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Anxiety Sensitivity, Emotional Avoidance, and PTSD Symptom Severity Among Crack/Cocaine Dependent Patients in Residential Treatment

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Abstract

High rates of co-occurring posttraumatic stress disorder (PTSD) have been found among patients receiving treatment for substance use disorders (SUD), and there is evidence that this particular co-occurrence is associated with negative SUD treatment outcomes. Thus, there is utility in establishing the role of psychological vulnerabilities related to PTSD within SUD populations, with the goal of ultimately informing targeted interventions and improving clinical outcomes. Anxiety sensitivity (AS) and emotional avoidance (EA) may be two important factors in this regard, as both have been found to demonstrate associations with posttraumatic stress in other clinical and nonclinical populations. To expand upon this literature, the current study examined the associations between AS and EA and PTSD symptom severity in a sample of traumatic event-exposed crack/cocaine dependent patients in residential SUD treatment ($n = 62$), as well as the extent to which EA mediated the relation between AS and PTSD symptom severity. As hypothesized, AS and EA were associated with PTSD symptom severity above and beyond the effects of gender and non-specific anxiety symptoms. However, the hypothesis that EA would mediate the relation between AS and PTSD symptom severity was only partially supported. Implications of these findings for understanding and treating co-occurring SUD-PTSD are discussed.

Keywords

Anxiety sensitivity; Cocaine; Comorbidity; Emotional avoidance; Posttraumatic stress disorder; Substance use disorder

Substance use disorders (SUD) and posttraumatic stress disorder (PTSD; American Psychiatric Association 2000) co-occur at high rates (Kessler et al. 1995), particularly among treatment-seeking substance abusers, where reported rates of lifetime and current

PTSD are 36–50 and 25–42%, respectively (for a review, see Jacobsen et al. 2001). The co-occurrence of these disorders is also clinically relevant, as co-occurring SUD-PTSD has been found to be associated with worse SUD treatment outcomes than both SUD alone and the co-occurrence of SUD with other Axis I disorders (Ouimette et al. 1997, 1999). Thus, a greater understanding of the psychological factors associated with PTSD symptom severity in substance-dependent populations may inform useful targets for interventions, ultimately mitigating the negative treatment outcomes associated with SUD-PTSD co-occurrence.

One such relevant factor may be anxiety sensitivity (AS), a cognitive vulnerability that has been implicated in a wide range of psychiatric problems, including PTSD (Taylor 1999, 2003). AS is defined as the tendency to fear anxiety-related symptoms due to beliefs that these symptoms will have harmful physical, social, or cognitive consequences (Reiss 1991). Research has consistently demonstrated an association between AS and PTSD (for a review, see Elwood et al. 2009). Further, PTSD appears to be associated with higher levels of AS than all other anxiety disorders with the exception of panic disorder (for which levels of AS are comparable to those observed among individuals with PTSD; Taylor et al. 1992). Stronger evidence for the role of AS in posttraumatic stress comes from studies that utilize a prospective research design. For example, Feldner et al. (2008) found that baseline AS uniquely predicted posttraumatic symptoms at the 12–24 month follow-up in a large nonclinical sample of young adults, above and beyond trait anxiety, baseline posttraumatic stress, and treatment condition (i.e., a brief, computerized AS intervention versus a control condition). Additionally, Fedoroff et al. (2000) found that reductions in AS corresponded with reductions in PTSD symptom severity over the course of cognitive behavioral treatment among survivors of motor vehicle accidents. Further, Marshall et al. (2010) found that AS predicts PTSD symptoms 6 and 12 months post-physical injury. Similar associations between AS and PTSD have also been observed among individuals in SUD treatment. For example, a cross-sectional study of patients in a community-based SUD treatment program found that individuals classified as having PTSD (based on a self-administered questionnaire) reported greater AS than those without PTSD and those with subclinical PTSD symptoms (Bonin et al. 2000). Further, a prospective study of patients receiving treatment for alcohol use disorders found that baseline AS was associated with the severity of PTSD symptoms concurrently and at 1 month follow-up (Simpson et al. 2006).

Another factor relevant to PTSD symptom severity is emotional avoidance (EA), considered to be an underlying mechanism of various forms of psychopathology, including PTSD (e.g., Chawla and Ostafin 2007; Hayes et al. 1996; Salters-Pedneault et al. 2004). EA is subsumed under the broader construct of experiential avoidance (Hayes et al. 1996), which consists of efforts to avoid or alter the form, frequency, or contexts of aversive internal experiences (e.g., thoughts, emotions, and bodily sensations). Although avoidance of trauma-specific thoughts and emotions is already recognized as a core feature of PTSD (American Psychiatric Association 2000), recent theoretical and empirical literature suggests that the generalized avoidance of aversive internal experiences (i.e., experiences that are not necessarily trauma-related) is associated with PTSD as well. For example, Roemer et al. (2001) found that combat veterans with PTSD reported more frequent and extreme withholding of emotions (e.g., concealment of emotions across multiple situations, concealment of all emotional reactions) than non-PTSD controls, and experiential avoidance has been found to be associated with PTSD and depression above and beyond the severity of combat exposure in a sample of veterans presenting for inpatient PTSD treatment (Plumb et al. 2004). Additionally, Marx and Sloan (2002) reported that experiential avoidance mediated the relation between reports of childhood sexual abuse and general psychological distress among female college students. Prospective studies have provided even stronger support for the role of experiential avoidance as a vulnerability factor for the development of post-stressor psychiatric problems. In a series of studies, Plumb et al. (2004) found that

experiential avoidance among college students was prospectively associated with greater psychological distress following exposure to stressful life events, and greater PTSD symptom severity following exposure to traumatic events. Likewise, in another prospective study of trauma-exposed college students, Marx and Sloan (2005) found that greater self-reported experiential avoidance at the initial assessment predicted PTSD severity both 4 and 8 weeks later, even after accounting for initial PTSD symptom levels. Despite these interesting findings, however, understanding the role of general emotional avoidance following a traumatic event is complicated by its conceptual overlap with PTSD-specific avoidance. Consequently, avoidance is often a dimension of both the independent and dependent variables in such studies.

In light of these findings, the aim of the current study was to examine AS and EA as potential emotion-related factors associated with PTSD symptom severity among a sample of crack/cocaine dependent patients with a history of traumatic event exposure. Crack/cocaine dependent patients are a particularly relevant population in which to explore these associations, given evidence of high levels of PTSD (Cottler et al. 1992), AS (Lejuez et al. 2006; McDermott et al. 2009), and emotion dysregulation (Fox et al. 2007) within this population. Further, given that previous theoretical and empirical work on the role of AS and EA in other trauma-exposed populations has informed the development of new treatment models (e.g., Hayes et al. 1999; Taylor 1999), understanding the relevance of these constructs to posttraumatic stress within SUD populations may highlight potentially useful targets of intervention among SUD patients with PTSD (an important consideration in light of the negative outcomes associated with this specific diagnostic co-occurrence; Hien et al. 2000; Najavits et al. 1999).

We hypothesized that AS and EA would be associated with PTSD symptom severity above and beyond any shared variance with relevant demographic variables and non-specific anxiety symptoms. In addition, in light of literature suggesting that fear of anxious arousal (i.e., AS) may motivate avoidance behaviors aimed at reducing this emotional distress (i.e., EA; Zvolensky and Forsyth 2002), and that persistent avoidance may, in turn, interfere with the processing of fear-based emotional responses (e.g., habituation, corrective learning; Foa et al. 2006; Foa and Kozak 1986), we further hypothesized that EA would mediate the association between AS and PTSD symptom severity. Specifically, by preventing the emotional processing of fear, EA may be a mechanism through which AS contributes to greater PTSD symptom severity (Taylor 2003). According to this perspective, it is pervasive efforts to avoid aversive emotions, rather than the emotions themselves, that leads to the development and maintenance of psychopathology, such as PTSD (Hayes et al. 1996). We are not aware of any studies examining this mediational model as a predictor of posttraumatic stress; however, previous research has found that experiential avoidance mediates the association between dimensions of AS and various other psychiatric and behavioral problems, including depression (Tull and Gratz 2008), borderline personality disorder (Gratz et al. 2008), and coping-motivated drinking (Stewart et al. 2002). Although the cross-sectional nature of the current study precludes a true test of mediation (which requires a specific temporal relationship among the variables in question, such that the mediator must occur after predictor and before the outcome; Kraemer et al. 2002), it does provide a preliminary test of this mediational model, similar to that provided in the studies cited above (Gratz et al. 2008; Stewart et al. 2002; Tull and Gratz 2008).

Method

Participants

Data were collected from 62 participants who had recently entered a residential substance abuse treatment center in Northeast Washington, DC for crack/cocaine dependence.

Participants ranged in age from 20 to 58 years ($M = 44.45$, $SD = 7.05$), and 53.23% ($n = 33$) were male. The vast majority of participants identified their race as Black/African American (96.77%, $n = 60$). Participants were predominantly single (75.81%, $n = 47$), unemployed (85.48%, $n = 53$), and reported a total family household income of less than \$10,000 (78.69%, $n = 48$). Self-reported history of trauma exposure was an inclusion criterion for the current study, and all individuals who agreed to participate reported experiencing at least one traumatic event (see “Results”).

Treatment at the center from which participants were recruited involves a mix of strategies adopted from Alcoholics and Narcotics Anonymous as well as group sessions focused on relapse prevention and functional analysis. Detoxification (if needed) is required prior to entering the treatment facility, minimizing the possibility of severe or acute withdrawal symptoms. Further, complete abstinence from drugs and alcohol is required upon entry into the center and throughout the duration of the program, with the exception of caffeine and nicotine; regular drug testing is provided and any drug or alcohol use results in immediate dismissal from the center. Aside from scheduled activities (e.g., group retreats, physician visits), residents are not permitted to leave the center grounds during treatment.

Measures

Traumatic Event Exposure—The *Life Events Checklist* (LEC; Blake et al. 1990) is a 17-item measure assessing exposure to a wide range of events that are consistent with the PTSD Criterion A1 definition of a traumatic stressor. Respondents rate their degree of exposure to each event on a 5-point nominal scale (1 = *happened to me*, 2 = *witnessed it*, 3 = *learned about it*, 4 = *not sure*, and 5 = *does not apply*). As a measure of direct trauma exposure (i.e., *happened to me*), the LEC has previously demonstrated adequate one-week test–retest reliability (mean kappa = .61, $r = .82$), with lower estimates for the full range of responses (mean kappa = .47; Gray et al. 2004). Convergent validity with the Traumatic Life Events Questionnaire (Kubany et al. 2000), an established measure of trauma exposure, was also found to be adequate (mean kappa = .55, $r = -.55$; Gray et al. 2004). Internal consistency of the LEC in the current study was .66. After completing the LEC, participants were asked to identify the event they experienced as most traumatic; this event was used as the index event for the assessment of PTSD symptoms (see below).

PTSD Symptom Severity—The *Clinician-Administered PTSD Scale* (CAPS; Blake et al. 1995, 1990) is a structured PTSD diagnostic interview and the most widely used PTSD measure (Elhai et al. 2005). It assesses the frequency and intensity of the 17 *DSM-IV* PTSD symptoms (plus eight associated symptoms). Frequency items are rated from 0 (*never or none/not at all*) to 4 (*daily or almost every day or more than 80%*). Intensity items are rated from 0 (*none*) to 4 (*extreme*). The CAPS has adequate interrater reliability (.92–.99), internal consistency (.73–.85), and convergent validity with the *Structured Clinical Interview for DSM-IV* and other established measures of PTSD (Weathers et al. 2001). Frequency and intensity ratings are summed for an overall PTSD severity score or can be used to generate a categorical diagnosis (Weathers et al. 1999). Given evidence that PTSD is best represented as a dimensional construct (Broman-Fulks et al. 2006; Forbes et al. 2005; Ruscio et al. 2002), we utilized overall CAPS severity scores as the dependent variable. For descriptive purposes, we also report diagnostic status using the *Item Severity* 4 (*ISEV4*) rule, which requires that at least one reexperiencing, three avoidance/emotional numbing, and two hyperarousal symptoms have a severity rating (frequency + intensity) of 4. The CAPS was administered by trained interviewers (post-baccalaureate to Ph.D.-level mental health professionals). All interviews were reviewed by a Ph.D. level clinician (MTT). In the four cases for which a discrepancy was evident, areas of disagreement were discussed as a group and a consensus was reached.

Anxiety Sensitivity—The *Anxiety Sensitivity Index* (ASI; Peterson and Reiss 1992) is a 16-item, self-report measure of AS. Using a 5-point Likert scale (0 = *very little* to 4 = *very much*), participants rate the extent to which they expect negative physical (e.g., “When I notice my heart is beating rapidly, I worry that I might have a heart attack”), social (e.g., “Other people notice when I feel shaky”), and cognitive (e.g., “When I cannot keep my mind on a task, I worry that I might be going crazy”) consequences from a variety of anxiety-related experiences. The ASI has been found to have good internal consistency and test–retest reliability, as well as adequate criterion, construct, and predictive validity (Antony 2001; Peterson and Reiss 1992; Schmidt et al. 1997, 1999; Zvolensky et al. 2001). Internal consistency of the ASI in the current study was good ($\alpha = .93$).

Emotional Avoidance—The *Emotional Avoidance Questionnaire* (EAQ; Taylor et al. 2004) is a 20-item self-report measure assessing various cognitive and behavioral aspects of emotional avoidance, including the avoidance of positive emotions (e.g., “If I start to feel strong positive emotions, I prefer to leave the situation”), negative beliefs about emotions (e.g., “I cannot handle feeling anxious or worried about things”), social concerns about displaying emotions (e.g., “I try to keep feelings of anxiety or worry to myself so that other people don’t think less of me”), and the avoidance of negative emotions (e.g., “If sad thoughts cross my mind, I try to push them away as much as possible”). Items are rated on a 5-point Likert scale (1 = *not true of me*, to 5 = *very true of me*). The EAQ subscales have demonstrated fair to good internal consistency (.66–.84) across multiple studies using college and treatment-seeking samples (Taylor et al. 2004; Tull et al. in press), and have been found to be significantly associated with severity of avoidant personality disorder symptoms (Taylor et al. 2004), as well as depression and anxiety symptom severity, worry, and PTSD symptom severity within a nonclinical population (Tull et al. in press). In this sample, internal consistencies were good for the full EAQ ($\alpha = .92$) and all EAQ subscales (alphas ranging from .75 to .83). Although the avoidance of trauma-related emotions is encompassed by general emotional avoidance, the EAQ is not trauma-specific and none of the items are keyed to traumatic experiences.

Anxiety Severity—The *Depression Anxiety Stress Scales* (DASS; Lovibond and Lovibond 1995) is a self-report questionnaire designed to differentiate between core symptoms of depression, anxiety, and stress. The DASS has demonstrated adequate test–retest reliability (Brown et al. 1997), and there is evidence for its construct and discriminant validity (Antony et al. 1998; Brown et al. 1997; Lovibond and Lovibond 1995). The 21- and 42-item versions of the DASS have been found to be consistent (Clara et al. 2001) and comparable in their ability to distinguish between different diagnostic groups (Antony et al. 1998); thus, the 21-item version was used in this study. Specifically, the anxiety subscale (DASS-A) was examined as a potential covariate, given evidence that associations between AS and various outcomes may be partly attributed to relations between AS and non-specific anxiety (Lilienfeld 1999). Internal consistency of the DASS-A subscale in the current study was good ($\alpha = .87$).

Past Year Substance Use Severity—Past year severity of alcohol and drug use was also assessed as a potential covariate in subsequent analyses. This variable was assessed through a self-report measure that asks participants to rate the frequency with which they have used 10 different substances (alcohol, cannabis, cocaine, MDMA, stimulants, sedatives, opiates, hallucinogens [other than PCP], PCP, and prescription drugs [illegal usage]) in the past year using a 6-point Likert scale (0 = *never*; 1 = *one time*; 2 = *monthly or less*; 3 = *2 to 4 times per month*; 4 = *2 to 3 times per week*; 5 = *4 or more times a week*). Despite being a non-published measure, this measure was modeled after established, psychometrically-sound measures (e.g., the Alcohol Use Disorders Identification Test;

Saunders et al. 1993) and has been used in multiple studies of inpatient substance users (e.g., Bornovalova et al. 2009; Daughters et al. 2008; Lejuez et al. 2006, 2007; McDermott et al. 2009). Further, scores on this measure have been found to be associated with variables relevant to the present study, including anxiety sensitivity, emotion dysregulation, and PTSD symptom severity (Bornovalova et al. 2009; Lejuez et al. 2006), as well as other SUD-relevant variables (e.g., impulsivity; Lejuez et al. 2007). Responses were summed to create an overall score representing past year substance use severity. This variable was used as a potential covariate in subsequent analyses. Internal consistency in the current study was adequate ($\alpha = .73$).

Procedure

Data for this study were collected as part of a larger study focused on the functional relationship between PTSD and crack/cocaine use. To be eligible for the larger study, participants were required to: (1) be 18 to 65 years of age; (2) meet criteria for crack/cocaine dependence; (3) have been in residential treatment for at least 72 h (in order to limit the potential impact of withdrawal symptoms on responding); (4) exhibit no significant cognitive impairment; and (5) not meet criteria for a current manic episode or psychotic disorder. All study procedures received Institutional Review Board approval. Eligible participants were informed that involvement in the study was voluntary and refusal to participate would not affect their treatment status. Participants who provided written informed consent were administered diagnostic interviews, including the CAPS, and then completed the self-report measures described above. Participants were provided with gift cards (\$30) to a local grocery store in exchange for their participation.

Results

Rates of Traumatic Exposure and PTSD

The total number of potentially traumatic events reported by participants varied greatly, from 1 to 102 ($M = 13.6$, $SD = 18.35$). Types of events most commonly endorsed on the LEC included the sudden, unexpected death of someone close (78.69%, $n = 48$), physical assault (68.85%, $n = 42$), transportation accident (62.3%, $n = 38$), assault with a weapon (59.02%, $n = 36$), and sexual assault (45.9%, $n = 28$). All participants met PTSD Criterion A for exposure to a traumatic event based on the CAPS, including the subjective experience of fear, helplessness, or horror. The most common index traumatic events serving as the basis for the PTSD assessment were serious injury or death caused to someone else (30.36%), being assaulted with a weapon (19.64%), other stressful event or experience (i.e., an event not falling into one of the LEC categories; 16.07%), and sexual assault (12.50%). CAPS current PTSD total scores ranged from 0 to 105 ($M = 43.79$, $SD = 31.94$), with 50% ($n = 31$) of participants meeting criteria for current PTSD based on the *ISEV4* rule (Weathers et al. 1999).

Identification of Covariates

Prior to conducting primary analyses, a series of analyses was conducted to identify potential covariates (i.e., any variable found to be significantly associated with the dependent variable; Tabachnick and Fidell 2001). Women reported significantly greater PTSD symptom severity ($M = 60.1$, $SD = 31.57$) than men ($M = 29$, $SD = 24.49$), $t(59) = 4.32$, $p < .001$. Further, anxiety severity was positively associated with PTSD symptom severity ($r = .52$, $p < .01$). No significant relations were found between PTSD symptom severity and age ($r = -.03$, $p = .80$), income level (\$9,999 vs. \$10,000; $t[60] = 1.61$, $p = .11$), employment status (unemployed vs. employed; $t[60] = 1.67$, $p = .10$), marital status (single vs. cohabitating/married; $t[60] = 1.20$, $p = .24$), or past year substance use severity (r

$= -.06, p = .63$). Therefore, only gender and anxiety severity were included as covariates in primary analyses.

Primary Analyses

Means, standard deviations, and bivariate correlations for all variables of interest are provided in Table 1. In order to examine whether AS and EA were associated with PTSD symptom severity (as indexed by the total score on the CAPS), we conducted a series of hierarchical linear regressions, controlling for the effects of gender and anxiety severity in the first step. The basic model including only the covariates was significant, $F(2, 58) = 18.62, p < .001$, accounting for 39% of the variance in PTSD symptom severity. Next, AS was included in the second step of the model. As hypothesized, AS ($\beta = .35$) accounted for a significant amount of variance above and beyond the covariates, $\Delta F(1, 57) = 6.13, p < .05$ ($\Delta R^2 = .06$). In the second regression, the covariates were entered in the first step and EA was entered in the second step. Consistent with our hypothesis, EA ($\beta = .40$) was incrementally associated with PTSD symptom severity above and beyond the influence of the covariates, $\Delta F(1, 57) = 14.59, p < .001$ ($\Delta R^2 = .12$).

Finally, a third regression analysis was conducted in which the covariates were entered in the first step and both AS and EA were entered simultaneously in the second step (Table 2). The full model was significant, $F(4, 56) = 15.73, p < .001$, accounting for 53% of the variance in PTSD symptom severity. Further, whereas EA continued to be a significant predictor, AS lost statistical significance with EA included in the model. To further examine the mediating role of EA, we used a nonparametric bootstrapping procedure (Preacher and Hayes 2004, 2008) to formally test the indirect effect of AS on PTSD symptom severity through EA (controlling for the effects of the covariates). Specifying 10,000 bootstrap resamples, we computed the point estimate of the indirect effect and a bias-corrected (BC) 95% confidence interval (CI). A significant indirect effect ($p < .05$) is indicated when the confidence interval does not contain zero. As hypothesized, AS had a significant indirect effect on PTSD symptom severity through its effect on EA, with a point estimate of .31 (BC 95% CI: .05 to .82).

Given the conceptual overlap between EA and the two trauma-specific avoidance symptoms of PTSD (Criterion C1 and C2), we conducted an additional set of analyses to examine how this overlap may have impacted our findings. We first examined bivariate correlations between EA and individual PTSD symptom clusters, finding that correlations with intrusion ($r = .56$), avoidance ($r = .44$), emotional numbing ($r = .56$), and hyperarousal ($r = .45$), were all statistically significant ($p < .001$). Not only were these correlations similar in magnitude, EA and trauma-specific avoidance actually demonstrated the weakest association. We next conducted the primary analyses again, excluding the two behavioral avoidance symptoms of PTSD from the dependent variable (CAPS total score).

The findings from these secondary regression and mediation analyses were generally unchanged. The basic regression model including only the covariates (gender and anxiety severity) accounted for significant variance in the modified CAPS total score, $F(2, 59) = 19.74, p < .001$ ($R^2 = .40$), and the addition of AS ($\beta = .37$) in the second step of the model accounted for a significant amount of variance above and beyond the covariates, $\Delta F(1, 58) = 7.01, p < .05$ ($\Delta R^2 = .07$). Similarly, in the second regression, the addition of EA ($\beta = .39$) in the second step of the model accounted for a significant amount of variance after controlling for the covariates, $\Delta F(1, 58) = 14.31, p < .001$ ($\Delta R^2 = .12$). The third regression analysis was also consistent with the original findings: when both AS and EA were entered simultaneously in the second step of the model, the full model accounted for a significant amount of variance in the modified CAPS score, $F(4, 57) = 16.55, p < .001$ ($R^2 = .54$), EA continued to be a significant predictor ($\beta = .33, p < .01$), and AS lost statistical significance

with EA in the model ($\beta = .21$; $p = .14$). A formal test of the indirect effect of AS based on 10,000 bootstrap resamples (described above) computed a point estimate of .26 (BC 95% CI: .03 to .67), indicating that EA mediated the association between AS and PTSD symptoms severity ($p < .05$), even after trauma-specific avoidance symptoms were excluded from the CAPS total score.

Finally, because cross-sectional data lead to ambiguity regarding the direction of mediation (Kraemer et al. 2002), we tested an alternative, reverse-mediation model with PTSD symptom severity serving as the mediator and EA serving as the dependent variable. Based on 10,000 bootstrap resamples, results indicated that AS had a significant indirect effect on EA through its effect on PTSD symptom severity. These findings were consistent regardless of whether the mediator was the full CAPS total score (point estimate = .17; BC 95% CI: .03 to .42) or the modified CAPS total score (point estimate = .18; BC 95% CI: .04 to .43). Thus, the directionality of the hypothesized mediational model was not supported.

Discussion

The current study examined AS and EA as potential factors associated with PTSD symptom severity among trauma-exposed crack/cocaine dependent patients in residential SUD treatment. Consistent with our hypotheses, AS and EA were both associated with PTSD symptom severity above and beyond the contributions of gender and non-specific anxiety symptoms. These findings are consistent with previous studies examining AS and EA in other populations exposed to potentially traumatic events (for reviews, see Elwood et al. 2009; Salters-Pedneault et al. 2004). The hypothesis that EA would mediate the relation between AS and PTSD symptom severity received only partial support, however. Although the hypothesized indirect effect was significant (even when the trauma-specific avoidance symptoms of PTSD were excluded from the PTSD outcome measure), an alternative model with PTSD symptom severity serving as the mediator and EA serving as the dependent variable was also supported, suggesting that mediation does not occur only in the hypothesized direction. These findings speak to the directional ambiguity of cross-sectional data and raise the possibility that PTSD and EA have a reciprocal relationship. That is, among trauma exposed substance abusers, general avoidance of emotional experiences may indeed be a mechanism through which fear of anxious arousal and PTSD are related; however, the presence of PTSD may also lead to greater emotional avoidance. Such a dynamic interplay would not be particularly surprising, given the central role that avoidance is hypothesized to play in the development, maintenance, and exacerbation of anxiety-related psychopathology over time (Foa et al. 2006).

Regardless of the directionality of these relationships, the tendency to engage in EA may have important implications for the treatment of individuals with SUD-PTSD. For example, although reducing trauma-related avoidance is a primary target of cognitive behavioral treatments for PTSD (Foa et al. 2007; Resick and Schnicke 1996), our results suggest that avoidance may extend to a fuller range of emotional experiences, consistent with the findings of Roemer et al. (2001). This tendency may have a negative impact on patient health, as studies have found that the purposeful suppression of emotional responses is associated with paradoxical increases in acute physiological arousal and emotional distress (e.g., Campbell-Sills et al. 2006; Gross and Levenson 1997), as well as lower long-term functioning and well-being (Gross and John 2003). Additionally, both AS and EA have previously demonstrated associations with negative SUD treatment outcomes. For example, AS was found to be prospectively associated with SUD residential treatment drop-out among heroin and crack/cocaine users (Lejuez et al. 2008). Similarly, avoidance of emotional distress during behavioral persistence tasks was associated retrospectively with self-reported duration of abstinence (Daughters et al. 2005) and prospectively with early

treatment drop-out (Daughters et al. 2005). Thus, interventions focused on decreasing AS and EA among trauma-exposed SUD patients may help mitigate the negative health- and treatment-related outcomes found among SUD patients with PTSD.

Although interesting, our findings must be considered in light of the limitations present. As previously noted, these correlational and cross-sectional data preclude identification of the precise nature or direction of the associations between these variables. In addition to the potential reciprocal relationship described above, it may be that AS and EA have an interactive effect on PTSD.¹ Longitudinal data are necessary to begin addressing these issues (e.g., Marshall et al. 2010). Second, these results must be interpreted with caution due to limited statistical power resulting from a small sample size. Third, whereas AS is an anxiety-specific vulnerability factor, EA is not specific to fearful or anxious responding. Thus, although EA theoretically captures avoidance of a variety of emotional experiences (including anxiety), a measure of anxiety-specific avoidance may prove to be a more powerful mediator of the association between AS and PTSD. Future research should test this possibility, as well as whether EA mediates the relation between other aversive emotions (e.g., guilt) and PTSD symptom severity. Further, given that data were collected from a primarily African American sample of crack/cocaine dependent patients in residential SUD treatment, findings may not be generalizable to individuals with co-occurring SUD and PTSD symptoms in general, or to those with specific SUDs (e.g., alcohol or heroin). For example, it was surprising that substance use severity was not correlated with PTSD symptom severity, a finding that may reflect factors specific to the current sample. Alternatively, this finding may be due to the differential reference periods of the assessment instruments. Whereas substance use severity was assessed retrospectively over the past year, PTSD symptoms were assessed retrospectively over the past month. It is possible that similar reference periods would have resulted in a stronger correlation between measures. Thus, it will be important for future studies to examine relations between AS, EA, and PTSD symptom severity in other SUD populations, and to assess both past and present self-reported PTSD symptoms.

In addition, one issue that may have influenced our assessment of EA is the degree to which participants were capable of attending to, identifying, and labeling emotions (i.e., emotional awareness and clarity). Recent theoretical and empirical work highlights the important role these processes play in effective emotion regulation (e.g., Gratz and Roemer 2004), and suggests that lack of emotional clarity may be greater among individuals reporting high rates of PTSD symptoms (Tull et al. 2007). Given that the reliable and valid assessment of EA will likely depend, in part, on an individual's awareness and clarity of emotional experiences (Tull et al. 2008), future research should attempt to account for this variability. The inclusion of behavioral measures of EA and related constructs (see Gratz et al. 2006) may have utility in addressing this limitation.

Future research is also needed to examine how AS and EA may contribute to a functional relationship between PTSD and SUD. In line with a self-medication model (Brady et al. 2004), previous research suggests that a worsening of trauma-related symptoms in individuals with SUD-PTSD may lead to an increase in substance cravings and usage (Back et al. 2006; Saladin et al. 2003). Further, there is growing evidence that substance use may function as a way for individuals with PTSD to modulate or avoid aversive emotions in general, not just those related to traumatic event exposure (Ouimette et al. 2007; Waldrop et al. 2007). Greater understanding of this functional relationship would likely be aided by further identification of the cognitive, emotional, and behavioral vulnerabilities associated

¹We examined this possibility but did not find a significant interaction effect for AS and EA after controlling for the covariates. However, due to limited statistical power, we cannot draw strong conclusions regarding the importance of this interaction.

with PTSD among individuals with SUD. For example, preliminary evidence suggests that several dimensions of emotion dysregulation (McDermott et al. 2009), including emotional nonacceptance (Gratz et al. 2007), may be useful targets for future research.

Despite limitations, these findings suggest that AS and EA are related to PTSD symptom severity among substance abusers. A deeper understanding of the nature, extent, and role of psychological factors in this population, as well as their impact on the course and effectiveness of SUD treatment, will facilitate improved assessment practices and the refinement of empirically-supported interventions. Consistent with the recent emergence of treatments that explicitly target experiential avoidance (e.g., Hayes et al. 1999), there may ultimately be utility in continuing to develop targeted interventions for SUD-PTSD that are aimed at promoting emotional acceptance, willingness, and mindfulness (e.g., Batten et al. 2009).

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Table 1

Measure means, standard deviations, and bivariate correlations

Measures	M (SD)	Bivariate Correlations			
		1	2	3	4
1. Depression anxiety stress scales-anxiety	11.71 (10.98)	–			
2. Anxiety sensitivity index	26.42 (16.95)	.72	–		
3. Emotional avoidance questionnaire	55.29 (20.03)	.47	.57	–	
4. Clinician-administered PTSD scale	43.79 (31.94)	.52	.53	.58	–

All correlations significant at $p < .001$

Hierarchical Regression Analysis of PTSD symptom severity as a function of anxiety sensitivity and emotional avoidance

Table 2

Predictor variables	B	β	p	sr^2	ΔR^2
Step 1					.39
Gender	−23.30	−.37	.001	.12	
Depression anxiety stress scales-anxiety	1.18	.41	<.001	.15	
Step 2					.14
Gender	−22.17	−.35	.001	.11	
Depression anxiety stress scales-anxiety	.34	.12	.387	.01	
Anxiety sensitivity index	.35	.18	.203	.01	
Emotional avoidance questionnaire	.55	.35	.003	.08	

sr^2 squared semi-partial correlation