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The Role of Anxiety Sensitivity and Difficulties in Emotion Regulation in Posttraumatic Stress Disorder among Crack/Cocaine Dependent Patients in Residential Substance Abuse Treatment

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Abstract

Current research suggests the importance of anxiety sensitivity (AS) in the risk for PTSD, and a growing body of research has demonstrated that difficulties in emotion regulation may also play a role. This study examined the unique relationships between AS dimensions, difficulties in emotion regulation, and a probable PTSD diagnosis among a sample of inner-city crack/cocaine dependent patients in residential substance abuse treatment. Probable PTSD participants exhibited higher levels of the AS dimension of social concerns and emotion regulation difficulties. Emotion regulation difficulties reliably distinguished probable PTSD participants from non-PTSD participants above and beyond both anxiety symptom severity and the AS dimension of social concerns. Further, social concerns did not account for unique variance when difficulties in emotion regulation was entered into the model. Results provide support for the central role of difficulties in emotion regulation relative to AS dimensions in the prediction of PTSD within a crack/cocaine dependent population.

Keywords

anxiety sensitivity; cocaine dependence; comorbidity; emotion regulation; posttraumatic stress disorder; substance use disorders

Posttraumatic stress disorder (PTSD) is characterized by the development and persistence of a variety of symptoms following direct or indirect (i.e., witnessing) exposure to a traumatic event in which an individual experiences fear, helplessness, and/or horror, as well as actual or threatened death or serious injury. The symptoms of PTSD are distributed across three separate symptom clusters described as re-experiencing, avoidance (which also includes symptoms of emotional numbing), and hyperarousal symptoms (American Psychiatric Association [APA],

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1994). According to the National Comorbidity Survey (NCS), the lifetime prevalence of PTSD in the general population is 8% (Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995). Further, studies have found that 20% to 50% of people will eventually develop symptoms consistent with a PTSD diagnosis following exposure to a potentially traumatic event (PTE; Alim et al., 2006; Zatzick et al., 2007). Findings that fewer than half the individuals exposed to a PTE go on to develop PTSD suggest that traumatic exposure is a necessary but not sufficient risk factor for PTSD. Consequently, there is a need to identify other factors that may increase the risk for PTSD following exposure to a PTE and account for this observed variability in the development of PTSD (see Brewin, Andrews, & Valentine, 2000).

Traditionally, research on PTSD vulnerability has focused on factors such as demographics, severity of the traumatic event, and previous traumatic exposure (Brewin et al., 2000), as well as peritraumatic responding (Shalev, Peri, Canetti, & Schreiber, 1996); however, emerging evidence suggests that cognitive and emotional individual difference factors may also be implicated in the risk for PTSD (Taylor, 2003; Tull, Barrett, McMillan & Roemer, 2007). One cognitive factor thought to play a particularly integral role in the development and maintenance of PTSD is anxiety sensitivity (AS; Taylor, 2003), defined as the tendency to fear anxiety-related symptoms due to beliefs that their occurrence will have negative somatic, cognitive, or social consequences (Reiss, 1991). AS has been found to be associated with increased fear responding (i.e., heightened amygdala reactivity) to emotionally-salient stimuli (Stein, Simmons, Feinstein, & Paulus, 2007). Consequently, following exposure to a PTE, a pre-existing tendency to fear anxiety-related symptoms and subsequent heightened reactivity to emotionally-salient stimuli may contribute to the extent to which an individual develops a conditioned response (i.e., avoidance) to anxious arousal stemming from that exposure. This fear and avoidance of anxiety-related symptoms may then prevent functional exposure to anxiety, as well as stimuli that activate this anxiety (e.g., memories or thoughts of the traumatic event, environmental cues), contributing to the development (and eventually the maintenance) of PTSD.

Providing support for the role of AS in PTSD, cross-sectional data have shown that PTSD is associated with the highest levels of AS compared to all other anxiety disorders (with the exception of panic disorder in which levels are comparable; see Cox, Borger, & Enns, 1999; Taylor, Koch, & McNally, 1992). In addition, Lang, Kennedy, and Stein (2002) found that women who developed PTSD following intimate partner violence reported higher levels of AS than women who did not develop PTSD as a result. Further, providing more direct evidence for its role as a risk factor for PTSD, studies have found that AS prospectively predicts PTSD symptomatology. For example, Feldner, Zvolensky, Schmidt, and Smith (2008) found that baseline AS uniquely predicted the severity of PTSD symptoms 12 to 24 months later in a large nonclinical sample of young adults. Fedoroff, Taylor, Asmundson, and Koch (2000) likewise found that AS predicted PTSD symptom severity following a motor vehicle accident, and reductions in AS due to cognitive-behavioral treatment for PTSD corresponded to reductions in PTSD symptom severity. Finally, among a sample of patients with a comorbid alcohol use disorder and PTSD, AS was found to be associated with the severity of re-experiencing, avoidance, and hyperarousal symptoms of PTSD, as well as to prospectively predict the severity of these symptoms one month later (Simpson, Jakupcak, & Luterek, 2006).

Although findings provide definite support for the role of AS in the development and maintenance of PTSD, AS is primarily a cognitive-based vulnerability (i.e., beliefs about the harmfulness of anxiety) and, thus, may not be sufficient to account for the emotion-related difficulties inherent in PTSD. The centrality of emotional dysfunction to PTSD suggests the additional relevance of emotion-based vulnerabilities to PTSD. In particular, theoretical and empirical literature suggests the key role of difficulties in emotion regulation in the etiology and maintenance of anxiety disorders in general (Amstadter, 2008; Barlow, Allen, & Choate,

2004) and PTSD in particular (Foa & Rothbaum, 1998; Frewen & Lanius, 2006). Specifically, in light of literature suggesting that PTSD is associated with heightened emotional intensity and reactivity (Orsillo, Batten, Plumb, Luterek, & Roessner, 2004; Tull, Jakupcak, McFadden, & Roemer, 2007), and that higher levels of emotional arousal are more difficult to modulate (see Mennin, 2005), deficits in the ability to adaptively respond to and modulate emotional experience may exacerbate post-traumatic emotional responses (e.g., fear), thereby contributing to the development and eventual maintenance of PTSD.

As defined here, emotion regulation refers to adaptive ways of responding to emotions (regardless of their intensity/reactivity). Specifically, Gratz and Roemer (2004) have identified several distinct but related domains involved in adaptive emotion regulation including: (a) awareness and understanding of emotions, (b) acceptance of emotions, (c) ability to control impulsive behaviors and engage in goal-directed behaviors when experiencing negative emotions, and (d) access to and flexible use of situationally appropriate emotion regulation strategies. Difficulties may appear in one or all of the above domains (also referred to as emotion dysregulation).

Based on this conceptualization of emotion regulation, Tull, Barrett et al. (2007) examined differences in emotion regulation difficulties among a sample of PTE-exposed individuals and found that those with PTSD symptoms at a severity level consistent with a diagnosis of PTSD (compared to those with subthreshold PTSD symptoms) exhibited significantly greater difficulties in overall emotion regulation, as well as the specific domains of lack of emotional acceptance, difficulties engaging in goal-directed behavior when distressed, difficulties controlling impulsive behavior when distressed, limited access to effective emotion regulation strategies, and lack of emotional clarity. However, when controlling for relevant covariates (e.g., negative affect), only differences pertaining to difficulties in controlling impulsive behavior when distressed, limited access to effective emotion regulation strategies, and lack of emotional clarity remained. Further speaking to the connection between emotion regulation difficulties and PTSD, Price, Monson, Callahan, and Rodriquez (2006) found that changes in emotion regulation among a sample of veterans (operationalized in a manner similar to Gratz and Roemer's [2004] conceptualization, and consisting of a focus on the understanding of emotions, the recognition that distress is temporary, and the recognition that distress provides important information) corresponded to changes in their PTSD symptoms following treatment for military-related PTSD. The above evidence suggests that both AS and difficulties in emotion regulation may contribute uniquely to the pathogenesis of PTSD. Yet, despite the growing body of research on the role of each of these characteristics in PTSD, no studies have examined their unique contributions to PTSD relative to one another.

One particularly relevant population in which to examine the unique roles of both AS and difficulties in emotion regulation in PTSD is treatment-seeking substance users – a population shown to be at high risk for PTSD and to exhibit heightened levels of emotion dysregulation (Fox, Axelrod, Paliwal, Sleeper, & Sinha, 2007; Fox, Hong, & Sinha, 2008) and AS (Lejuez, Paulson, Daughters, Bornovalova, & Zvolensky, 2006). For example, studies of treatment-seeking substance users have found lifetime prevalence rates of PTSD ranging from 36% to 50%, with the prevalence of current PTSD ranging from 25% to 42% (see Brady, Back, & Coffey, 2004; Jacobson, Southwick, & Kosten, 2001). Moreover, evidence suggests that crack/cocaine users are at particularly high risk for PTSD, compared to users of other substances (Cottler, Compton, Mager, Spitznagel, & Janca, 1992; Falck, Wang, Siegal, & Carlson, 2004; Najavits et al., 2003). Studies have also found evidence for heightened levels of emotion dysregulation (Fox et al., 2007) and AS (Lejuez et al., 2006) among cocaine-dependent inpatients in residential substance abuse treatment. Thus, treatment-seeking crack/cocaine users may be a particularly relevant population in which to examine the unique roles of AS and difficulties in emotion regulation in PTSD.

The goal of the current study was to provide preliminary data on the unique relationships between AS dimensions, difficulties in emotion regulation, and a probable PTSD diagnosis among a sample of inner-city crack/cocaine dependent patients in residential substance abuse treatment. It was hypothesized that participants with a probable PTSD diagnosis (vs. those without PTSD) would report higher levels of AS, and that dimensions of AS would predict probable PTSD status above and beyond relevant covariates. Further, it was hypothesized that emotion regulation difficulties would account for additional unique variance in probable PTSD status above and beyond AS dimensions (thus providing support for the relevance of both AS and difficulties in emotion regulation to PTSD).

Method

Participants

Participants were 58 crack/cocaine dependent patients consecutively admitted to a residential substance abuse treatment facility in Northeast Washington, D.C. Participants were predominantly male (n = 41, 70.7%), and ranged in age from 28 to 65 (M_{age} = 45.43, SD = 7.04). In terms of racial/ethnic background, the majority of participants self-identified as Black/African-American (n = 52, 89.7%). The remaining 6 participants self-identified as White (n = 5, 8.6%) or Other (n = 1, 1.7%). Most participants reported an annual income under $10,000 ($n$ = 37, 63.8%), as well as no higher than a high school education (n = 41, 70.7%).

All participants included in the study endorsed experiencing (directly or indirectly) at least one PTE at some point in their lifetime, as assessed by the Life Events Checklist (see *Measures*). On average, participants reported directly experiencing an average of 4.54 (SD = 2.93) PTEs, witnessing an average of 1.85 (SD = 1.89) PTEs, and learning about an average of 1.95 (SD = 3.31) PTEs (see Table 1 for data pertaining to the number and types of PTEs experienced by participants with and without a probable PTSD diagnosis).

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 extent to which patients are experiencing severe/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. Typical treatment lasts between 30 and 180 days, and aside from scheduled activities (e.g., group retreats, physician visits), residents are not permitted to leave the center grounds during treatment.

Measures

All patients entering the treatment facility were interviewed using the current substance use disorders module of the *Structured Clinical Interview for the Diagnostic and Statistical Manual of Mental Disorders, 4th edition* (DSM-IV; First, Spitzer, Gibbon, & Williams, 1996). Interviews were conducted by senior graduate students trained in the administration of the interview. Twenty-five percent of the interviews were reviewed by a PhD-level clinician (C. W. Lejuez). In cases for which a discrepancy was evident, areas of disagreement were discussed as a group and a consensus was reached. Only those participants who met criteria for current crack/cocaine dependence were approached for this study.

The *Life Events Checklist* (LEC; see Gray, Litz, Hsu, & Lombardo, 2004) provides a list of 17 PTEs and participants are asked to indicate whether: (a) the PTE happened to them, (b) the PTE was witnessed, (c) they learned about the PTE, (d) they are not sure about the PTE's

occurrence, or (e) they did not experience the PTE in any manner. The list of PTEs includes natural disaster, unexpected death of a loved one, assault with a weapon, sexual assault, and physical assault, among others. For each event endorsed, participants are asked the number of times the event occurred as well as their age at the time of the event. The LEC used in this study is commonly used in combination with the Clinician Administered PTSD Scale (CAPS-IV; Blake et al., 1995).

The *PTSD Checklist* (PCL; Weathers, Litz, Herman, Huska, & Keane, 1993) is a widely used 17-item self-report measure of the severity of re-experiencing, avoidance, emotional numbing, and hyperarousal symptoms experienced in response to their most stressful PTE. The items on the PCL correspond to the *Diagnostic and Statistical Manual of Mental Disorders, 4th edition* (DSM-IV; APA, 1994) criteria for a PTSD diagnosis. Using a 5-point Likert scale (1 = *not at all*, 5 = *extremely*), participants rate the extent to which each symptom has bothered them in the past month. The validity of the PCL has been demonstrated in both military and civilian populations (Blanchard, Jones-Alexander, Buckley, & Forneris, 1996; Weathers et al., 1993), as well as samples of substance users (Harrington & Newman, 2007). The PCL has been demonstrated to have strong test-retest reliability (r = .96), as well as moderate to strong correlations with other PTSD measures (Weathers et al., 1993). Further, the subscales of the PCL demonstrate high levels of agreement with the CAPS (Blake et al., 1990), a well-established and supported interview-based measure of PTSD (Grubaugh, Elhai, Cusack, Wells, & Frueh, 2007; Palmieri, Weathers, Difede, & King, 2007). For the present study, participants' responses to each item were summed to provide a total score representing overall PTSD symptom severity in order to establish the presence of a probable PTSD diagnosis (consistent with previous studies; e.g., Schumm, Hobfoll, & Keogh, 2004). Specifically, 25 participants (43.1%) scored 44 or above on the PCL, indicating that they met criteria for a probable PTSD diagnosis according to Blanchard and colleagues' cut-off score for civilians (Blanchard et al., 1996). This rate is consistent with what has been found in previous studies of substance users seeking treatment or in residential substance use treatment (e.g., Najavits et al., 1998; Harrington et al., 2007). Internal consistency within this sample for total score was excellent (α = .93).

The *Anxiety Sensitivity Index – Revised* (ASI-R; Taylor & Cox, 1998) is a 36-item self-report questionnaire designed to better assess the lower-order dimensions of AS. However, for the present study, only the items belonging to the Anxiety Sensitivity Index – 3 (ASI-3; Taylor et al., 2007) were utilized. The ASI-3 is an 18-item measure constructed from items contained within both the original Anxiety Sensitivity Index (ASI; Peterson & Reiss, 1993) and the ASI-R. In order to develop a measure of AS with a more replicable factor structure, Taylor et al. (2007) utilized only those items that unambiguously represent one of the following AS dimensions: (a) physical concerns, (b) cognitive concerns, or (c) social concerns. Factor analyses identified a three-factor structure (physical, cognitive, and social concerns) with each factor consisting of 6 items. This factor structure has been found to be stable across diverse clinical and non-clinical populations. In addition, the ASI-3 demonstrated better internal consistency and factorial validity than the original ASI, and evidence was found for the convergent, discriminant, and criterion validity of the ASI-3 (Taylor et al., 2007). Consequently, this three factor structure was utilized in the present study. Internal consistency of each factor in the current sample was excellent: physical concerns (α = .93), cognitive concerns (α = .91), and social concerns (α = .86).

The *Difficulties in Emotion Regulation Scale* (DERS; Gratz & Roemer, 2004) is a 36-item, self report measure that provides a comprehensive assessment of overall emotion regulation difficulties, as well as six specific dimensions: nonacceptance of negative emotions, difficulties engaging in goal-directed behaviors when experiencing negative emotions, difficulties controlling impulsive behaviors when experiencing negative emotions, limited access to

effective emotion regulation strategies, lack of emotional awareness, and lack of emotional clarity. Participants rate the extent to which each item applies to themselves using a 5-point Likert scale (1 = *almost never*, 5 = *almost always).* The DERS has been found to have adequate construct and predictive validity and good test-retest reliability over a period of 4 to 8 weeks (ρ_I = .88; Gratz & Roemer, 2004). Further, the DERS has been found to be strongly correlated with an experimental measure of emotion regulation within a clinical population (r = -.63; Gratz, Rosenthal, Tull, Lejuez, & Gunderson, 2006). Higher scores on the overall scale and each individual subscale are indicative of greater emotion regulation difficulties. Internal consistency in this sample was good for the overall scale (α = .91) and subscales (α's = .88, .82, .82, .79, .84, and .72, for the lack of emotional acceptance, difficulties engaging in goal-directed behavior when distressed, difficulties controlling impulsive behaviors when distressed, lack of emotional awareness, lack of access to effective emotion regulation strategies, and lack of emotional clarity subscales, respectively).

A self-report measure of substance use severity, modeled after the *Alcohol Use Disorders Identification Test* (AUDIT; Saunders, Aasland, Babor, De la Fuente, & Grant, 1993), was used to identify a potential covariate for primary analyses. On this measure, participants rate the frequency with which they used a variety of 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*). A total score representing frequency of past year substance use across all substances was calculated and examined as a potential covariate in analyses. As participants in this study were crack/cocaine dependent, all participants reported using crack/cocaine at least 2 to 3 times per week ($M_{crack/cocaine}$ = 4.73, SD = 0.53).

The *Depression Anxiety Stress Scales* (DASS; S.H. Lovibond & P.F. Lovibond, 1995) is a self-report questionnaire designed to differentiate between the core symptoms of depression, anxiety, and stress. The DASS has demonstrated adequate test-retest reliability (Brown, Chorpita, Korotitsch, & and Barlow, 1997), and there is extensive evidence for its construct and discriminant validity (Antony, Bieling, Cox, Enns, & Swinson, 1998; Brown et al., 1997; P.F. Lovibond & S.H. Lovibond, 1995; S.H. Lovibond & P.F. Lovibond, 1995). There are two versions of the DASS, a 21-item version and a 42-item version. These versions have been found to be consistent (Clara, Cox, & Enns, 2001) and comparable in their ability to distinguish between different diagnostic groups (Antony et al., 1998). The anxiety symptom severity subscale of the 21-item version of the DASS was used in this study as a potential covariate. Internal consistency was good (α = .79).

Finally, participants completed a questionnaire assessing basic demographic information (including age, gender, and racial/ethnic background), as well as the current use of prescribed psychotropic medications. Forty-two percent (n = 10) of probable PTSD participants and 21% (n = 6) of non-PTSD participants reported the current use of prescribed psychotropic medications.

Procedure

Procedures for this study were approved by the University of Maryland's Institutional Review Board. Only crack/cocaine dependent patients were recruited for this study. All participants were recruited no sooner than 72 hours after entering the treatment facility in order to minimize the potential impact of withdrawal symptoms on responding to the self-report measures. As part of a larger study (focused on the functional relationship between PTSD and crack/cocaine use), following the provision of written informed consent, participants completed a questionnaire packet including the measures described above. Participants were instructed that their involvement in the study was voluntary, their responses were completely confidential,

and participation would in no way influence their treatment. Participants were provided with monetary compensation (in the form of a $15 gift card to a local grocery store) upon leaving treatment.

Results

Preliminary Analyses

Analyses were first conducted to identify potential covariates for subsequent analyses (i.e., variables demonstrating a significant relationship with the variable of probable PTSD). No significant differences were found between probable PTSD and non-PTSD participants in age, (t [56] = -1.26, p = .21), gender (χ^2 [1] = 2.26, p = .13), racial/ethnic background (represented by a dichotomous variable of Black/African-American versus other racial/ethnic backgrounds, χ^2 [1] = .13, p= .72), income (χ^2 [8] = 7.67, p = .47), or education level (χ^2 [7] = 6.15, p = .52). In addition, participants with a probable PTSD diagnosis did not differ from non-PTSD participants in the number of PTEs experienced (t [56] = -1.52, p = .13), witnessed (t [56] = -1.11, p = .27), or learned about (t [56] = -0.26, p = .80). Probable PTSD and non-PTSD participants also did not differ in their current use of psychotropic medications (χ^2 [1] = 2.74, p = .10). Finally, participants did not differ on frequency of past year substance use (t [56] = 0.06, p = .96) as a function of probable PTSD status. However, as would be expected, participants with a probable PTSD diagnosis reported significantly more severe symptoms of anxiety ($M_{anxiety}$ = 16.00, SD = 9.15) than non-PTSD participants ($M_{anxiety}$ = 9.71, SD = 8.04), t (56) = -2.73, p = .008. Thus, anxiety symptom severity was included in subsequent analyses as a covariate.

Primary Analyses

Correlations between primary variables of interest are presented in Table 2 for both probable PTSD and non-PTSD participants. Univariate analyses of covariance (ANCOVAs) were conducted to examine between-group (probable PTSD vs. non-PTSD) differences in the primary variables of interest (i.e., AS dimensions and emotion regulation difficulties), controlling for anxiety symptom severity. With regard to AS, participants with a probable PTSD diagnosis were found to exhibit significantly greater levels of only social concerns. No significant between-group differences were found for physical concerns or cognitive concerns. In regard to difficulties in emotion regulation, participants with a probable PTSD diagnosis reported significantly greater difficulties in overall emotion regulation, as well as all specific domains of emotion regulation difficulties with the exception of lack of emotional awareness (see Table 3).

Next, a logistic regression analysis was conducted to determine the extent to which AS and difficulties in emotion regulation reliably predict probable PTSD status above and beyond anxiety symptom severity. Anxiety symptom severity was entered as a covariate in the first step of the model, followed by the AS dimension of social concerns (given that this was the only AS dimension that differed significantly between probable PTSD and non-PTSD participants) in the second step, and overall difficulties in emotion regulation in the final step. The model including only the covariate was significant, χ^2 (1) = 7.08, p = .008, accounting for 15% of the variance in probable PTSD status, and reliably distinguishing between probable PTSD and non-PTSD participants. This model correctly classified 48% of probable PTSD participants and 75.8% of non-PTSD participants (with an overall correct classification rate of 63.8%). The model including social concerns was found to be reliably different from the previous model, χ^2 (1) = 5.47, p = .02, accounting for an additional 11% of the variance in probable PTSD status. This model correctly classified 48% of probable PTSD participants and 78.8% of non-PTSD participants, with an overall correct prediction rate of 65.5%. Finally, inclusion of difficulties in emotion regulation in the final step of the model was found to

significantly improve the model, χ^2 (1) = 20.34, p = .001, and accounted for an additional 32% of the variance in PTSD status (with the overall model accounting for 58% of the variance in probable PTSD status, χ^2 [3] = 32.89, p = .001). Interestingly, however, social concerns did not remain significant in the final step of the model (p = .20), suggesting that this AS dimension does not account for unique variance in probable PTSD status above and beyond that associated with emotion regulation difficulties. This final model correctly classified 84% of participants with a probable PTSD diagnosis and 81.8% of non-PTSD participants (with an overall correct classification rate of 82.8%; see Table 4).[1]

Next, in order to examine the *unique* relationship between the particular AS dimension of social concerns and probable PTSD status (above and beyond its relationships with the other AS dimensions), we conducted another logistic regression analysis including all AS dimensions in the second step of the model. The model including all of the AS dimensions was not reliably different from the model with only the covariate, χ^2 (3) = 6.10, p = .11, although the AS dimensions together accounted for an additional 12% of the variance in probable PTSD status. Further, no individual AS dimension emerged as a reliable predictor of probable PTSD status in this model. However, inclusion of overall emotion regulation difficulties in the final step of the model once again reliably improved the model (above and beyond all AS dimensions), χ^2 (1) = 22.02, p = .001, accounting for an additional 34% of the variance in probable PTSD status (with the overall model accounting for 61% of the variance in probable PTSD status). This final model correctly classified 76% of participants with a probable PTSD diagnosis and 84.8% of non-PTSD participants (with an overall correct classification rate of 81%; see Table 5).

Discussion

The present study examined the unique roles of AS dimensions and difficulties in emotion regulation in the prediction of a probable PTSD diagnosis among a sample of crack/cocaine dependent patients in residential substance abuse treatment. Consistent with past research (Cox et al., 1999; Fedoroff et al., 2000; Feldner et al., 2008; Lang et al., 2002; Price et al., 2006; Simpson et al., 2006; Taylor et al., 1992; Tull, Barrett et al., 2007), results provide some support for the relevance of AS (specifically in the form of social concerns) and difficulties in emotion regulation to PTSD among crack/cocaine dependent patients.

With regard to AS, participants with a probable diagnosis of PTSD (compared to those without PTSD) reported significantly higher levels of social concerns. Further, this dimension of AS was found to reliably predict probable PTSD status above and beyond anxiety symptom severity. Despite providing some support for the role of AS in PTSD among this population, findings that the probable PTSD and non-PTSD groups differed only on the particular dimension of social concerns were unexpected. Specifically, given that PTSD includes symptoms of intrusive thoughts and hyperarousal, we expected that individuals with a probable

[1]Given that multiple factor structures for the ASI-R currently exist and to ensure that our findings were not simply due to our use of the ASI-3 factors, we reran analyses utilizing three different ASI-R factor solutions that are commonly presented in the AS literature: (a) the four factor solution (fear of respiratory symptoms, fear of publicly observable anxiety reactions, fear of cardiovascular symptoms, and fear of cognitive dyscontrol) identified by Taylor and Cox (1998); (b) the two-factor solution (fear of somatic sensations and social-cognitive concerns) identified by Zvolensky et al. (2003); and (c) the four factor solution (beliefs about the harmful consequences of somatic sensations, fear of publicly observable anxiety reactions, fear of cognitive dyscontrol, and fear of somatic sensations without explicit consequences) identified by Deacon, Abramowitz, Woods, and Tolin (2003). Regardless of the factor structure used, analyses remained the same. Specifically, probable PTSD and non-PTSD participants differed only on the Taylor and Cox (1998) and Deacon et al. (2003) fear of publicly observable anxiety reactions (ps < .05), consistent with the group differences in social concerns reported in the results section. No significant differences were found between probable PTSD and non-PTSD participants on the Zvolensky et al. (2003) AS dimensions (ps > .06). Further, in all analyses, emotion dysregulation significantly predicted probable PTSD status above and beyond the social AS dimension represented in the Taylor and Cox (1998), Deacon et al. (2003), and Zvolensky et al. (2003) factor structures (ps < .001), with the social AS dimension emerging as a non-significant predictor of probable PTSD status in each final model (ps > .20).

PTSD diagnosis would evidence heightened levels of fears of both cognitive concerns and physical concerns. Nonetheless, heightened levels of social concerns may increase the risk for the avoidance symptoms of PTSD, such as the avoidance of people or places that serve as reminders of a traumatic experience and feeling distant, detached, or cut-off from others. This vulnerability may also exacerbate the social anxiety and fear/distrust of others that is a common consequence of PTE exposure and PTSD (Fields, 2007; McDevitt-Murphy, Weathers, Adkins, & Daniels, 2005; Orsillo, Heimberg, Juster & Garrett, 1996). Yet, despite evidence that social concerns was the only AS dimension to distinguish between probable PTSD and non-PTSD crack/cocaine users, and predicted probable PTSD status above and beyond anxiety symptom severity, it did not remain a reliable predictor of probable PTSD status once emotion regulation difficulties were included in the model. Thus, findings suggest that, at least within this sample of inner-city crack/cocaine users, AS may play a less fundamental role in probable PTSD than emotion regulation difficulties.

Indeed, findings indicated that emotion regulation difficulties reliably distinguished crack/cocaine dependent patients with a probable PTSD diagnosis from those without PTSD, contributing to the prediction of probable PTSD status above and beyond both anxiety symptom severity and AS. Findings of heightened emotion regulation difficulties among probable PTSD participants (both overall and across the specific dimensions of nonacceptance of emotional responses, difficulties engaging in goal-directed behavior when distressed, difficulties controlling impulsive behaviors when distressed, limited access to effective emotion regulation strategies, and lack or emotional clarity) are consistent with findings obtained by Tull, Barrett, et al. (2007). In addition, the finding that emotion regulation difficulties reliably predicted probable PTSD above and beyond the variance associated with both anxiety symptom severity and AS dimensions highlights the unique role and relevance of emotion regulation difficulties to PTSD among crack/cocaine users. These findings are consistent with past theoretical and empirical literature indicating that crack/cocaine users with PTSD exhibit heightened emotion regulation difficulties (see Fox et al., 2007), as well as the suggestion that crack/cocaine use may serve a self-medication (or emotion regulating) function in the context of PTSD (Back, Brady, Jaanimagi, & Jackson, 2006).

Although interesting, the results of this study must be interpreted in light of its limitations. First and foremost, data were cross-sectional and correlational in nature, and, as a result, the exact nature and direction of the relationships between variables cannot be determined. Consequently, future studies should examine alternative models, including the potential moderating role of difficulties in emotion regulation in relationships between AS and clinical outcomes. For example, a recent study by Vujanovic, Zvolensky, and Bernstein (in press) found that the interaction of AS and difficulties in emotion regulation predicted several anxiety-related outcomes (including worry, catastrophic thinking about bodily events, and anxious arousal), above and beyond either variable alone and negative affectivity. Future research should continue to examine the likely complex interrelationships between temperamental vulnerabilities and emotion regulation difficulties. Further, longitudinal, prospective studies are needed to examine the precise nature of the relationships between AS, emotion regulation difficulties, and the development of PTSD following traumatic exposure.

Second, data on PTEs and PTSD symptoms were collected with self-report measures; thus, we could only establish a *probable* diagnosis of PTSD. However, it is important to note that the PCL demonstrates high levels of agreement with well-established, empirically-supported, interview-based measures of PTSD, such as the CAPS (Grubaugh et al., 2007; Palmieri et al., 2007). In addition, rates of probable PTSD found within this sample are consistent with rates previously reported for crack/cocaine dependent patients seeking treatment or in inpatient care and demonstrated through the use of interview-based measures of PTSD (Najavits et al., 1998; Harrington et al., 2007). Relatedly, although all participants reported the direct or indirect

experience of a PTE, it was not possible to determine whether or not these events were consistent with Criterion A for PTSD (i.e., involving the experience of fear, helplessness, and/or horror, as well as actual or threatened death or serious injury). Thus, it will be important for future studies to utilize clinical interviews to establish PTSD diagnoses.

The reliance on self-report measures of emotional responding may also introduce bias, as individuals with high levels of emotion dysregulation may have difficulties accurately reporting on their internal states (Tull, Bornovalova, Patterson, Hopko, & Lejuez, 2008). Likewise, individuals with high levels of AS may not have complete awareness or understanding of their emotions (Devine, Stewart, & Watt, 1999). Therefore, future studies may benefit from the use of non-self-report (e.g., behavioral, physiological) measures of emotionality (see Gratz et al., 2006).

An additional limitation is the absence of data on co-occurring psychopathology among participants. In particular, given that PTSD has been found to co-occur with a number of psychiatric disorders and related difficulties, including major depression, other anxiety disorders, personality disorders, and physical health problems (Kessler et al., 1995; Litz, Keane, Marx, & Monaco, 1992; Sareen et al., 2007; Southwick, Yehuda, & Giller, 1993), it is possible that findings of a relationship between emotion regulation difficulties and probable PTSD may not be unique to the presence of posttraumatic stress pathology *per se*, but due to the presence of greater and/or more severe co-occurring psychopathology among our probable PTSD participants. In order to establish a unique relationship between difficulties in emotion regulation and PTSD, it will be important for future studies to more closely examine the effects of co-occurring psychopathology and related difficulties on this relationship.

It also warrants mention that, although this study examined AS as a continuous variable, some researchers have found evidence that the latent structure of AS may be taxonic (suggesting that the examination of AS as continuously distributed may not provide the best representation of this variable; see Bernstein, Zvolensky, Feldner, Lewis, & Leen-Feldner, 2005; Bernstein et al., 2006, 2007). However, it is important to note that a recent large-scale study that utilized four mathematically-independent taxometric procedures to examine the latent structure of AS did not find support for a taxonic structure; on the contrary, these procedures provided consistent evidence for a dimensional conceptualization of AS (Broman-Fulks et al., 2008). Given the equivocal nature of these findings, future research is needed to determine whether AS taxonicity varies as a function of the population studied, the measure of AS used (to date, the latent structure of the ASI-3 has not been examined), or the type of taxometric procedure conducted (Broman-Fulks et al., 2008).

It also warrants mention that findings were obtained in a primarily male African-American inner-city sample of crack/cocaine dependent patients. As a result, findings from this unique sample may not be generalizable to other substance using populations. Although this focus on an underserved and understudied population may be considered an asset of this study, findings must be replicated across a more diverse group of substance users with a history of PTE exposure. In addition, given the relatively small number of women in this study, it was not possible to examine gender differences in the relationship between difficulties in emotion regulation and probable PTSD status. Given evidence of potential gender differences in emotion regulation (Barrett, Lane, Sechrest, & Schwartz, 2000; McRae, Ochsner, Mauss, Gabrieli, & Gross, 2008) and the presentation of PTSD (Olff, Langeland, Draijer, & Gersons, 2007), future studies should examine the moderating role of gender in the relationship between emotion regulation difficulties and probable PTSD. Finally, it is important to note that we did not control for the effect of possible withdrawal symptoms in the analyses. Although participants were not recruited into the study any sooner than 72 hours following entry into the treatment program, it is possible that some participants may have been experiencing lingering

withdrawal symptoms that could have influenced their ability to complete the questionnaires. Therefore, it will be important for future studies to directly assess the severity of withdrawal symptoms and examine the effect of this variable on outcomes.

Despite limitations, findings add to the literature on the role of AS in PTSD, as well as the growing body of research demonstrating the particular importance of difficulties in emotion regulation to PTSD. Future studies should build upon these findings by examining neurobiological indices of emotion regulation that may underlie PTSD (e.g., hypothalamic-pituitary-adrenal [HPA] axis dysregulation) within substance using populations, particularly those with crack/cocaine dependence. Indeed, a substantial body of literature provides evidence for the role of HPA axis dysfunction in both PTSD (e.g., see Yehuda, 2000) and crack/cocaine dependence (Fahlke, Hard, Thomasson, Engel, & Hansen, 1994; Sinha, Garcia, Paliwal, Kreek, Rounsaville, 2006).

Finally, findings of this study suggest the importance of developing novel interventions that focus on teaching crack/cocaine users with PTSD more adaptive ways of responding to their emotions. Treatments that focus specifically on the development of adaptive emotion regulation strategies for crack/cocaine dependent patients with PTSD may help reduce the risk for negative clinical outcomes observed within this population.

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Table 1

Rates of PTE Exposure for Probable PTSD and Non-PTSD Participants

	Non-PTSD (n = 33)			Probable PTSD (n = 25)		
Event	**Experienced**	**Witnessed**	**Learned**	**Experienced**	**Witnessed**	**Learned**
Natural Disaster	9.09%	12.12%	21.21%	8.00%	8.00%	36.00%
Fire or Explosion	18.18%	21.21%	15.15%	12.00%	44.00%	4.00%
Transportation Accident	51.51%	12.12%	12.12%	64.00%	16.00%	8.00%
Serious Accident	39.39%	9.09%	9.09%	36.00%	12.00%	16.00%
Exposure to Toxic Substance	9.09%	0.00%	24.24%	4.00%	8.00%	28.00%
Physical Assault	48.48%	9.09%	0.00%	56.00%	12.00%	4.00%
Assault with a Weapon	42.42%	6.06%	9.09%	60.00%	12.00%	4.00%
Sexual Assault	15.15%	0.00%	12.12%	12.00%	0.00%	16.00%
Other Unwanted Sexual Experience	21.21%	0.00%	6.06%	24.00%	4.00%	8.00%
Combat or Exposure to a War-Zone	6.06%	6.06%	15.15%	8.00%	4.00%	8.00%
Captivity	3.03%	3.03%	15.15%	12.00%	0.00%	16.00%
Life-Threatening Illness or Injury	24.24%	12.12%	3.03%	52.00%	12.00%	8.00%
Severe Human Suffering	9.09%	21.21%	9.09%	40.00%	20.00%	12.00%
Sudden, Violent Death	N/A	27.27%	12.12%	N/A	28.00%	20.00%
Sudden, Unexpected Death to Someone Close	57.58%	9.09%	3.03%	64.00%	16.00%	12.00%
Caused Serious Injury, Harm, or Death	12.12%	9.09%	6.06%	24.00%	8.00%	0.00%
Other Stressful Event or Experience	33.33%	3.03%	12.12%	44.00%	12.00%	8.00%

Table 2

Correlations between Primary Variables of Interest for Probable PTSD (n = 25) and Non-PTSD (n = 33) Participants

	1	2	3	4	5	6	7	8	9	10	11
1. Anxiety symptom severity	---	.28	.40*	.43*	.36*	.08	.36*	.33	.45*	-.08	.33
2. Physical concerns	.45**	---	.82**	.69**	.33	.43*	.41*	.07	.34	-.13	.29
3. Cognitive concerns	.41*	.76**	---	.80**	.45**	.54**	.41*	.15	.55**	-.09	.34
4. Social concerns	.31	.53**	.77**	---	.25	.31	.35*	.05	.42*	-.23	.24
5. Overall emotion dysregulation	.33	.11	.32	.39	---	.45**	.63**	.72**	.81**	.48**	.70**
6. Non-acceptance of emotional responses	.41*	.32	.25	-.03	.54**	---	.18	.21	.48**	-.13	.20
7. Difficulties engaging in goal-directed behavior	-.01	-.26	-.12	.17	.65**	.15	---	.67**	.53**	-.15	.30
8. Difficulties controlling impulsive behaviors	.18	-.10	-.02	.24	.79**	.25	.74**	---	.62**	.07	.15
9. Limited access to emotion regulation strategies	.45*	.19	.50*	.42*	.69**	.55**	.21	.37	---	.13	.41*
10. Lack of awareness of emotional experience	-.22	.06	.10	.09	-.02	-.42*	-.28	-.12	-.35	---	.57**
11. Lack of emotional clarity	.04	.10	.23	.36	.51**	-.16	.26	.30	.05	.64**	---

Note. Correlations for probable PTSD participants are listed in the bottom portion of the table and correlations for non-PTSD participants are listed in the upper portion of the table.

* $p < .05$.

** $p < .01$.

Table 3

Mean Differences between Probable PTSD and Non-PTSD Participants on Primary Variables of Interest Controlling for Anxiety Symptom Severity

	Non-PTSD (n = 33)		Probable PTSD (n = 25)		ANCOVAs	
Variable	**Mean**	**SD**	**Mean**	**SD**	**F (1,55)**	**η_p^2**
Physical concerns	9.70	9.15	16.00	8.04	0.51	.01
Cognitive concerns	5.58	5.11	11.20	8.02	2.55	.04
Social concerns	6.70	5.22	9.55	6.30	5.79*	.10
Overall emotion dysregulation	75.33	15.83	103.24	16.73	30.26***	.36
Non-acceptance of emotional responses	10.39	3.67	15.64	5.54	12.51**	.19
Difficulties engaging in goal-directed behavior	12.00	4.10	16.20	5.03	7.96*	.13
Difficulties controlling impulsive behaviors	12.03	4.57	16.76	5.00	8.51*	.13
Limited access to emotion regulation strategies	14.55	3.88	22.68	6.83	22.16***	.29
Lack of awareness of emotional experience	16.24	5.83	18.00	4.00	2.31	.04
Lack of emotional clarity	10.12	3.42	13.96	3.72	11.14**	.17

Note. Means presented in this table are non-adjusted means.

* $p < .05$.

** $p < .01$.

*** $p < .001$.

Table 4

Logistic Regression Analysis Examining the Role of Social Concerns and Emotion Dysregulation in Probable PTSD Status Controlling for Anxiety Symptom Severity

	B	Wald test	OR	95% CI
Step 1				
Anxiety symptom severity	.08	6.05*	1.09	1.02-1.16
Step 2				
Anxiety symptom severity	.05	1.91	1.05	0.98-1.13
Social concerns	.12	4.84*	1.13	1.01-1.26
Step 3				
Anxiety symptom severity	-.02	.19	.98	0.88-1.09
Social concerns	.09	1.61	1.10	0.95-1.27
Overall emotion dysregulation	.10	11.67***	1.11	1.05-1.18

Note. OR = Odds Ration; CI = Confidence Interval.

* $p < .05$.

** $p < .01$.

*** $p < .001$.

Table 5

Logistic Regression Analysis Examining the Role of Anxiety Sensitivity Dimensions and Emotion Dysregulation in Probable PTSD Status Controlling for Anxiety Symptom Severity

	B	Wald test	OR	95% CI
Step 1				
Anxiety symptom severity	.08	6.05*	1.09	1.02-1.16
Step 2				
Anxiety symptom severity	.06	2.11	1.06	0.98-1.14
Physical concerns	-.05	0.51	0.95	0.82-1.10
Cognitive concerns	.02	0.04	1.02	0.82-1.27
Social concerns	.15	2.64	1.16	0.97-1.38
Step 3				
Anxiety symptom severity	-.03	0.30	0.97	0.86-1.09
Physical concerns	-.01	0.02	0.99	0.83-1.18
Cognitive concerns	-.17	1.18	0.84	0.62-1.15
Social concerns	.25	3.25	1.29	0.98-1.69
Overall emotion dysregulation	.12	11.40**	1.13	1.05-1.21

Note. OR = Odds Ration; CI = Confidence Interval.

* $p < .05$;

** $p < .001$.