and differences for AUD were due to significantly higher scores in the avoidance cluster for those with an AUD.

The second model explored differences in symptom cluster scores by type of comorbid DUD to determine the extent to which results in the first model were driven by specific substances. Significant omnibus differences were identified for cocaine use disorder and sedative/hypnotic/anxiolytic use disorder (Table 5), indicating that there are differences in cluster scores for people with each of these disorders as compared to people with other SUDs. Between-subjects effects indicated that differences for cocaine use disorder were driven by significantly higher hyperarousal scores as compared to those without cocaine use disorder and differences in sedative/hypnotic/anxiolytic use disorder were driven by significantly higher numbing scores as compared to those without sedative/hypnotic/anxiolytic use disorder.

The third and fourth models explored specific symptoms (Table 6). In the model predicting hyperarousal symptoms, the omnibus test was significant for cocaine use disorder, and between-subjects effects revealed that people with a cocaine use disorder had significantly higher scores on irritability/angry outbursts and hypervigilance. In the model predicting numbing symptoms, the omnibus test was significant for sedative/hypnotic/anxiolytic use disorders, and between-subjects effects indicated that people with a sedative/hypnotic/anxiolytic use disorder scored significantly higher on inability to remember important parts of the event, decreased interest or participation in activities, restricted range of affect, and foreshortened future.

Discussion

This study examined relationships between PTSD symptom clusters and past-year SUDs with active use in treatment-seeking sample with PTSD and SUD. Results highlight the differential relationship between PTSD symptom clusters and various SUDs.

PTSD avoidance symptoms were significantly elevated in individuals with AUD or AUD +DUD as compared to individuals with DUD only. Similar relationships were not identified in past studies of people with SUDs (Read et al., 2004; Steward et al., 1999; Tull et al., 2010), although these studies did not select participants for likely PTSD, as in the current study. Support for these findings can be found in a recent daily study of PTSD and drinking, in which the association between PTSD symptoms and later drinking was moderated by

avoidance coping, such that greater avoidance coping motives were associated with greater alcohol use in response to increases in PTSD symptoms compared to lower avoidance coping motives (i.e., Possemato et al., 2015). Thus, it is possible that symptoms of avoidance in this sample were self-medicated by alcohol, which could have, in turn, worsened symptoms of avoidance.

Results suggested a strong relationship between cocaine use disorder and the hyperarousal symptom cluster among the subsample with AUD. The specific symptoms of irritability or angry outbursts and hypervigilance were significantly greater in individuals with a cocaine use disorder than those without this disorder. It is possible that heightened symptoms of hyperarousal are symptomatic of acute cocaine intoxication or chronic use. This effect of cocaine might be intentionally sought out among people with already-heightened hypervigilance, as cocaine could function to maintain attention to environmental threats. Inconsistent with these findings, two studies on this topic did not identify an association between PTSD symptom clusters and cocaine use (Avant et al., 2011) or dependence in remission (Tull et al., 2010) among individuals exposed to trauma but unselected for PTSD or AUD. It is possible that the differences identified in the current study emerge when examining individuals selected for likely PTSD, individuals with cocaine use disorder comorbid to AUD, or individuals with a SUD with active use.

Sedative/hypnotic/anxiolytic use disorder appeared to be associated with symptoms of numbing among individuals with AUD, consistent with past research (Stewart et al., 1999). Follow-up analyses indicated that people with sedative/hypnotic/anxiolytic use disorder scored higher on the following symptoms: inability to remember important parts of the event, decreased interest or participation in activities, restricted range of affect, and foreshortened future. Given the cognitive effects of acute and long-term sedative/hypnotic/anxiolytic use, including impairment in memory, processing speed, and problem-solving (Barker, Greenwood, Jackson, & Crowe, 2004; Vermeeren, & Coenen, 2011), it is possible that these symptoms were heightened as a result of substance use. It is also possible that patients exhibiting these symptoms might have been more likely to request or be prescribed benzodyazepines (which are in the anxiolytic class).

In light of the theoretical debate regarding the functional relationship between PTSD and SUDs, it is notable that our findings regarding a relationship between AUD and avoidance symptoms were consistent with a self-medication or mutual maintenance theory, and a logical explanation for two of the relationships we observed—cocaine use disorder with hyperarousal and sedative/hypnotic/anxiolytic use disorder with numbing—is that these PTSD symptoms are increased as a result of the use of this specific substance. It seems somewhat less likely that these latter substances would be sought out to reduce the severity of these symptoms as posited by the self-medication hypothesis, as one would expect that hyperarousal would be worsened by cocaine use and emotional numbing would be worsened by sedative/hypnotic/anxiolytic use. It is also possible that people with either disorder use substances intentionally to increase these symptoms in response to other symptoms. For example, people with cocaine use disorder might be particularly bothered by the presence of strong negative beliefs about the safety of the world (even if these symptoms themselves are not particularly severe), which leads them to seek increases in hypervigilance via cocaine to

protect themselves. People with sedative/hypnotic/anxiolytic use disorder might be more bothered than others with PTSD by strong negative emotions or intrusion symptoms, which could lead them to seek increases in numbing via sedatives/hypnotics/anxiolytics. In either example, self-medication of certain symptoms would be accomplished via the observed increases in other symptoms. It also is possible that these symptoms are heightened via chronic use in response to trauma triggers or other symptoms, consistent with the mutual maintenance model. Although evidence has supported both the self-medication hypothesis and mutual maintenance theories for AUD (Coffey et al., 2007; Kaysen et al., 2011; Read et al., 2013), the functional relationship between PTSD and cocaine and sedative/hypnotic/anxiolytic use disorders has been unexplored, and it is possible that a different functional relationship between PTSD and SUD exists for these substances. This question should be examined in future research, as it could have implications for how comorbid PTSD-SUD is treated for specific substances. For example, if PTSD symptoms are exacerbated by cocaine or sedatives/hypnotics/anxiolytics but the reverse is not true, treatment for these SUDs comorbid to PTSD might be more effective if, particularly in early stages of treatment, it focused primarily on reduction of substance use. Additionally, research that investigates phenomenological and biological similarities and differences between specific PTSD symptoms and the effects of various substances (e.g., hyperarousal and cocaineinduced arousal) is needed; this could help to refine PTSD assessment measures to avoid mischaracterizing the effects of substance use as symptoms of PTSD.

This study addressed the relationship between SUDs with active use (i.e., past year diagnosis with use of that substance in the past 3 months) and PTSD symptom severity in the past month. These overlapping timeframes provide important evidence of their concurrent association. This stands in contrast to past studies that examined relationships between PTSD and substance use (Avant et al., 2011) and dependence in remission (Tull et al., 2010), which identified different relationships between PTSD and SUD. Although daily assessments of both PTSD and SUD would be ideal to explore concurrent relationships, to date, such research has focused primarily on alcohol use (e.g., Simpson et al., 2014). Our findings suggest that daily assessments of relationships between symptom clusters and cocaine and sedative/hypnotic/anxiolytics are warranted.

These findings highlight the importance of addressing specific forms of co-occurring SUDs when investigating the role of SUDs in PTSD symptom patterns. Although our initial analyses suggested that DUD was associated with hyperarousal, it appears that this finding was driven primarily by the relatively large proportion of individuals with cocaine use disorder—who evidenced significantly higher symptoms of hyperarousal—among those with a DUD. It is clear from these findings that drugs of abuse are differentially associated with PTSD symptom clusters, and as such, collapsing them in investigations of their relationship with PTSD symptomatology could produce misleading findings or mask differential relationships.

The results of this study are consistent with a broader literature demonstrating variability in clinical presentation across the different PTSD symptom clusters. In a longitudinal study examining the course of PTSD symptoms in young adult survivors of community violence, Schell and colleagues (2004) demonstrated that respondents for whom hyperarousal was the

most pronounced baseline symptom showed lower overall symptom improvement relative to counterparts for whom hyperarousal was less prominent. Such findings highlight the importance of the hyperarousal symptom cluster specifically, and call for studies that investigate the efficacy of tailoring treatment to address individual symptom clusters (Noorholm & Jovanovic, 2010). An approach to treatment development that takes into account individual differences in PTSD symptom presentation, though representing a departure from current models of PTSD treatment, has the potential to improve outcomes.

Strengths of this study include the use of a relatively large sample, the attention to issues of SUD comorbidity, and the selection of participants with active PTSD symptoms and SUDs. However, this study also had several limitations. First, although we accounted statistically for multiple SUD diagnoses and conducted analyses that examined DUDs that were comorbid to AUD, we did not control for other mental health diagnoses, and it is not possible to compare the many possible permutations of SUD comorbidity in a single observational study. Second, we did not control for severity of SUD, as it would be difficult to represent global SUD severity using a single metric in a population highly heterogeneous for substance of abuse with multiple comorbid substances. It is possible, though, that differences in PTSD symptoms could be driven by differences in severity of SUDs. Third, because participants were assessed soon after seeking treatment, their symptoms may reflect the early stages of withdrawal rather than a period of active use. Fourth, cross-sectional studies, like the current study, may confound symptoms of drug use (e.g., increased irritability and mental alertness from cocaine use) with specific PTSD symptoms (e.g., hyperarousal). Finally, relationships between PTSD symptom clusters and SUDs may be sample specific, which could also explain the inconsistent findings in the literature. For example, factors such as the relative cost and access to a specific drug in a given geographical area may determine use patterns.

In conclusion, this study's results tentatively suggest that the functional relationship between PTSD symptoms and SUDs might differ across SUDs. Future research is needed to elucidate the relationship between PTSD symptoms and both cocaine and sedative/hypnotic/anxiolytic use disorders in particular, as greater understanding of the nature of these functional relationships could inform the development of treatments that improve outcomes for patients with comorbid diagnoses.

Acknowledgments

Role of Funding Sources

Manuscript preparation was supported in part by grants from the National Institute for Alcohol Abuse and Alcoholism (R01AA016816, PI: S. Coffey; T32AA007455-33, PI: M. Larimer).

References

Back SE, Brady KT, Sonne SC, Verduin ML. Symptom improvement in co-occurring PTSD and alcohol dependence. Journal of Nervous and Mental Disease. 2006; 194:690–696. DOI: 10.1097/01.nmd.0000235794.12794.8a [PubMed: 16971821]

Barker MJ, Greenwood KM, Jackson M, Crowe SF. Cognitive effects of long-term benzodiazepine use: A meta-analysis. CNS Drugs. 2004; 18(1):37–48. [PubMed: 14731058]

Begle AM, Hanson RF, Danielson CK, McCart MR, Ruggiero KJ, Amstadter AB, Kilpatrick DG. Longitudinal pathways of victimization, substance use, and delinquency: Findings from the National Survey of Adolescents. Addictive Behaviors. 2011; 36:682–689. DOI: 10.1016/j.addbeh.2010.12.026 [PubMed: 21377805]

Chilcoat HD, Breslau N. Investigations of causal pathways between PTSD and drug use disorders. Addictive Behaviors. 1998; 23:827–840. DOI: 10.1016/S0306-4603(98)00069-0 [PubMed: 9801719]

Coffey SF, Saladin ME, Drobes DJ, Brady KT, Dansky BS, Kilpatrick DG. Trauma and substance cue reactivity in individuals with comorbid posttraumatic stress disorder and cocaine or alcohol dependence. Drug and Alcohol Dependence. 2002; 65:115–127. [PubMed: 11772473]

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

Coffey SF, Schumacher JA, Nosen E, Littlefield AK, Henslee AM, Lappen A, Stasiewicz PR. Trauma-focused exposure therapy for chronic posttraumatic stress disorder in alcohol and drug dependent patients: A randomized clinical trial. Psychology of Addictive Behaviors. 2016; 30(7):778–790. http://doi.org/10.1037/adb0000201. [PubMed: 27786516]

Hien DA, Jiang H, Campbell ANC, Hu MC, Miele GM, Cohen LR, Nunes EV. Do treatment improvements in PTSD severity affect substance use outcomes? A secondary analysis from a randomized clinical trial in NIDS's Clinical Trials Network. American Journal of Psychiatry. 2010; 167:95–101. DOI: 10.1176/appi.ajp.2009.09091261 [PubMed: 19917596]

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

Kaysen D, Atkins DC, Moore SA, Lindgren KP, Dillworth T, Simpson T. Alcohol use, problems, and the course of posttraumatic stress disorder: A prospective study of female crime victims. Journal of Dual Diagnosis. 2011; 7:262–279. DOI: 10.1080/15504263.2011.620449 [PubMed: 23538605]

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

Murphy JG, Yurasek AM, Dennhardt AA, Skidmore JR, McDevitt-Murphy ME, MacKillop J, Martens MP. Symptoms of depression and PTSD are associated with elevated alcohol demand. Drug and Alcohol Dependence. 2013; 127:129–136. DOI: 10.1016/j.drugalcdep.2012.06.022 [PubMed: 22809894]

Read JP, Wardell JD, Colder CR. Reciprocal associations between PTSD symptoms and alcohol involvement in college: A three-year trait-state-error analysis. Journal of Abnormal Psychology. 2013; 122:984–997. DOI: 10.1037/a0034918 [PubMed: 24364601]

Robins, LN., Cottler, LB., Bucholz, KK., Compton, WM., North, CS., Rourke, KM. Diagnostic Interview Schedule for DSM-IV (DIS-IV). St. Louis, MO: Washington University; 2000.

Simpson TL, Stappenbeck CA, Luterek JA, Lehavot K, Kaysen LD. Drinking motives moderate daily relationships between PTSD symptoms and alcohol use. Journal of Abnormal Psychology. 2014; 123:237–247. DOI: 10.1037/a0035193 [PubMed: 24661174]

Stewart SH. Alcohol abuse in individuals exposed to trauma: A critical review. Psychological Bulletin. 1996; 120:83–112. DOI: 10.1037/0033-2909.120.1.83 [PubMed: 8711018]

Stinson FS, Grant BF, Dawson DA, Ruan WJ, Huang B, Saha T. Comorbidity between DSM-IV alcohol and specific drug use disorders in the United States: Results from the National Epidemiologic Survey on Alcohol and Related Conditions. Drug and Alcohol Dependence. 2005; 80(1):105–116. DOI: 10.1016/j.drugalcdep.2005.03.009 [PubMed: 16157233]

Vermeeren, A., Coenen, AML. Effects of the use of hypnotics on cognition. In: Van Dongen, HPA., Kerkhof, GA., editors. Progress in brain research. New York, NY: Elsevier; 2011. p. 89-103.

Weathers FW, Keane TM, Davidson JR. Clinician-Administered PTSD Scale: A review of the first ten years of research. Depression and Anxiety. 2001; 13:132–156. DOI: 10.1002/da.1029 [PubMed: 11387733]

Highlights

- Treatment-seeking patients with substance use disorders and likely PTSD are sampled
- Associations between PTSD symptoms and substance use disorders are tested
- Different symptom patterns are identified across types of substance use disorder

Table 1

Literature Review of Comparisons between Substances of Abuse and Symptom Cluster Scores

Substance	Intrusion/re-experiencing	Numbing/Avoidance	Hyperarousal
Cocaine	= alcohol (Saladin et al., 1995) = other SUDs (Read et al., 2004) = other SUDs (Tull et al., 2010)	= alcohol (Saladin et al., 1995) = other SUDs (Read et al., 2004) = other SUDs (Tull et al., 2010)	< alcohol (Saladin et al., 1995) = other SUDs (Read et al., 2004) = other SUDs (Tull et al., 2010)
Alcohol	> other SUDs (Read et al., 2004) = cocaine (Saladin et al., 1995) = other SUDs (Tull et al., 2010) = other SUDs (Stewart et al., 1999)	= other SUDs (Read et al., 2004) = cocaine (Saladin et al., 1995) = other SUDs (Tull et al., 2010) = other SUDs (Stewart et al., 1999)	= other SUDs (Read et al., 2004) > cocaine (Saladin et al., 1995) = other SUDs (Tull et al., 2010) > other SUDs (Stewart et al., 1999)
Opioids	= other SUDs (Read et al., 2004) = other SUDs (Tull et al., 2010) > other SUDs (Stewart et al., 1999)	= other SUDs (Read et al., 2004) < SUDs (Tull et al., 2010) > other SUDs (Stewart et al., 1999)	= other SUDs (Read et al., 2004) > other SUDs (Tull et al., 2010) > other SUDs (Stewart et al., 1999)
Cannabis	= other SUDs (Read et al., 2004)	= other SUDs (Read et al., 2004)	= other SUDs (Read et al., 2004)
Sedatives	= other SUDs (Read et al., 2004) = other SUDs (Stewart et al., 1999)	= other SUDs (Read et al., 2004) > other SUDs (Stewart et al., 1999)	= other SUDs (Read et al., 2004) > other SUDs (Stewart et al., 1999)

Table 2

Descriptive Statistics

			Correlations					
	Range	***M*(*SD*)**	**1**	**2**	**3**	**4**	**5**	**6**
1. CAPS intrusion	0–40	19.47 (8.54)	0.54***					
2. CAPS avoidance	0–16	9.27 (3.75)		0.30***				
3. CAPS numbing	0–40	20.88 (7.67)	0.32**	0.34***	0.47***			
4. CAPS hyperarousal	0–40	22.86 (7.48)	0.38***	–				
5. Age	18–60	33.82 (10.35)	−0.11	0.19**	0.05	−0.17*	0.03	
6. Income	0–300,000	33,418.18 (35,822.52)	−0.03	0.01	0.10	0.14*		–
7. Number of criterion A events	1–29	9.44 (5.36)	0.39***	0.23**	0.16*	0.30***	0.08	0.01

Note: CAPS = Clinician Administered PTSD Scale

Table 3

Frequency of Substance Use Disorders and Comorbidities

		Percent of those with disorder in column with disorder in row as comorbid SUD						
		Frequency	**Alcohol**	**Cannabis**	**Amphetamine**	**Cocaine**	**Opioid**	**S/H/A**
Alcohol	*n* = 192, 92.3%	–	32.3	15.6	42.2	27.1	22.9	
Cannabis	*n* = 67, 32.2%	92.5	–	17.9	46.3	32.8	29.9	
Amphetamine	*n* = 33, 15.9%	90.9	36.4	–	39.4	45.5	39.4	
Cocaine	*n* = 91, 43.8%	89.0	34.1	14.3	–	26.4	26.4	
Opioid	*n* = 59, 28.4%	88.1	37.3	25.4	40.7	–	59.3	
S/H/A	*n* = 49, 23.6%	89.8	40.8	26.5	49.0	71.4	–	

Note:

* $p < .05$,

** $p < .01$,

*** $p < .001$;

S/H/A = sedative/hypnotic/anxiolytic; SUD = substance use disorders

Addict Behav. Author manuscript; available in PMC 2019 January 01.

Table 4

CAPS severity scores by substance of abuse

		T-tests by cluster			
	Total Score	**Intrusion**	**Avoidance**	**Numbing**	**Hyperarousal**
Type of SUDs (full sample, N = 208)					
Alcohol	$M = 73.06$, $SD = 19.91$	$t(206) = -1.58$	$t(206) = -2.56^{*}$	$t(206) = -0.54$	$t(206) = -0.27$
Drugs	$M = 75.17$, $SD = 19.66$	$t(206) = -1.73$	$t(206) = -1.33$	$t(206) = -2.54^{*}$	$t(206) = -4.30^{***}$
Comorbidities (AUD-only sample, N = 192)					
Cannabis	$M = 76.32$, $SD = 18.82$	$t(190) = -0.99$	$t(190) = 0.03$	$t(190) = -0.89$	$t(190) = -2.19^{*}$
Amphetamine	$M = 75.20$, $SD = 17.31$	$t(190) = -0.30$	$t(190) = -0.33$	$t(190) = 0.02$	$t(190) = -1.23$
Cocaine	$M = 76.57$, $SD = 19.93$	$t(190) = -1.04$	$t(190) = -0.82$	$t(190) = -0.35$	$t(190) = -3.77^{***}$
Opioid	$M = 82.37$, $SD = 18.89$	$t(190) = -2.06^{*}$	$t(190) = -1.88$	$t(190) = -4.55^{***}$	$t(190) = -2.93^{**}$
S/H/A	$M = 80.80$, $SD = 19.89$	$t(190) = -1.05$	$t(190) = -1.49$	$t(88.17) = -5.05^{***}$	$t(62.48) = -1.43$

$^{*}p < .05$,

$^{**}p < .01$,

$^{***}p < .001$;

S/H/A = sedative/hypnotic/anxiolytic; CAPS = Clinician Administered PTSD Scale; SUDS = substance use disorders; AUD = alcohol use disorder

Table 5

PTSD Symptom Cluster Presentation by Substance Type

	Overall Model			Intrusion		Avoidance		Numbing		Hyperarousal	
	Wilks' lambda	***F***	η^2_p	***F***	η^2_p	***F***	η^2_p	***F***	η^2_p	***F***	η^2_p
Comparison of alcohol and drug use disorders (N = 208)											
Intercept	0.56	38.57***	0.44	50.26***	0.20	77.74***	0.28	67.85***	0.25	97.48***	0.33
# criterion A events	0.83	9.78***	0.17	34.53***	0.15	12.05***	0.06	5.18*	0.03	14.72***	0.07
Age	0.93	3.45**	0.07	5.31*	0.03	11.29***	0.05	0.44	0.00	4.88*	0.02
Income	0.96	2.13	0.04	0.04	0.00	0.21	0.00	2.51	0.01	6.90**	0.03
Drugs (*n* = 158)	0.95	2.72*	0.05	0.01	0.00	0.03	0.00	3.05	0.01	9.56**	0.05
Alcohol (*n* = 192)	0.95	2.44*	0.05	3.33	0.02	9.53**	0.05	0.49	0.00	1.47	0.01
Specific drug use disorders comorbid with alcohol use disorder (N = 192)											
Intercept	0.43	59.03***	0.57	78.77***	0.30	127.14***	0.41	90.63***	0.33	155.15***	0.46
# criterion A events	0.84	8.27***	0.16	30.56***	0.14	8.89**	0.05	2.31	0.01	9.34**	0.05
Age	0.90	5.06***	0.10	6.80**	0.04	14.89***	0.08	0.10	0.00	7.40**	0.04
Income	0.93	3.52**	0.07	2.33	0.01	0.10	0.00	5.39*	0.03	5.91*	0.03
Cannabis (*n* = 62)	0.97	1.25	0.03	0.25	0.00	2.61	0.01	0.10	0.00	1.16	0.01
Amphetamine (*n* = 30)	0.98	0.82	0.02	0.25	0.00	0.75	0.00	2.32	0.01	0.00	0.00
Cocaine (*n* = 81)	0.92	4.11**	0.08	0.09	0.00	0.14	0.00	0.01	0.00	13.04***	0.07
Opioid (*n* = 51)	0.96	1.88	0.04	1.17	0.01	0.55	0.00	6.25*	0.03	4.38*	0.02
S/H/A (*n* = 43)	0.94	2.73*	0.06	0.01	0.00	0.18	0.00	7.16**	0.04	0.31	0.00

*$p < .05$,

**$p < .01$,

***$p < .001$;

S/H/A = sedative/hypnotic/anxiolytic

Table 6

F Values from MANCOVAs Testing Symptom Differences Within Clusters by Substance Type

Hyperarousal	**Overall**	**Difficulty falling or staying asleep**	**Irritability or angry outbursts**	**Difficulty concentrating**	**Hypervigilance**	**Exaggerated startle response**
Intercept	36.31***	89.63***	71.55***	53.85***	38.99***	20.13***
Cocaine	4.60**	0.04	10.27**	2.08	15.50***	0.36
Age	2.98*	0.31	9.58**	1.57	5.83*	3.97*
Income	1.77	1.27	2.29	0.63	1.03	6.96**
# criterion A events	3.39**	4.45*	3.54	3.07	7.03**	7.56**
Numbing	**Overall**	**Inability to remember parts of event**	**Decreased interest or participation in activities**	**Feeling detached or estranged from others**	**Restricted range of affect**	**Foreshortened future**
Intercept	22.52***	32.71***	19.51***	50.17***	57.36***	18.10***
S/H/A	4.86***	9.25**	5.75*	2.16	4.01*	12.49***
Age	2.48*	7.16**	3.23	0.24	0.09	0.31
Income	1.08	0.01	0.28	3.24	4.08*	1.03
# criterion A events	2.09	2.65	7.60**	3.28	0.88	0.00

* $p < .05$,

** $p < .01$,

*** $p < .001$;

S/H/A = sedative/hypnotic/anxiolytic