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Alcohol and Marijuana Polysubstance Use: Comparison of PTSD Symptom Endorsement and Severity Patterns

Nathan T. Kearns¹, Renee M. Cloutier¹, Caitlyn Carey¹, Ateka A. Contractor¹, Heidemarie Blumenthal¹

¹Department of Psychology, University of North Texas, Denton, TX, USA

Abstract

A growing body of work links posttraumatic stress disorder (PTSD) symptoms and substance use. Unfortunately, much of the literature has examined associations in isolation (e.g., alcohol only). Failure to account for simultaneous or concurrent substance use may limit conclusions that can be drawn from existing research, including the extent to which specific substances contribute differentially to PTSD symptom patterns. The current study examined differences in PTSD symptom profiles between individuals using one or both of the most commonly co-administered psychoactive substances – alcohol and marijuana. Trauma-exposed participants ($N = 533$; $M_{age} = 21.15$) comprised two mutually-exclusive groups: past-month alcohol-only use ($n = 334$) or past-month alcohol and marijuana use ($n = 199$). Cluster-level and symptom-level profile analyses evaluated mean differences and shape (parallelism) of PTSD symptom severity profiles between the groups. Follow-up analyses examined symptom-specific difference in PTSD symptom endorsement and severity. Overall, individuals using marijuana and alcohol evidenced greater PTSD negative cognition (30.8% greater) and hyperarousal (26.4% greater) symptom severity. Alcohol and marijuana users were more likely to endorse, and report greater severity of, mood-related PTSD negative cognition symptoms (e.g., anhedonia, negative affect) and externalizing hyperarousal symptom (e.g., irritability/aggression, risky behaviors) than alcohol-only users. Findings highlight important PTSD differences between individuals that are often lumped into homogenous categories of isolated substance users. Findings provide preliminary support for an ‘additive’ self-medication model between PTSD and polysubstance use. Lastly, findings indicate that mood-related negative cognition symptoms and externalizing hyperarousal symptoms may be important targets for PTSD-polysubstance use intervention.

Keywords

substance use; alcohol; marijuana; PTSD; trauma

An extensive and growing body of work has identified strong associations between posttraumatic stress disorder (PTSD) symptoms and problematic substance use (Debell et

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Corresponding Author: Nathan Kearns, Department of Psychology, University of North Texas, 1155 Union Circle, Denton, TX, 76203-5017, NathanKearns@my.unt.edu.

al., 2014; Jacobsen, Southwick, & Kosten, 2001). Generally, research examining these relationships has supported a self-medication or mutual maintenance model of co-occurrence (Ouimette & Brown, 2003; Stewart & Conrod, 2008), whereby individuals use substances as a maladaptive means of coping with psychological or physiological distress associated with PTSD symptoms. Although much of the extant work has focused on the link between PTSD and subsequent alcohol use, similar associations have been demonstrated with other substances, including both stimulants (e.g., Najavits et al., 2003; Smith et al., 2010) and other anxiolytic intoxicants (e.g., marijuana; Wilkinson, Stefanovics, & Rosenheck, 2015; Betthausen, Pilz, & Vollmer, 2015).

Unfortunately, much of the existing PTSD-substance use research has purposefully examined these associations in isolation (e.g., alcohol only) – in most cases not assessing for simultaneous or concurrent use of other substances (e.g., alcohol and marijuana). This methodological approach is problematic given the high prevalence of polysubstance use, particularly among individuals exposed to trauma and/or that are evidencing substance use-related harms. For example, results from the National Alcohol Survey indicate that, among all current alcohol users, approximately 11.4% also report simultaneous or concurrent use of marijuana in the past year (Subbaraman & Kerr, 2015). However, this prevalence rate triples to approximately 34.1–36.2% among individuals evidencing alcohol use-related negative consequences in the past year (e.g., deteriorating social relationships; Subbaraman & Kerr, 2015); such problematic behavioral outcomes have also been linked to increased PTSD symptom severity (e.g., Debell et al., 2014). According to the Global Drug Survey ($n = 14,869$ adults from US, Australia, and UK), involvement in or exposure to violence also substantively increased risk of polysubstance use – in fact, it is among the strongest predictors of past-year use of six or more substances (Morley et al., 2015). Other work among trauma-exposed veterans indicates that 26.7% of individuals with one substance use disorder also met diagnostic criteria for at least one other substance use disorder – with alcohol and cannabis use disorder being the most common co-occurrence (Bhalla, Stefanovics, & Rosenheck, 2017). Given high prevalence of polysubstance use in both general and trauma-associated subpopulations, failure to account for other forms of simultaneous or concurrent substance use limits conclusions that can be drawn from the existing PTSD-substance use literature. In particular, our understanding of the extent to which specific substances – and combinations of substances – contribute or associate differentially to PTSD symptom patterns (e.g., endorsement, severity) may be limited, hindering both our theoretical modeling of PTSD-substance use interplay, as well as our ability to meaningfully integrate these findings into efficacious prevention and remedial efforts.

Some work has attempted to ameliorate this concern by evaluating associations between PTSD and various combinations of substances. Although limited, this empirical work indicates that individuals who use other substances, in addition to alcohol, evidence greater PTSD severity and other forms of psychological distress in comparison to both alcohol-only and “other substance”-only users (Mills, Teesson, Ross, & Peters, 2006; Ruglass, Shevorykin, Brezing, Hu, & Hien, 2017; Salgado, Quinlan, & Zlotnick, 2007; Ullman, Townsend, Starzynski, & Long, 2006). For example, Ullman and colleagues (2006) examined sexual assault survivors with PTSD-only, PTSD and illicit drug use (e.g., any

cocaine, heroin, or psychedelics), PTSD and problematic drinking, and PTSD and polysubstance use (e.g., both drinking and any cocaine, heroin, or psychedelics). Results of their study indicated that women with PTSD that were polysubstance users evidence more extensive trauma histories and greater PTSD symptom severity than sexual assault survivors in the other groups (Ullman et al., 2006). However, generally, this research has either broadly examined PTSD (e.g., dichotomous PTSD diagnosis or overall PTSD severity; Ruglass et al., 2017; Salgado et al., 2007), categorized “other substances” with varying neurological effect (e.g., cocaine, heroin) into a generic polysubstance use variable (Ullman et al., 2006), and/or did not account for other forms of concurrent or simultaneous substance use in their modeling (e.g., excluding participants currently using other substances). While general associations between (a) PTSD and (b) single-substance use and/or unspecified polysubstance use may be useful for laying an empirical foundation to build upon in future work, research examining specific PTSD symptoms (i.e., at the cluster- and individual-level) and differences between singular substance users and specified polysubstance users is a key “first step” to understanding the complexities of PTSD-(poly)substance interplay needed to inform effective prevention and intervention efforts.

Current Study

The current study purports to extend the literature by examining differences in PTSD symptom profiles (i.e., intrusion, avoidance, negative cognitions, hyperarousal) between individuals using one or both of the most commonly used and co-administered psychoactive substances in the United States – alcohol and marijuana (Grant et al, 2015; Hasin et al., 2015). More specifically, the current study will examine PTSD symptom profiles between individuals that are currently only using alcohol and individuals that are currently only using alcohol and marijuana (i.e., excluding individuals that are currently abstaining or currently using other substances). Given extant work examining PTSD-alcohol use (Jacobson et al., 2001; Lehavot, Stappenbeck, Luterek, Kaysen, & Simpson, 2014; Walton et al., 2018) and PTSD-marijuana use in isolation (Turna, Patterson, & Ameringen, 2017; Roitman, Mechoulam, Cooper-Kazaz, & Shalev, 2014) – both denoting strong associations between both PTSD negative cognitions and hyperarousal symptom and substance use – it was hypothesized that individuals who reported using both alcohol and marijuana in the past month would be more likely to (1) endorse PTSD negative cognition and hyperarousal symptoms at a clinical level and (2) report greater severity of PTSD negative cognition and hyperarousal symptoms than individuals who only reported using alcohol in the past month. Conversely, given mixed – largely null – results in the literature (see Debell et al., 2014; Yarnell, 2015 for overviews), it was hypothesized that there would be no differences between the alcohol only and alcohol and marijuana groups on PTSD intrusion and avoidance symptoms.

METHOD

Participants and Procedure

The final sample comprised 533 undergraduate students ($M_{age} = 21.15$, $SD = 3.17$, range = 18–58; 77.90% female) taken from a larger study ($N = 1,658$) on psychological well-being

and substance use. Data were collected from November 2016 through February 2018 at a large university in the state of Texas, in which marijuana use is illegal for medical and recreational purposes (with the exception of low-tetrahydrocannabinol [THC] cannabidiol for patients with intractable epilepsy). Participants were included in the final sample if they met the following eligibility criteria: (1) above the age of 18, (2) experienced at least one Diagnostic and Statistical Manual of Mental Disorders – 5th Edition (DSM-5) defined PTSD Criterion A traumatic event (APA, 2013) as measured by the Life Events Checklist for DSM-5 (LEC-5; Weathers et al., 2013a); (3) endorsed experiencing at least one PTSD symptom at any severity in the past month (i.e., not asymptomatic); (4) had consumed alcohol at least once in the past month; (5) had not consumed any other illicit substance(s) aside from marijuana in the past month; and (6) were able to provide informed consent for participation in the study. Eligible participants completed a general assessment battery of psychological well-being and substance use through Qualtrics – an online data management software that complies with HIPAA regulations. All procedures were approved by the institutional review board at [omitted for blind review].

Participants that met eligibility criteria were divided into two groups based on whether they reported (1) only using alcohol in the past month ($n = 334$) or (2) using both alcohol and marijuana in the past month ($n = 199$). Comparative analyses indicated that the two groups did not differ in terms of biological sex or race/ethnicity. However, the alcohol and marijuana group ($M_{age} = 20.46$) was statistically significantly younger than the alcohol-only group ($M_{age} = 21.56$). See Table 1 for full descriptives of the overall sample and substance use groups, as well as detailed results of the comparative analyses.

Measures

Trauma Exposure—The Life Events Checklist for DSM-5 (LEC-5; Weathers et al., 2013a) was administered to establish the presence of a DSM-5 Criteria A traumatic event (APA, 2013). The LEC-5 demonstrates adequate to good psychometric properties in comparison to other established, self-report measures of trauma history (Gray, Litz, Hsu, & Lombardo, 2004). The LEC-5 consists of 16 specified potentially traumatic events (e.g., *life-threatening illness or injury, transportation accidents*), as well as one unspecified traumatic event. Response options include “*Happened to me,*” “*Witnessed it,*” “*Learned about it,*” “*Part of my job,*” “*Not sure,*” and “*Doesn’t apply.*” Only participants who endorsed “*Happened to me*” for at least one of the 16 specified traumatic events were included in the current study. This conservative approach to defining traumatic event exposure has been used extensively in other trauma research (e.g., Paulus et al., 2016; Thornley et al., 2016).

PTSD Symptom Endorsement and Severity—The PTSD Checklist for DSM-5 (PCL-5; Weathers et al., 2013b) is a 20-item self-report measure that was used to assess PTSD symptom endorsement and severity. Participants are asked to recall their most stressful event from the LEC-5 and indicated how much they were bothered by specific symptoms in the *past month*. Items 1–5 on the PCL-5 correspond with the intrusion symptom cluster (e.g., “*Repeated, disturbing dreams of the stressful experience?*”); items 6–7 with the avoidance symptom cluster (e.g., “*Avoiding memories, thoughts, or feelings related to the stressful experience?*”); items 8–14 with the negative cognition symptom

cluster (e.g., “*Loss of interest in activities that you used to enjoy?*”); and items 15–20 with the hyperarousal symptom cluster (e.g., “*Feeling jumpy or easily startled?*”). Responses range from 0 (“Not at all”) to 4 (“Extremely”). The PCL-5 is a psychometrically-sound measure (Blevins et al., 2015) and evidenced good reliability on each of the PTSD symptom cluster subscales (Cronbach’s $\alpha_s = .89$ to $.92$).

PTSD subscale scores were calculated by summing responses for each symptom cluster, with higher scores indicating more severe PTSD symptoms. PTSD symptom endorsement was defined as a participant reporting 2 (“Moderately”) or greater severity on a given symptom (Weathers et al., 2013b). In accordance with the DSM-5, to meet criteria for a particular symptom cluster, participants needed to endorse one symptom from the intrusion cluster (Criteria B), one symptom from the avoidance cluster (Criterion C), two symptoms from the negative cognition cluster (Criterion D), and two symptoms from the hyperarousal cluster (Criterion E), respectively (APA., 2013).

Substance use—Past-month alcohol and marijuana use were assessed via single-item, face-valid questions. More specifically, for alcohol use, participants were asked, “*In the past month, how many times have you used alcohol?*”; for marijuana use, participants were asked, “*In the past month, how many times have you used cannabis/marijuana?*” Responses on both questions ranged from “0 occasions” to “20 or more occasions”. Participants reporting at least “1–2 occasions” of consuming alcohol met eligibility criteria for the overall sample. Among eligible participants, those reporting at least “1–2 occasions” for marijuana use were included in the alcohol and marijuana group.

Data analytic plan

Independent-samples t-tests and chi-squared test, as appropriate, were used to evaluate differences between the alcohol-only group and alcohol and marijuana group on sociodemographic characteristics (i.e., age, biological sex, race/ethnicity). Significant sociodemographic variables ($p < .05$ level) were included in the subsequent analyses as covariates. Given the influence of time since trauma on posttraumatic symptom severity (Jovanovic et al., 2013; Weems & Carrion, 2007), analyses also controlled for time since trauma.

For the primary analyses, profile analysis (see Kelly et al., 2009; Tabachnick & Fidell, 2007) was used to evaluate level effects (i.e., mean differences) and parallelism (i.e., the extent to which groups evidence the same pattern or “shape” of symptoms) between the groups on the four PTSD symptoms clusters. More specifically, the two groups were compared on the elevation and shape of their profile on the four PTSD symptom cluster severity scores (intrusion, avoidance, negative cognitions, hyperarousal) with a 2 (Group) \times 4 (Cluster) multivariate analysis of variance (MANOVA). Symptom-level profile analyses then compared groups at the symptom level within each of the four PTSD symptom clusters using 2 (Group) \times 5 (Intrusion); 2 (Group) \times 2 (Avoidance); 2 (Group) \times 7 (Negative Cognition); and 2 (Group) \times 6 (Hyperarousal) MANOVAs.

For secondary analyses, individual symptom level differences were examined using (1) univariate analyses of variance (ANOVA) for symptom severity and (2) binary logistic

regression for odds of PTSD symptom endorsement between the alcohol-only and alcohol and marijuana groups. For all analyses, p -values were interpreted for significance; eta-squared coefficients (η^2) and adjusted odd ratios (OR) were interpreted for effect size. Missing data were minimal (i.e. < 2% on any given PCL-5 item) and were missing completely at random (MCAR; $\chi^2 = 416.74, p = .704$), therefore missing data were accounted for via listwise deletion in the analyses.

RESULTS

Cluster Level Analyses

Results of the cluster level profile analysis indicated a statistically significant main effect for group ($F[1,527] = 6.29, p = .012$) and a statistically significant departure from parallelism ($F[3, 1581] = 12.84, p < .001$). Examination of group means indicated that individuals in the alcohol and marijuana group ($M = 24.91$) reported greater overall PTSD symptom severity than individuals in the alcohol-only group ($M = 21.08$). However, the test of parallelism indicated that the PTSD symptom cluster profile for the alcohol and marijuana group was not coincident with the symptom cluster profile of the alcohol-only group, necessitating further evaluation at the symptom-level.

Symptom Level Analyses

Symptom level profile analysis for the PTSD intrusion symptoms indicated no main effect of substance use group ($F[1,527] = 0.18, p = .672$) and that parallelism between the groups was tenable ($F[4,2108] = 0.48, p = .749$). Similarly, the symptom level profile analysis for PTSD avoidance indicated no main effect ($F[1,527] = 0.80, p = .372$) or departure from parallelism ($F[1,527] = 0.13, p = .715$). Generally, these findings suggest that the PTSD intrusion and avoidance symptom profiles for the alcohol-only group and the alcohol and marijuana group were similarly shaped and did not differ, in terms of symptom severity.

Alternatively, symptom level profile analysis for PTSD negative cognitions symptoms indicated a significant main effect ($F[1,527] = 11.49, p = .001$) between groups and that parallelism was tenable ($F[6,3162] = 1.73, p = .109$). Examination of group means showed that the alcohol and marijuana group ($M = 8.87$) reported greater PTSD negative cognitions severity than the alcohol-only group ($M = 6.78$). Follow-up univariate analyses showed users of both alcohol and marijuana reported significantly greater severity of traumatic event amnesia (N1 in Table 2), negative beliefs about oneself or the world (N2), negative trauma-related emotions (e.g., guilt; N4), post-trauma anhedonia (N5), feeling isolated (N6), and negative affect (N7) symptoms than alcohol-only users (all $p < .05, \eta^2: .01-.03$). Follow-up logistic regression analyses indicated that users of both alcohol and marijuana were at significantly greater odds of endorsing negative beliefs about oneself or the world, anhedonia, feeling isolated, and negative affect symptoms than alcohol-only users (all $p < .05, OR: 1.63-2.00$).

Symptom level profile analysis for PTSD hyperarousal symptoms also indicated a significant main effect ($F[1,527] = 9.21, p = .003$) and parallelism ($F[5,2635] = 1.18, p = .315$) between groups. Examination of group means showed that the alcohol and marijuana

group ($M = 6.80$) reported more PTSD hyperarousal symptom severity than the alcohol-only group ($M = 5.38$). Follow-up univariate analyses showed users of both alcohol and marijuana reported significantly greater severity of irritability/aggression (H1 in Table 2), risky or destructive behaviors (H2), difficulty concentrating (H5), and difficulty sleeping (H6) symptoms than alcohol-only users (all $p < .05$, $\eta^2: .01-.02$). Follow-up logistic regression analyses indicated that users of both alcohol and marijuana were at significant greater odds of endorsing irritability/aggression, risky or destructive behaviors, and sleep difficulties symptoms than alcohol-only users (all $p < .05$, $OR: 1.47-2.02$).

Full cluster-level and symptom-level descriptives for PTSD symptom endorsement and PTSD symptom severity, as well as detailed results for the comparative analyses between the two substance use groups, can be seen in Table 2. Symptom-level PTSD severity profiles for the two substance use groups can be seen in Figure 1. Differences in PTSD symptom endorsement and severity between the two substance use groups can be seen in Figure 2.

DISCUSSION

Despite an extensive body of work linking PTSD and substance use (Debell et al., 2014; Jacobson et al., 2001), the focus in the literature on (a) singular, isolated substance use – while not accounting for simultaneous or concurrent use of other substances – and/or (b) *unspecified* polysubstance use may not provide the specificity of information necessary to meaningfully advance our understanding of PTSD-(poly)substance use etiology and interplay. This concern may be particularly important given the high prevalence of polysubstance use among individuals exposed to trauma and those evidencing substance use-related problematic behaviors that are linked with PTSD (Morley et al., 2015; Subbaraman & Kerr, 2015). The current study is the first, to the author's knowledge, to examine differences in PTSD symptom profiles (at both the cluster- and symptom-level) between current alcohol-only and current alcohol and marijuana users, while controlling for other forms of current illicit substance use.

Consistent with the first hypothesis, individuals currently using marijuana, in addition to alcohol, were at significantly greater odds of both endorsing PTSD negative cognition and hyperarousal symptoms and reported greater severity of PTSD negative cognition and hyperarousal symptoms. In fact, as can be seen in Table 2 and Figure 2, approximately 15.0% more participants in the alcohol and marijuana group met provisional diagnostic criteria for PTSD Criterion D (negative cognitions; two or more symptoms in the cluster) and 7.9% more participants in the alcohol and marijuana group met provisional diagnostic criteria for PTSD Criterion E (hyperarousal; two or more symptoms); further, participants in the alcohol and marijuana group evidenced 30.8% greater overall negative cognition symptom severity and 26.4% greater overall hyperarousal symptom severity compared to participants in the alcohol-only group. These results are generally consistent with extant work from separate literatures indicating strong associations between PTSD negative cognition and hyperarousal symptom and both alcohol (e.g., Jacobson et al., 2001; Lehavot et al., 2014; Walton et al., 2018) and marijuana use (e.g., Turna et al., 2017; Roitman et al., 2014). Consistent with the second hypothesis, results also indicated that use of marijuana, in addition to alcohol, did not increase odds of PTSD intrusion or avoidance symptom

endorsement, nor were there significant differences between the substance use groups regarding PTSD intrusion or avoidance symptom severity. Although some work has found these links within contextually-specific trauma subpopulations (e.g., Vietnam veterans, plane crash survivors; McFall et al., 1992; Stewart, Mitchell, Wright, & Loba, 2004), these findings support a broader literature that has not consistently demonstrated associations between PTSD intrusion and avoidance symptoms and substance use outcomes in non-clinical, trauma-exposed populations (see Debell et al., 2014; Yarnell, 2015 for overviews).

Although individual symptoms are broadly categorized into four clusters in the DSM-5, many of the symptoms within the negative cognition and hyperarousal symptoms clusters are notably distinct – as such, evaluation of at the symptom-level is needed to better understand which *specific* symptom(s) are most strongly influencing observed differences between the substance use groups. Results from the current study at the symptom-level suggest that mood-related negative cognition symptoms (e.g., loss of interest, negative affect) and externalizing hyperarousal symptoms (e.g., aggression, reckless or destructive behaviors) are the primary catalysts driving differences in PTSD symptom severity between the alcohol-only and alcohol and marijuana users. Although no work has directly evaluated these specific PTSD-related symptoms and polysubstance use, these findings are generally consistent with broader research findings indicating positive associations between polysubstance use and both externalizing behavior (e.g., aggression and impulsivity; Martinotti et al., 2009) and internalizing depressive symptoms (Maslowsky, Schulenberg, O'Malley, & Kloska, 2014; Trudeau, Spoth, Randall, & Azevedo, 2007).

Although replication and extension are needed, findings from the current study have at least three important implications for PTSD-substance use modeling, prevention, and intervention. First and foremost, these results highlight important differences in PTSD symptom endorsement and PTSD symptom severity between individuals that are often lumped or grouped together into homogenous categories of isolated substance users (e.g., “past-month alcohol use” participants). This problem may be exacerbated in research attempting to isolate non-alcohol, singular substance use populations (e.g., marijuana users, cocaine users). For example, only 3.4% of trauma-exposed participants in our larger sample ($n = 1,029$) reported *only* using marijuana in the past month (i.e., no alcohol or other substance use), meaning that any evaluation of “past-month marijuana use” participants would actually be overwhelmingly comprised of alcohol-marijuana polysubstance users. Given this confounding limitation, future work targeting PTSD-substance use comorbidity should, at a minimum, evaluate, acknowledge, and meaningfully control for other forms of current substance use.

Second, regarding theoretical implications, these collective findings provide preliminary support for an ‘additive’ self-medication model between PTSD and polysubstance use. More specifically, individuals may use multiple substances (i.e., alcohol and marijuana) with similar neurological effects (e.g., anxiolytics) to cope with more severe, specific types of PTSD symptoms. For example, an individual may use alcohol as a means of coping (e.g., dampening) with worsened irritability following their trauma; however, if that irritability is more severe and persists – despite minimal, temporary relief from alcohol use – they may incorporate simultaneous or concurrent use of another anxiolytic substance (e.g., marijuana)

in a maladaptive attempt to fully ameliorate those feelings of irritability. As such, specific forms of polysubstance use among individuals exposed to trauma may serve as risk markers indicating greater severity of specific PTSD symptoms – an important, empirically-supported consideration that may inform future prevention-oriented research and implementation efforts.

Last, findings from the current study may inform existing and developing interventions for individuals evidencing comorbid PTSD-substance use– important given that polysubstance use has been problematic for both substance use (Dutra, et al., 2008) and comorbid PTSD-substance use treatment outcomes (Donovan, Padin-Rivera, & Kowaliw, 2001; McFarlane, 2001). For example, symptom-level results from the current study indicate that mood-related negative cognition symptoms and externalizing hyperarousal symptoms may serve as important intervention targets for individuals reporting PTSD symptoms and alcohol/marijuana polysubstance use. Although more research is needed to understand associations between PTSD symptomatology and other specified combinations of polysubstance use, these findings may also reinforce the importance of utilizing specialized, integrated treatments for comorbid PTSD-substance use – interventions that target transdiagnostic mechanisms underlying multiple types of (poly)substance use (e.g., developing adaptive coping skills in *Seeking Safety*; Najavits, 2002; Najavits, 2004), as opposed to concurrent or sequential treatments that target problematic use of a specific substance (e.g., disulfiram; Alcoholics Anonymous; Humphreys, Blodgett, & Wagner, 2014.; Jørgensen, Pedersen, & Tønnesen, 2011).

Limitations and Future Directions

Results should be interpreted while considering several limitations. First, the cross-sectional nature of the data did not allow for evaluation of the directionality of the findings. Indeed, our results could alternatively suggest that use of multiple substances with similar neurological effects exacerbate the severity of specific PTSD symptoms – more strongly implicating a mutual maintenance model of comorbid interplay. Short-term, prospective (e.g., daily diary) and longitudinal designs are needed to evaluate the temporal sequencing of PTSD and alcohol-marijuana polysubstance use. Second, findings in the current study may be limited by response bias (e.g., recall bias) inherent in self-report of trauma exposure, PTSD symptom severity, and substance use. Future work should consider use of established timeline follow-back procedures (TLFB; Robinson, Sobell, Sobell, & Leo, 2014; Sobell & Sobell, 1992) for more accurate evaluation of (poly)substance use history, structured clinical interviews for evaluation of trauma exposure and PTSD symptoms (e.g., CAPS; Weathers et al., 2013c), and/or incorporating ecological momentary assessment (e.g., daily diary) designs.

Third, the sample in the current study was comprised entirely of undergraduate students. Although this subpopulation may be ideal for preliminary evaluation of PTSD and substance use – given elevated rates of past-year trauma exposure (Read, Ouimette, White, Colder, & Farrow, 2011), prevalence of current PTSD (Smyth, Hockemeyer, Heron, Wonderlich, & Pennebaker, 2008), and lifetime peaks for problematic substance use (Johnston, O'Malley, Bachman, & Schulenberg, 2011) – replication and extension are needed to evaluate if the

current findings generalize to other developmentally- and contextually-distinct populations. Further, although the current sample was relatively diverse, in terms of race/ethnicity, the majority of participants were female. Given established gender and biological sex differences regarding PTSD (e.g., symptom severity, diagnosis rates; Olf, Langeland, Draijer, & Gersons, 2007), future work should also consider a stratified sampling approach to ensure relatively equal representation. Last, the current study did not differentiate between concurrent and simultaneous alcohol-marijuana polysubstance use. Given notable differences between these distinct forms of polysubstance use regarding alcohol-related problematic outcomes (e.g., drunk driving; Subbaraman & Kerr, 2015), incorporating more nuanced assessment of types of polysubstance use into future research is warranted. Further, future work should consider evaluating potential differentiations in PTSD symptom profiles between other specified singular and polysubstance use groups (e.g., alcohol-only vs. alcohol and cocaine).

Despite these limitations, the current study meaningfully extends the PTSD-(poly)substance use literature as the first study, to the authors knowledge, to examine differences in PTSD symptom profiles (i.e., severity and endorsement) at the *individual* symptom level between two *specified* substance use groups – current alcohol-only and current alcohol and marijuana users – while comprehensively controlling for other forms of current illicit substance use. Although replication and extension with intensive prospective and/or longitudinal designs (e.g., daily diary) are needed in other contextually- and developmentally-distinct populations, these findings (a) highlight the importance of evaluating and meaningfully controlling for other simultaneous and concurrent forms of substance use; (b) provide preliminary support for an ‘additive’ self-mediation model of PTSD-(poly)substance use co-occurrence; and (c) posit that that mood-related negative cognition symptoms and externalizing hyperarousal symptoms may serve as important intervention targets for individuals reporting PTSD symptoms and alcohol/marijuana polysubstance use.

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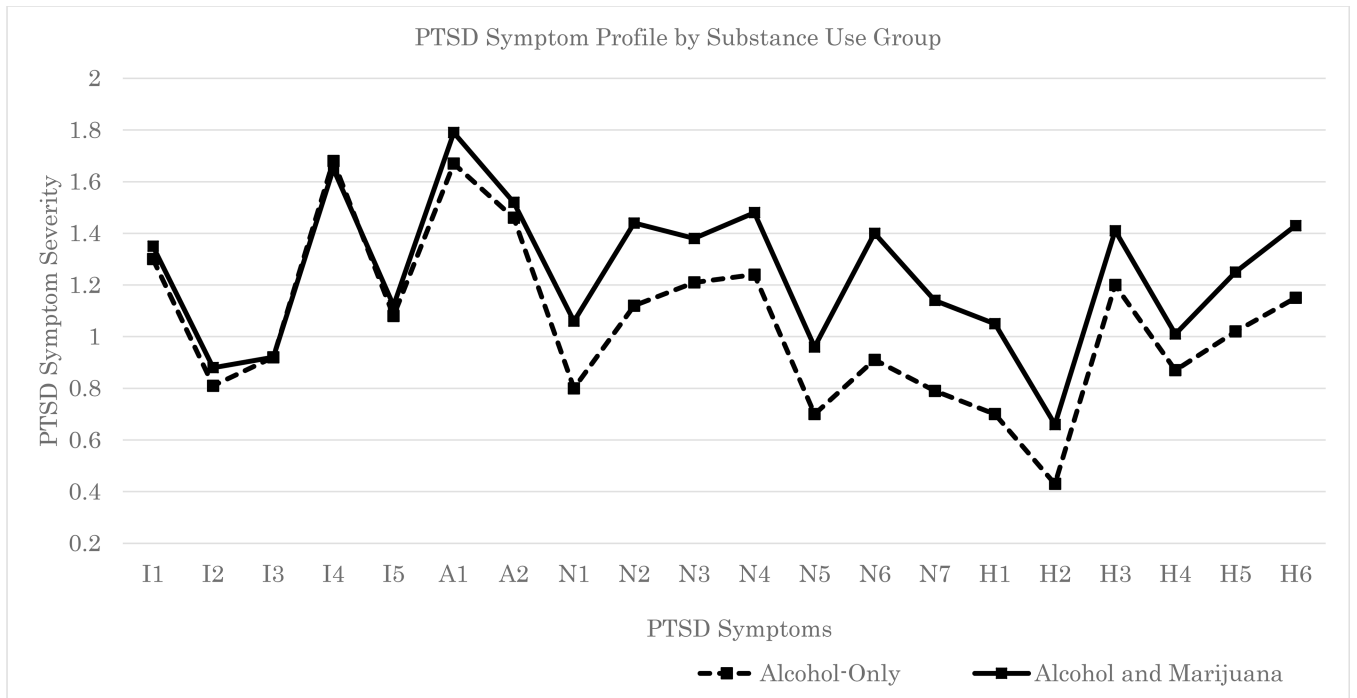


Figure 1. PTSD Symptom Profiles on the PTSD Checklist for DSM-5 (PLC-5; Weather et al., 2013b) by Substance Use Group
 Note. I: Intrusion symptoms; A: Avoidance symptoms; N: Negative cognition symptoms; H: Hyperarousal symptoms.

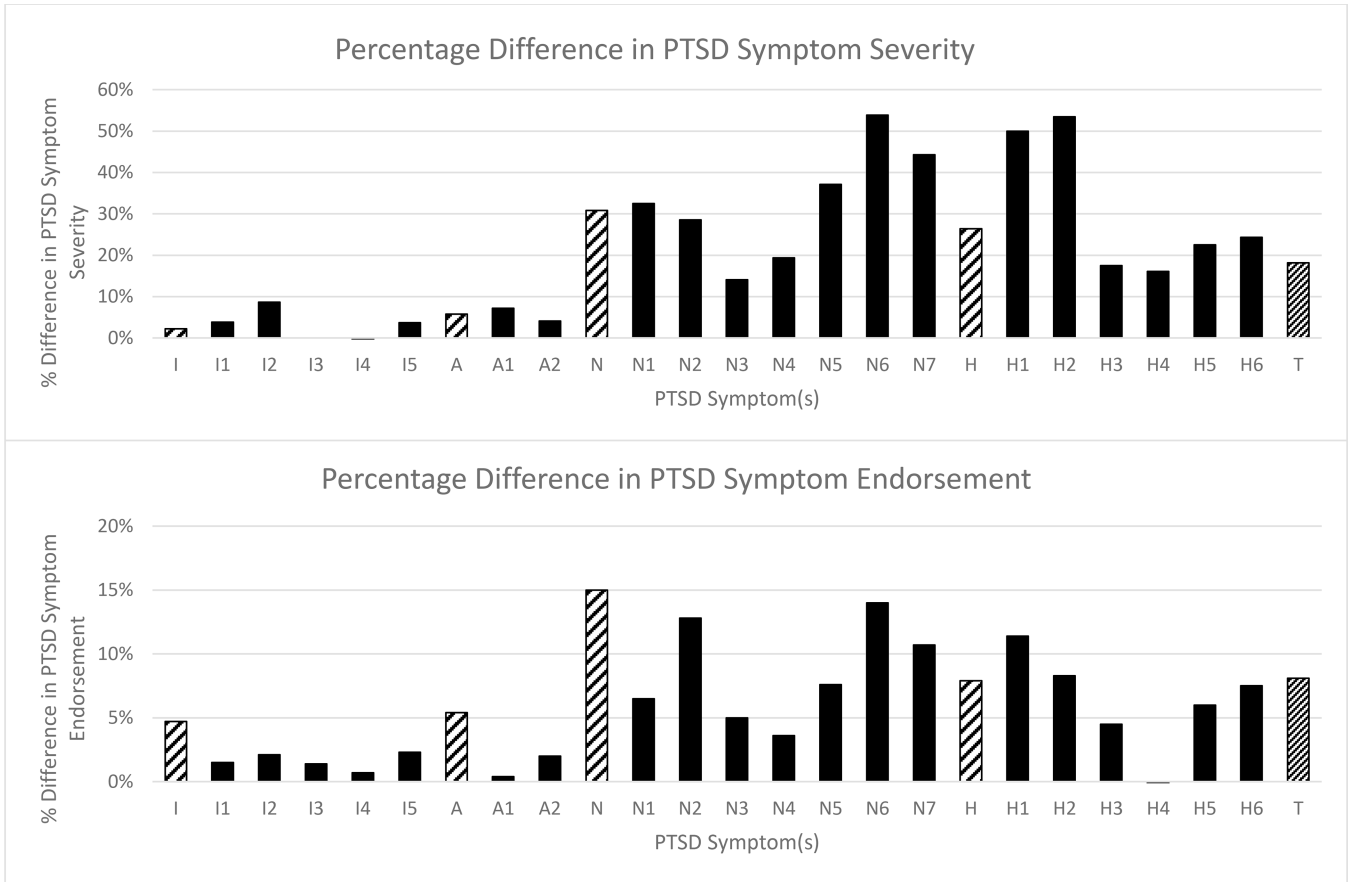


Figure 2. Percentage Difference in PTSD Symptom Severity and PTSD Symptom Endorsement
Note. I: Intrusion symptoms; A: Avoidance symptoms; N: Negative cognition symptoms; H: Hyperarousal symptoms; T: Total PTSD symptoms. All differences represent increases in PTSD symptom severity and endorsement in the alcohol and marijuana group over alcohol-only group. For clarity, negative values were excluded; the alcohol-only group reported 1.8% greater symptom severity on distress caused by reminders of the traumatic event (R4) and 0.5% more of the alcohol-only group endorsed startle response symptoms (H4) than the alcohol and marijuana group.

Table 1. Descriptives for Overall Sample and Descriptive Comparison between Alcohol-Only and Alcohol/Marijuana Groups

Variables	Overall Sample (n = 533)	Alcohol Only (n = 334)	Alcohol and Marijuana (n = 199)	Test Statistics	
				χ^2	t
Age	21.15 ± 3.17	21.56 ± 3.42	20.46 ± 2.56		3.91**
Biological Sex				0.05	
Female	415 (77.9%)	259 (77.5%)	156 (78.4%)		
Male	118 (22.1%)	75 (22.5%)	43 (21.6%)		
Race ^a				10.14	
Asian	34 (6.4%)	29 (8.7%)	5 (2.5%)		
African-American	73 (13.7%)	44 (13.2%)	29 (14.6%)		
White/Caucasian	246 (46.2%)	156 (46.7%)	90 (45.2%)		
Latino/Hispanic	96 (18.0%)	53 (15.9%)	43 (21.6%)		
Multiracial	70 (13.1%)	42 (12.6%)	28 (14.1%)		
Other	14 (2.7%)	10 (3.0%)	4 (2.0%)		

Note.

^a Given small sample size, "Other" was excluded from comparative analysis

** $p < .01$

Table 2. Symptom-Level Univariate Analyses for PTSD Symptoms by Alcohol Only vs. Alcohol and Marijuana Groups

ID	PTSD Symptoms	PTSD Symptom Severity						PTSD Symptom Endorsement ^d				Test Statistics	
		Alcohol Only		Alcohol and Marijuana		Test Statistics		Alcohol Only		Alcohol and Marijuana		Test Statistics	
		M	SD	M	SD	p	η^2	%	%	OR	95% CI		
I	Intrusion	5.79	4.81	5.92	4.90	.672	<.001	58.1	62.8	1.18	0.81–1.70		
I1	Unwanted memories	1.30	1.12	1.35	1.17	.559	.001	36.2	37.7	1.06	0.73–1.54		
I2	Disturbing dreams	0.81	1.14	0.88	1.16	.470	.001	22.5	24.6	1.08	0.71 – 1.62		
I3	Flashback	0.92	1.13	0.92	1.19	.903	<.001	25.7	27.1	1.05	0.70 – 1.57		
I4	Distress by reminder	1.68	1.29	1.65	1.26	.741	<.001	48.5	49.2	1.05	0.74 – 1.51		
I5	Physiological reactivity	1.08	1.25	1.12	1.27	.503	.001	31.4	33.7	1.13	0.77–1.65		
A	Avoidance	3.13	2.64	3.31	2.63	.972	.002	50.9	56.3	1.24	0.87–1.78		
A1	Avoidance of thoughts	1.67	1.36	1.79	1.39	.331	.002	47.3	47.7	1.23	0.86–1.76		
A2	Avoidance of people/places	1.46	1.42	1.52	1.38	.466	.001	42.2	44.2	1.11	0.78–1.60		
N	Negative cognitions	6.78	7.05	8.87	7.70	.001	.021	39.8	54.8	1.85**	1.29–2.66		
N1	Traumatic event amnesia	0.80	1.20	1.06	1.34	.025	.009	23.1	29.6	1.42	0.95–2.12		
N2	Negative beliefs	1.12	1.35	1.44	1.42	.016	.011	31.4	44.2	1.69**	1.17 – 2.45		
N3	Blame	1.21	1.37	1.38	1.43	.076	.006	34.7	39.7	1.27	0.88 – 1.84		
N4	Negative feelings	1.24	1.31	1.48	1.38	.044	.008	37.1	40.7	1.18	0.82 – 1.70		
N5	Loss of interest	0.70	1.16	0.96	1.29	.004	.016	19.5	27.1	1.63*	1.07 – 2.49		
N6	Feeling disconnected	0.91	1.24	1.40	1.42	<.001	.034	25.7	39.7	2.00**	1.36 – 2.94		
N7	Negative affect	0.79	1.19	1.14	1.36	.001	.022	22.5	33.2	1.78**	1.19–2.66		
H	Hyperarousal	5.38	5.69	6.80	6.13	.003	.017	38.3	46.2	1.43	0.99–2.06		
HI	Irritable or aggressive	0.70	1.11	1.05	1.35	<.001	.024	19.8	31.2	2.02**	1.34–3.06		
H2	Risk taking	0.43	0.94	0.66	1.05	.008	.013	12.3	20.6	1.88*	1.16 – 3.04		

ID	PTSD Symptoms	PTSD Symptom Severity						PTSD Symptom Endorsement ^a					
		Alcohol Only		Alcohol and Marijuana		Test Statistics		Alcohol Only		Alcohol and Marijuana		Test Statistics	
		M	SD	M	SD	p	η^2	%	%	OR	95% CI		
H3	Hypervigilant	1.20	1.35	1.41	1.39	.058	.007	36.2	40.7	1.25	0.86 – 1.80		
H4	Startle response	0.87	1.21	1.01	1.25	.203	.003	25.1	24.6	0.97	0.64 – 1.46		
H5	Concentration problems	1.02	1.27	1.25	1.37	.015	.011	30.2	36.2	1.36	0.93 – 1.99		
H6	Sleep problems	1.15	1.38	1.43	1.45	.015	.011	34.7	42.2	1.47*	1.02 – 2.13		
T	Total	21.08	18.27	24.91	19.47	.012	.012	28.1	36.2	1.48*	1.01 – 2.17		

Note. All analyses are adjusted for time since trauma and age. Odds Ratios for Intrusion, Avoidance, Negative Cognition, Hyperarousal, and Total represent odds of meeting criteria for each symptom cluster and for the provisional diagnosis of PTSD.

^aSymptom endorsement defined as participant reporting 2 (“Moderately”) or greater symptom severity on a given symptom on the PTSD Checklist for DSM-5 (PCL-5; Weathers et al., 2013b).

* p < .05

** p < .01