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Brief report

# Posttraumatic stress disorder with and without alcohol use disorders: Diagnostic and clinical correlates in a psychiatric sample

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ABSTRACT

This study compared outpatients ($n = 196$) with PTSD versus PTSD + alcohol use disorders (AUD) on clinical measures. PTSD + AUD patients were more likely to meet criteria for Borderline and Antisocial Personality Disorders. Emotion dysregulation may help account for the relationship between PTSD and AUD.



## 1. Introduction

It is estimated that 6.8% of individuals in the United States experience Posttraumatic Stress Disorder (PTSD) in their lifetime, and often in combination with alcohol use disorders (AUD) (Kessler et al., 2005a,b). Among clinical populations, approximately 40% of patients presenting for substance abuse treatment meet criteria for co-occurring PTSD (Ouimette et al., 2005; Read et al., 2004). Patients with co-occurring PTSD and substance use disorders have more severe PTSD symptoms than those with PTSD alone (Ouimette et al., 1996; Saladin et al., 1995). Moreover, a considerable body of research has documented the deleterious effects of co-occurring PTSD on substance abuse treatment outcomes with dually diagnosed patients exhibiting poorer substance use and psychosocial outcomes (Brown et al., 1995; Ouimette et al., 1999).

Several models of addictions have attempted to delineate the relationship between PTSD and substance use. These models include the Stress-Response Dampening (SRD; Levenson et al., 1980), which posits that individual differences in the stress-response dampening effects of alcohol predicts the development of alcohol problems. Researchers have suggested that dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis in response to stress may provide a mechanistic link between the two disorders (Brady et al., 2006a,b). Similarly, the co-occurrence of PTSD and AUD has been explained in the context of the self-medication hypothesis, by which the substance use disorder develops as an attempt to alleviate the unpleasant symptoms of PTSD, particularly the symptoms of increased physiological hyperarousal (Brady et al., 2000; Kosten and Krystal, 1988). According to this model, substance use is driven by negative reinforcement (Miller et al., 2006).

Recently, a growing body of research has focused on the influence of personality variables in the link between PTSD and substance use disorders. Specifically, the dimension of disconstraint, or behavioral undercontrol, has been found to predict comorbid substance use disorders in individuals with the externalizing subtype of PTSD (Miller et al., 2004; Miller and Resick, 2007). A recent study by Miller and colleagues (2006) found that disconstraint mediated the relationship between PTSD and alcohol/drug use outcomes.

Examining differences in PTSD symptom profiles, as well as diagnostic and clinical variables, between PTSD patients with and without comorbid AUD has the potential to elucidate some of the clinical mechanisms underlying the association between these disorders. To that end, this report from the Rhode Island Methods to Improve Diagnostic Assessment and Service (MIDAS) Project will compare psychiatric outpatients who met lifetime criteria for PTSD to those who met criteria for PTSD + AUD on measures of PTSD symptomatology, diagnostic, and clinical variables. Specifically, we sought to investigate the role of hyperarousal and personality factors in the relationship between PTSD and AUD in a clinical sample. We hypothesized that patients with co-occurring PTSD and AUD would exhibit greater levels of hyperarousal, Axis I, and Axis II psychopathology than those with PTSD alone.

## 2. Method

### 2.1. Participants

Participants were recruited from the Rhode Island Hospital Department of Psychiatry's outpatient practice (Zimmerman, 2003). In an initial telephone screen,

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patients were invited to participate in a face-to-face diagnostic evaluation prior to meeting with their treating clinician. The current report is based on 196 (84% female) patients who met criteria for PTSD in their lifetime and for whom symptom-level data were available. Patients who met criteria for any drug use disorders or for whom the onset of AUD predated the onset of PTSD ($n=31$) were excluded from these analyses in order to more directly examine the relationship between PTSD symptoms thought to increase the risk of subsequent AUD. Of the 196 patients, 158 (85% female) met criteria for PTSD without comorbid substance use disorders and 38 (82% female) met criteria for PTSD and AUD (58% met lifetime criteria for alcohol dependence and 42% for alcohol abuse). An AUD-only comparison group ($n=296$, 42.6% female) was also used to probe for the findings. See Table 1 for additional demographic information.

### 2.2. Procedures and assessments

DSM-IV Axis I diagnoses were obtained using the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I/P; First et al., 1995) and the Structured Interview for DSM-IV Personality (SIDP-IV; Pfohl et al., 1997) assessed DSM-IV Axis II personality disorders. Clinical Global Impression–Severity of depression (CGI-S; Guy, 1976) and Global Assessment of Functioning (GAF) ratings were also obtained for each patient by the diagnostic rater. The Family History Research Diagnostic Criteria (FH-RDC; Andreasen et al., 1977) interview was used to assess family history of psychiatric disorders among first degree relatives. The inter-rater reliability of the diagnoses in the MIDAS study is adequate (Zimmerman and Mattia, 1999; Zimmerman et al., 2005) with a previously reported Kappa coefficient, $\kappa=0.91$ for PTSD and $\kappa=0.64$ for AUD (Zimmerman et al., 2008). The Rhode Island Hospital institutional review board approved the research protocol and after complete description of the study written informed consent was obtained from each participant.

### 2.3. Data analysis

The primary analytic approach involved comparing PTSD patients with and without comorbid AUD on measures of PTSD symptomatology, diagnostic, demographic, and clinical variables. For the purpose of this investigation, diagnostic categories were combined (e.g., cluster A personality disorders), when appropriate, in order to increase overall base rates (i.e., cell sizes), thereby increasing statistical power to detect group differences and minimizing the chances of type I error due to multiple comparisons. Student t-tests and Chi-square tests were used for tests of continuous and categorical variables, respectively. Student t-tests were used as they account for potential violations of the assumption of the homogeneity of variance, an important issue when comparing two groups with an unequal number of participants ($n$). Specifically, for variables in which the homogeneity of variance assumption was held, results of $t$-tests using pooled variance are reported, whereas for variables that violated the homoscedasticity assumption in our sample, the individual sample standard deviation was used to calculate the $t$-test.

## 3. Results

As shown in Table 1, the two groups did not differ significantly on demographic measures or on measures of PTSD symptomatology, either in terms of symptom count within criterion or individual PTSD symptoms ($P>0.10$). Regarding diagnostic variables, patients with PTSD + AUD were significantly more likely to meet DSM-IV criteria for cluster B personality disorders, as compared to PTSD only patients (32.4% s. 14.7%). Analyses of the individual cluster B disorders indicated that there was a significantly higher likelihood of comorbid Borderline Personality Disorder (BPD) and Antisocial Personality Disorder (ASPD) among the PTSD + AUD group. The groups did not differ significantly on the prevalence of other axis I diagnoses ($P>0.10$). Analyses of clinical characteristics suggested that the PTSD + AUD group had a later age of onset of PTSD [29.5(10.6) vs. 19.3(12.3)] and lower GAF scores [50.7 (9.0) vs. 46.1 (13.4)], as compared to the PTSD only group. Group differences in the prevalence of comorbid BPD and ASPD remained significant even after controlling for group differences on age of onset of PTSD.

In order to further probe the group differences in lifetime ASPD and BPD diagnoses, an AUD-only comparison group ($n=296$) was added. Results revealed that the AUD-only group had a lower lifetime prevalence of ASPD (2.6%) compared to the PTSD-only group (3.4%) and the PTSD + AUD group (13.5%). The AUD-only group did not differ significantly from the PTSD-only group ($\chi^2[1]<1.0$, $P=0.63$) on lifetime ASPD, however PTSD + AUD patients were significantly more likely to meet criteria for ASPD than AUD-only patients ($\chi^2[1]=10.5$, $P<0.01$), suggesting that the lifetime co-occurrence of PTSD and AUD may be partially accounted for by the higher rates of ASPD. A similar pattern was noted for BPD such that lifetime rates of BPD were lower in the AUD-only group (7.4%), as compared to the PTSD-only group (13.3%) ($\chi^2[1]=4.03$, $P<0.05$), and the PTSD + AUD group (30.6%) ($\chi^2(1)=18.9$, $P<0.0001$). These results provide further support for the robustness of the relationships reported above.

**Table 1**
Demographic, diagnostic, and clinical characteristics by presence of lifetime alcohol use disorders (AUD) in psychiatric outpatients with a lifetime PTSD.

| | 1. PTSD ONLY ($n=158$) | 2. PTSD + AUD ($n=38$) | 3. AUD ONLY ($n=296$) | $t/\chi^2$ Group1 vs. 2 | $P$ |
|---|---|---|---|---|---|
| **Demographic characteristics** | | | | | |
| Age, *M* (S.D.) | 36.0 (11.9) | 36.3 (12.4) | 39.9 (11.7) | -0.16 | 0.87 |
| Gender: Female, % | 85.4 | 81.6 | 42.6 | 0.35 | 0.55 |
| Ethnicity: Caucasian, % | 77.9 | 84.2 | 91.2 | 0.75 | 0.39 |
| Marital status, % married | 50.6 | 26.3 | 52.0 | 7.4 | 0.06 |
| Education, % college degree/higher | 21.5 | 18.4 | 33.8 | 2.19 | 0.53 |
| **Diagnostic characteristics** | | | | | |
| PTSD Criteria, Symptom Count | | | | | |
| Criterion B, *M* (S.D.) - Range: 1-5 | 3.4 (1.2) | 3.5 (1.1) | n/a | -0.23 | 0.82 |
| Criterion C, *M* (S.D.) - Range: 3-7 | 4.5 (1.2) | 4.4 (1.2) | n/a | 0.04 | 0.97 |
| Criterion D, *M* (S.D.) - Range: 2–5 | 3.6 (1.1) | 3.8 (1.1) | n/a | -1.16 | 0.25 |
| Axis I Disorders - Lifetime History, % | | | | | |
| Major Depressive Disorder | 84.8 | 81.6 | 73.3 | 0.24 | 0.62 |
| Bipolar Disorder (I or II) | 9.5 | 7.8 | 6.1 | 0.09 | 0.76 |
| Panic Disorder | 25.3 | 31.6 | 15.2 | 0.62 | 0.43 |
| Social Anxiety Disorder | 40.5 | 47.4 | 30.1 | 0.59 | 0.44 |
| Generalized Anxiety Disorder | 18.4 | 21.1 | 18.9 | 0.15 | 0.70 |
| Axis II Disorders, % | | | | | |
| Cluster A | 5.1 | 0.0 | 4.4 | 1.28 | 0.26 |
| Cluster B | 14.7 | 32.4 | 10.2 | 6.29 | <0.05 |
| Borderline PD | 13.3 | 30.6 | 7.4 | 6.20 | <0.05 |
| Antisocial PD | 3.4 | 13.5 | 2.6 | 5.95 | <0.05 |
| Histrionic PD | 0.0 | 4.2 | 0.5 | 3.27 | 0.20* |
| Narcissistic PD | 1.0 | 8.3 | 3.0 | 3.20 | 0.10* |
| Cluster C | 29.9 | 16.7 | 21.7 | 1.70 | 0.19 |
| **Clinical characteristics** | | | | | |
| Age of onset of PTSD, *M* (S.D.) | 19.3 (12.3) | 29.5 (10.6) | n/a | -4.62 | <0.0001 |
| History of a suicide attempt, % | 29.8 | 44.7 | 15.2 | 3.13 | 0.08 |
| History of psych hospitalization, *M* (S.D.) | 29.3 | 26.3 | 22.3 | 0.13 | 0.72 |
| Family history positive alcoholism, % | 45.8 | 62.2 | 42.2 | 3.20 | 0.07 |
| GAF rating, *M* (S.D.) | 50.7 (9.0) | 46.1 (13.4) | 54.6 (9.8) | 2.04 | <0.05 |
| CGI-S rating, *M* (S.D.) | 2.7 (1.0) | 2.6 (1.1) | 2.1 (1.2) | 0.63 | 0.53 |

* As recommended for cases of small cell sizes (<5 observations per cell), Fisher's Exact Test was used instead of standard Chi-Square tests in these analyses.

## 4. Discussion

The objective of this study was to compare psychiatric outpatients who met lifetime criteria for PTSD to those who met criteria for PTSD + AUD on measures of PTSD symptomatology, diagnostic, and clinical variables in order to elucidate the clinical correlates underlying the association between these disorders. Consistent with our hypothesis and previous etiological research, the present results indicate that individuals with lifetime PTSD + AUD were more likely to meet criteria for cluster B personality disorders, namely Borderline PD and Antisocial PD, as compared with individuals with PTSD only. Although no causal links can be inferred, these results raise the question of whether emotion dysregulation and poor distress tolerance, associated with personality disorders such as BPD and ASPD, may be relevant in explaining the association between PTSD and the subsequent development of AUD. Future research examining emotion regulation and distress tolerance constructs as mediators of PTSD and the subsequent onset of substance use disorders would address this empirical question.

The present findings are consistent with previous research demonstrating greater borderline personality traits among women with co-occurring PTSD and alcohol abuse as compared with women with PTSD alone (Ouimette et al., 1996). Additionally, results of this study may provide further support for the role of externalizing behaviors in the relationship between PTSD and AUD. While we did not explicitly assess disconstraint, our finding that PTSD + AUD patients had greater incidence of ASPD, an externalizing disorder characterized in part by disconstraint (Miller et al., 2007), lends credence to the role of personality variables in this comorbidity (Miller et al., 2006). Conversely, the diagnostic overlap observed in this study may reflect common etiological pathways.

In contrast to our expectations and previous research suggesting a positive association between hyperarousal symptoms and alcohol use and misuse (Back et al., 2006; Taft et al., 2007), this study found no between-group differences at the PTSD criterion or individual symptom level. Moreover, the results did not support the self-medication hypothesis; patients with comorbid PTSD + AUD did not report increased symptoms of physiological hyperarousal, as defined in Criterion D of the DSM-IV diagnostic criteria. The self-medication hypothesis posits that hyperarousal experienced as insomnia, irritability, impaired concentration, hypervigilance, and exaggerated startle response may be temporarily relieved by alcohol use (Brady et al., 2000), but this finding was not replicated in our sample.

Limitations of the present study include the small sample size and the cross-sectional nature of the data. Additionally, as the majority of patients in our sample were women (84%), these results may not generalize to male psychiatric outpatients. These results should be interpreted with caution as they are based on lifetime prevalence reports in a mixed-aged sample (Kraemer et al., 2006). However, the well ascertained age of onset, the relatively small variance around the mean age of the sample, and the fact that PTSD and personality disorder diagnoses generally stay stable over time all serve to reduce the risk of pseudocomorbidity (Kraemer et al., 2006). Nevertheless, in the absence of a more detailed timeline among the diagnoses under study, comorbidity cannot be assumed as the disorders may or may not have co-occurred in time.

In summary, these results extend previous findings due to our focus on a treatment-seeking general psychiatric sample. This is especially relevant as seeking treatment is related to a number of clinical, social, and demographic factors (Alegria et al., 2000; Goodwin et al., 2002), suggesting that studies of the frequency and correlates of psychiatric disorders should be replicated in clinical populations to provide the practicing clinician with information that might have more direct clinical utility. These findings at the diagnostic level of analysis also suggest avenues for basic research on common risk factor for psychopathology and suggest that further examination of emotion regulation and distress tolerance may be useful in understanding the biobehavioral mechanisms underlying the risk for PTSD, AUD, and their lifetime co-occurrence.

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