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A Preliminary Examination of the Relationships between Posttraumatic Stress Symptoms and Crack/Cocaine, Heroin, and Alcohol Dependence

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Abstract

High rates of co-occurrence between posttraumatic stress (PTS) and substance use disorders (SUDs) have led to the suggestion that substance use among individuals experiencing PTS symptoms might serve a self-medication function. However, research is still needed to provide a more comprehensive evaluation of the unique associations between PTS symptom clusters and substances (licit and illicit) with both anxiolytic/depressant and stimulant properties. Consequently, this study examined the relationship between severity of different PTS symptom clusters and heroin, crack/cocaine, and alcohol dependence among 48 treatment-seeking SUD patients with a history of traumatic exposure. No evidence was found for a relationship between PTS symptom clusters and crack/cocaine or alcohol dependence; however, results suggested a relationship between hyperarousal and avoidance (inversely-related) symptoms and heroin dependence. Results are discussed in terms of their implications for understanding motivations underlying the substance of choice among individuals with PTS symptoms, as well as the development of treatments for co-occurring PTS and SUDs.

Keywords

alcohol; cocaine; emotion regulation; heroin; PTSD; self-medication; substance use disorders

Substance use disorders (SUDs) have been found to be associated with numerous co-occurring difficulties, including mood and anxiety disorders (Conway, Compton, Stinson, & Grant, 2006; Grant et al., 2004; Kessler, Chiu, Demler, & Walters, 2005; Tull, Baruch, Duplinsky, & Lejuez, 2008), as well as high rates of traumatic exposure. Indeed, as many as 95% of individuals with SUDs report a history of traumatic exposure (e.g., Brown, Stout, & Mueller, 1999; Ford, Hawke, Alessi, Ledgerwood, & Petry, 2007), with rates of current diagnoses of posttraumatic stress disorder (PTSD) ranging from approximately 20% to 60% among samples of substance users in inpatient or outpatient treatment (Brady, 2001; Brady, Back, & Coffey,

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2004; Jacobson, Southwick, & Kosten, 2001). In addition, separate from PTSD, elevated rates of posttraumatic stress (PTS) symptoms have been found among substance users exposed to potentially traumatic events (PTEs; Harrington & Newman, 2007; Sullivan & Holt, 2008). Further, a particularly strong association has been found among PTSD, PTS symptoms, and illicit drug use, including both crack/cocaine and heroin use (Back et al., 2000; Clark, Masson, Delucchi, Hall, & Sees, 2001; Cottler, Compton, Mager, Spitznagel, & Janca, 1992; Sullivan & Holt, 2008).

High rates of co-occurrence between PTS and SUDs have led researchers to suggest that the experience of PTS symptoms and substance use may be functionally related (Stewart & Conrod, 2003). One prominent hypothesis concerning the functional relationship between PTS and SUDs focuses on the notion that substance use may operate as a form of self-medication among individuals experiencing symptoms of PTS following exposure to a PTE. Specifically, among PTE-exposed individuals, substance use may function to modulate or escape aversive PTS symptoms (see Brady et al., 2004; Chilcoat & Breslau, 1998; Khantzian, 1985; Stewart & Conrod, 2003). Extant data on the temporal relationship between PTS and SUDs provide support for the self-medication hypothesis. For example, Chilcoat and Breslau (1998) found that whereas a PTSD diagnosis greatly increased risk for subsequent development of a drug use disorder, a drug use disorder did not increase risk for traumatic exposure or PTSD. Likewise, Wasserman, Havassy, and Boles (1997) found that 69% of cocaine dependent inpatients with PTSD indicated that their PTSD developed prior to their cocaine dependence, consistent with findings that 86% of a sample of cocaine dependent individuals with PTSD reported that a worsening of their PTS symptoms was associated with an increase in cocaine use and 64% reported that an improvement in PTS symptoms was associated with a decrease in cocaine use (Back, Brady, Jaanimagi, & Jackson, 2006). In examining the longitudinal course of PTS symptoms and substance use among Vietnam veterans, Bremner, Southwick, Darnell, and Charney (1996) demonstrated that increases in both licit (i.e., alcohol) and illicit (i.e., heroin, marijuana, and cocaine) substance abuse paralleled a worsening of PTS symptoms. In addition, veterans reported that alcohol, heroin, benzodiazepines, and marijuana were useful in managing the hyperarousal symptoms of PTSD, and alcohol and heroin were also useful in reducing the severity of re-experiencing symptoms. Moreover, findings indicate that alcohol and cocaine dependent individuals with PTSD are more likely to report that their substance use is motivated by the experience of aversive internal states, compared to those without PTSD (Simpson, 2003; Waldrop, Back, Verduin, & Brady, 2007). Finally, experimental studies of the interplay between PTS symptoms and substance use have demonstrated that the severity of PTS symptoms predicts cravings in response to trauma-related imaginary cues among alcohol and cocaine dependent individuals with PTSD (Saladin et al., 2003).

With the exception of Bremner et al. (1996), a limitation of the studies discussed above is their focus on PTS symptoms in general, as opposed to an examination of the unique relationships between particular PTS symptom clusters (i.e., re-experiencing, avoidance, emotional numbing, and hyperarousal symptoms) and use of specific types of substances. Such studies may provide further support for the self-medication hypothesis by documenting a unique association between particular clusters of PTS symptoms and substances with properties that may counter or act in opposition to those symptoms. Indeed, preliminary research generally suggests a positive association between hyperarousal and re-experiencing PTS symptoms and use of substances that have a depressant or anxiolytic effect (e.g., alcohol, prescription anxiolytic medications; McFall, Mackay, & Donovan, 1993; Stewart, Conrod, Pihl, & Dongier, 1999), as well as positive associations between illicit drug use in general and re-experiencing, avoidance/emotional numbing, and hyperarousal PTS symptoms (McFall et al., 1992 Shipherd, Stafford, & Tanner, 2005; Sullivan & Holt, 2008). Further, although both Shipherd et al. (2005) and Sullivan and Holt (2008) did not find evidence for associations between particular PTS symptom clusters and alcohol use, Saladin, Brady, Dansky, and Kilpatrick (1995) found

higher levels of hyperarousal symptoms among inpatients with PTSD and alcohol dependence than among those with PTSD and cocaine dependence, suggesting that hyperarousal symptoms may be more strongly associated with substances that have a depressant, rather than a stimulant, effect. Likewise, Najavits et al. (2003) found that a restricted range of affect and feelings of detachment (both considered to be emotional numbing symptoms of PTS) were two of the most prominent PTS symptoms reported by patients with cocaine dependence. Despite preliminary evidence for an association between particular PTS symptom clusters and use of specific substances that could counter those symptoms, none of these studies has examined the unique associations between particular PTS symptom clusters and specific substances (both licit and illicit).

Thus, research is needed to provide a more comprehensive evaluation of the unique associations between particular PTS symptom clusters and the use of licit and illicit drugs with both anxiolytic/depressant properties (i.e., alcohol, heroin) and stimulant properties (i.e., crack/cocaine). To this end, the present study sought to examine the relationship between severity of specific PTS symptom clusters and heroin, crack/cocaine, and alcohol dependence among a sample of substance dependent patients in a residential substance abuse treatment center with a history of traumatic exposure in adulthood. Given evidence that crack/cocaine use has a stimulant effect, it was expected that emotional numbing symptom severity in particular would be associated with a dependence on crack/cocaine (consistent with Najavits et al. [2003]). Further, consistent with previous studies demonstrating an association between hyperarousal symptoms and the use of substances that have an anxiolytic/depressant effect (e.g., McFall et al., 1992; Stewart et al., 1999), it was expected that hyperarousal symptom severity would be uniquely associated with heroin and alcohol dependence.

Method

Participants

Participants were drawn from a larger group of patients in a residential substance use treatment facility in Northeast Washington, D.C., for 30 days of treatment. 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. Aside from scheduled activities (e.g., group retreats, physician visits), residents are not permitted to leave the center grounds during treatment.

Exclusion criteria for the larger study included current psychotic disorder and/or current mania. Of these participants, 48 endorsed the experience of a PTE in adulthood and, thus, were included in this study. These participants ranged in age from 18 to 55 (Mean age = 36.94, $SD = 11.55$), and the majority were male ($n = 39$, 81.3%). In regard to racial/ethnic background, 89.6% were Black/African-American, 4.2% were White, 2.1% were Asian/Asian-American, 2.1% were Latino, and 2.1% reported being from another racial/ethnic background. Participants were primarily low-income (52.1% reported an annual income of under $10,000) and the majority had at most a high school education (79.3%).

Measures

Current (past year) crack/cocaine, heroin, and alcohol dependence was assessed using the substance dependence modules of the *Mini International Neuropsychiatric Interview for the*

DSM-IV (MINI; Sheehan et al., 2002), a measure with established reliability and validity (Lecrubier et al., 1997; Sheehan et al., 1998). For the present study, interviews were conducted by senior graduate students and Ph.D. level clinicians trained in the administration of the MINI. Twenty-five percent of the interviews were reviewed by a Ph.D. level clinician (CWL). If a discrepancy was evident, areas of disagreement were discussed as a group until a consensus was reached. In addition to crack/cocaine, heroin, and alcohol dependence, participants were also interviewed to determine dependence on other substances, including stimulants (e.g., amphetamines), hallucinogens (e.g., PCP), inhalants, and marijuana. Number of substances on which participants were dependent was examined as a potential covariate for analyses. For analyses, crack/cocaine, heroin, and alcohol dependence were coded as separate dichotomous variables (0 = *no dependence* and 1 = *dependence*).

The *Traumatic Events Questionnaire* (TEQ; Vrana & Lauterbach, 1994) was designed to assess a variety of PTEs, including physical and sexual assault. For the present study, a modified version of the TEQ was used to identify a sample of participants who had been exposed to a violent PTE during adulthood (age 18+). Specifically, participants were asked the following items on the TEQ: 1) Have you been a victim of a violent crime, such as rape, robbery, or assault?; 2) As an adult, have you had any unwanted sexual experiences that involved threat or use of force?; 3) As an adult, have you ever been in a relationship in which you were abused either physically or otherwise?; 4) Have you witnessed someone who was mutilated, seriously injured, or violently killed?; and 5) Have you been in serious danger of losing your life or of being seriously injured? For each event endorsed, participants are asked the number of times the event occurred, their age at the time of the event, and (on a 7-point scale ranging from 1 [*not at all*] to 7 [*extremely*]) whether they were injured at the time of the event, whether their life was threatened, how traumatic the event was for them at the time, and how traumatic the event is for them presently. After completing all items, participants were instructed to identify the event that was the most traumatic and then to complete the *PTSD Checklist* (see below) based upon this event. In order to ensure that the index traumatic event from which participants' PTS symptoms stemmed occurred *prior to* their report of past-year substance use (consistent with the self-medication hypothesis), participants were included in the sample only if their index traumatic event occurred more than one year ago (prior to the time period in which their substance dependence was assessed). Given that no participants reported an index traumatic event within the 12 months prior to the start of the study (with the number of years since the event ranging from 1 to 37 years), none was excluded on the basis of this criterion. The TEQ has demonstrated validity within a civilian sample (Vrana & Lauterbach, 1994).

The *PTSD Checklist* (PCL; Weathers, Litz, Herman, Huska, & Keane, 1993) is a widely-used self-report measure including 17 statements corresponding to DSM-IV PTSD symptom clusters (American Psychiatric Association [APA], 1994). Referencing the index event identified on the TEQ, participants rated the extent to which they had been bothered by each symptom in the past month using a 5-point Likert scale (1 = *not at all*, 5 = *extremely*). The PCL provides an overall score, as well as subscale scores representing the different symptom clusters of PTSD. Given that researchers have suggested using the four theoretical PTSD symptom dimensions (i.e., re-experiencing, avoidance, hyperarousal, and emotional numbing) when examining the relationship between substance use and PTS symptoms (see Stewart, 1996; Stewart et al., 1999), we derived four separate subscale scores from the PCL representing these theoretical symptom dimensions of PTS. Higher scores on each subscale indicate greater symptom severity. The PCL has been found to have strong test-retest reliability, as well as moderate to strong correlations with other PTSD measures (Weathers et al., 1993). Further, subscales of the PCL demonstrate high levels of agreement with the Clinician Administered PTSD Scale (CAPS; Blake et al., 1990), an interview-based measure of PTSD (e.g., Palmieri, Weathers, Difede, & King, 2007). Internal consistency for each symptom cluster severity score

ranged from marginally adequate to good (α's range from .63 to .82). All PTS symptom clusters were significantly positively correlated with one another (*r*s range from .62. to .82, *p*s < .001).

In order to identify potential covariates, participants were administered a demographic form, and given the high rates of depression among individuals with PTSD and/or SUDs (Conway et al., 2006; Grant et al., 2004; Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995), participants were also administered the *Center for Epidemiological Studies – Depression Scale* (CES-D; Radloff, 1977), an established and well-supported measure of depression severity (see Nezu, Ronan, Meadows, & McClure, 2000). Internal consistency for the CES-D in this study was good (α =.87).

Procedure

Procedures were approved by the University of Maryland's Institutional Review Board. Data for this study were drawn from a larger study focused on understanding the effect of residential substance abuse treatment on risk-taking propensity (Aklin, Tull, Kahler, & Lejuez, 2009). The relationships examined in this study are separate from those presented in Aklin et al. (2009). Participants were approached in the last week of their treatment and asked if they would be interested in participating in a study on treatment, mood, and personality characteristics. Participants were told that involvement in the study was completely voluntary, and refusal to participate would not affect their treatment status. Participants who provided written informed consent completed a diagnostic interview and questionnaire packet including the measures described above. Participants were reimbursed $20 (provided upon discharge from the treatment center) in exchange for participation.

Results

Sample Characteristics

Of the 48 participants included in this study, 54.2% (*n* = 26) met criteria for crack/cocaine dependence, 25% (*n* = 12) for heroin dependence, and 37.5% (*n* = 18) for alcohol dependence (the distribution of participants across diagnoses is presented in Table 1). Participants reported experiencing an average of 2.31 (*SD* = 1.32) PTEs in adulthood. See Table 2 for data pertaining to the types of PTEs reported by participants, as well as the rates with which different PTEs were reported as index events (i.e., the event identified as most traumatic and the basis of participants' ratings of associated PTS symptoms). Speaking to the severity of the index events identified, participants reported that their index traumatic event was associated with moderate life threat (mean = 5.29, *SD* = 1.81) and was considered moderately-extremely traumatic at the time of its occurrence (mean = 5.54, *SD* = 1.77). Descriptive statistics (means for depression severity and PTS symptom clusters) for participants are provided in Table 3.

Preliminary Analyses

Analyses were conducted to identify potential covariates for subsequent analyses (i.e., any variable found to be significantly associated with any PTS symptom cluster or dependent variables). Racial/ethnic background, income level, and education level were not significantly associated with any PTS symptom cluster or crack/cocaine, heroin, or alcohol dependence, and gender was not significantly associated with any PTS symptom cluster or heroin or alcohol dependence (all *ps* > .05). However, gender was significantly associated with crack/cocaine dependence, with women having higher rates of crack/cocaine dependence than men (100% vs. 44%; χ^2 [1] = 9.37, *p* < .01). Although age was not significantly associated with any PTS symptom cluster or alcohol dependence (*p*s > .05), individuals who were dependent on crack/cocaine (vs. those who were not) were found to be significantly older (44.19 ± 7.95 vs. 28.36 ± 9.02, *t* [46] = 6.46, *p* < .001), as were individuals who were dependent on heroin (vs. those without heroin dependence; 45.08 ± 12.49 vs. 34.22 ± 10.00; *t* [46] = -3.06, *p* < .01). Depression

severity did not differ as a function of crack/cocaine, heroin, or alcohol dependence; however, it was significantly associated with the severity of reexperiencing ($r = .62$, $p < .001$), avoidance ($r = .67$, $p < .001$), emotional numbing ($r = .74$, $p < .001$), and hyperarousal ($r = .72$, $p < .001$) symptoms. Finally, number of substances on which participants were dependent, number of PTEs experienced, type of traumatic events experienced, average level of life threat accompanying the index traumatic event, and participants' average report of how traumatic the index event was at the time of its occurrence did not differ as a function of crack/cocaine, heroin, or alcohol dependence nor were these variables significantly associated with any of the PTS symptom clusters (ps > .05). Given these findings, age and depression severity were included as covariates for subsequent analyses involving crack/cocaine dependence;[1] depression severity and age were included as covariates for analyses involving heroin dependence; and depression severity was included as a covariate for analyses involving alcohol dependence. Finally, given that some participants were dependent on two or more of the substances under investigation, dependence on each substance was controlled for in analyses involving the other in order to establish a *unique* relationship between PTS symptom clusters and dependence on crack/cocaine, heroin, or alcohol (unless otherwise indicated below).

Primary Analyses

To examine the unique associations between PTS symptom clusters and crack/cocaine, heroin, and alcohol dependence, a series of hierarchical logistic regression analyses was conducted. The first logistic regression analysis included the categorical variable of crack/cocaine dependence as the dependent variable. Covariates of heroin dependence, alcohol dependence, depression severity, and age were initially entered in the first step of the model, followed by the four PTS symptom clusters in the second step; however, findings that the alcohol dependence variable was associated with a very high odds ratio ($OR = 16.77$) suggested that an inadequate number of observations within one or more of the cells associated with this variable (Table 1 indicates that several cells include less than 5 observations) may be introducing bias into our model (Greenland, Schwartzbaum, & Finkle, 2000). Thus, the decision was made to exclude alcohol dependence as a covariate in this model. Nonetheless, it is important to note that results presented below do not change if alcohol dependence is included as a covariate in the model. In analyses predicting crack/cocaine dependence, the model including the covariates was significant, χ^2 (3) = 33.93, $p < .001$, accounting for 68% of the variance in crack/cocaine dependence and reliably distinguishing crack/cocaine dependent participants from those who were not dependent on crack/cocaine. Contrary to hypotheses, however, inclusion of PTS symptom clusters did not significantly improve the model, χ^2 (4) = 4.02, $p > .05$, suggesting that PTS symptom severity is not uniquely associated with crack/cocaine dependence. However, the overall model, χ^2 (7) = 37.94, $p < .001$, reliably distinguished crack/cocaine dependent participants from participants who were not dependent on crack/cocaine, correctly classifying 88.5% of crack/cocaine dependent participants and 90.9% of participants without crack/cocaine dependence, with an overall correct classification rate of 89.6% (see Table 3).

To ensure that absence of findings of a significant relationship between PTS symptom clusters and crack/cocaine dependence was not due solely to relationships between heroin dependence and both PTS symptom clusters and crack/cocaine dependence, the logistic regression analysis predicting crack/cocaine dependence was rerun without the inclusion of heroin dependence as a covariate. Findings did not change, with inclusion of the PTS symptom clusters in the second

[1]Analyses examining the influence of gender on crack/cocaine dependence indicated that all women were dependent on crack/cocaine (compared to only 44% of the men). The presence of a zero cell value can lead to incorrect results within a logistic regression (see de Irala, Navajas, & Serrano del Castillo, 1997); consequently, despite its association with crack/cocaine dependence, we decided not to include it as a covariate in analyses involving crack/cocaine dependence as the dependent variable.

step failing to significantly improve the model, χ^2 (4) = 3.75, $p > .05$, and none of the PTS symptom clusters emerging as reliable predictors of crack/cocaine dependence.

In the logistic regression analysis utilizing heroin dependence as the dependent variable, covariates of crack/cocaine dependence, alcohol dependence, depression severity, and age were initially entered in the first step of the model, followed by the four PTS symptom clusters in the second step. However, as with the initial analyses involving crack/cocaine dependence, the alcohol dependence variable was again associated with a very high odds ratio (*OR* = 36.16) in the model. Thus, alcohol dependence was once again excluded from the analysis as a covariate (although it warrants mention that the results presented below do not change if alcohol dependence is included in the first step of the model as a covariate). The model including only the covariates was significant, χ^2 (3) = 15.59, $p < .01$, accounting for 41% of the variance in heroin dependence and reliably distinguishing between heroin dependent participants and participants without heroin dependence. Further, the model including the four PTS symptom clusters was reliably different from the model with only the covariates, χ^2 (4) = 12.10, $p < .05$, reliably distinguishing between heroin dependent participants and participants without heroin dependence and accounting for an additional 24% of the variance in heroin dependence. The final model correctly classified 66.7% of heroin dependent participants and 88.9% of participants without heroin dependence, with an overall correct prediction rate of 83.3%. Further, with regard to specific PTS symptom clusters uniquely associated with heroin dependence, findings indicated that severity of hyperarousal (positively associated) and avoidance (negatively associated) symptom clusters emerged as significant unique predictors (see Table 4). As with analyses focused on crack/cocaine dependence, results remained when the covariate of crack/cocaine dependence was excluded from the model. Specifically, inclusion of PTS symptom clusters in the second step significantly improved the model, χ^2 (4) = 16.51, $p < .01$, and severity of hyperarousal and avoidance symptoms remained significant predictors of heroin dependence.

Our final hierarchical logistic regression analysis involved alcohol dependence as the dependent variable, with crack/cocaine dependence, heroin dependence, and depression severity entered as covariates in the first step of the model and the four PTS symptom clusters included in the second step. The model including the covariates was not significant, χ^2 (3) = 5.03, $p > .05$, and, contrary to hypotheses, inclusion of the PTS symptom clusters did not significantly improve the model, χ^2 (4) = 9.19, $p > .05$, suggesting that PTS symptom severity is not uniquely associated with alcohol dependence within this sample of substance users (see Table 5). As with our analyses involving crack/cocaine and heroin dependence, findings did not change when crack/cocaine and heroin dependence were excluded from the model as covariates. Specifically, inclusion of the PTS symptom clusters in the second step failed to significantly improve the model, χ^2 (4) = 1.31, $p > .05$, and none of the PTS symptom clusters emerged as reliable predictors of alcohol dependence.[2]

Discussion

The goal of this study was to provide a preliminary examination of the unique relationships between particular PTS symptom clusters and heroin, crack/cocaine, and alcohol dependence

[2]Given emerging evidence that marijuana use may function to cope with emotional vulnerabilities relevant to PTS (e.g., anxiety sensitivity and emotion dysregulation; Bonn-Miller, Vujanovic, & Zvolensky, 2008; Zvolensky et al., 2009), we conducted an exploratory logistic regression analysis examining relationships between the severity of PTS symptom clusters and marijuana dependence. Twenty-one percent of our sample (n = 10) met criteria for current marijuana dependence. The model including the four PTS symptom clusters was not found to be reliably different from the model with only the identified relevant covariates (crack/cocaine dependence, heroin dependence, alcohol dependence, depression severity, age), χ^2 (4) = 0.88, $p > .05$, and no PTS symptom clusters were uniquely associated with marijuana dependence. Further, results remained the same when crack/cocaine, heroin, and alcohol dependence were removed from the model.

among substance users exposed to a PTE. Consistent with the self-medication hypothesis (as well as previous findings of relationships between the hyperarousal symptoms of PTSD and the use of drugs that have an anxiolytic effect; e.g., Bremner et al., 1996; Stewart et al., 1999), results demonstrated a unique association between severity of hyperarousal symptoms and a dependence on heroin. Given that heroin functions to dampen central nervous system activity, individuals with a history of PTE exposure and subsequent PTS symptoms may be motivated to use heroin as a way of alleviating or escaping hyperarousal symptoms in particular.

Interestingly, avoidance symptoms were also found to be related (albeit inversely) to heroin dependence. Although unexpected, these findings are consistent with those of Stewart et al. (1999), who found a small positive association between avoidance symptoms and prescription analgesic dependence. One possible explanation for these findings is that hyperarousal symptoms may become a conditioned stimulus for drug-seeking behavior due to past experiences in which heroin was successful in reducing those hyperarousal symptoms. Consequently, motivations to diminish hyperarousal symptoms and subsequent drug-seeking behavior may override the desire to avoid trauma-related cues, thus establishing an inverse relationship between avoidance symptoms and heroin dependence (consistent with animal models of drug-seeking behavior; e.g., Di Ciano & Everitt, 2003; Robinson & Berridge, 1993, 2002). Another possibility is that heroin use may decrease avoidance symptoms of PTSD. Specifically, as individuals increase their use of heroin, they may have less of a need for other strategies aimed at avoiding internal or external experiences associated with a traumatic event, resulting in a reduction in their reported PTS-related avoidance symptoms. Finally, given that heroin use has been found to be associated with social isolation (McNamee, Mirin, Kuehnle, & Meyer, 1976), it is also possible that the regular use of heroin may limit exposure to trauma-related cues, thereby reducing the opportunity and/or need for avoidance behaviors.

Conversely, no evidence was found for a specific relationship between any of the PTS symptom clusters and crack/cocaine or alcohol dependence. Although contrary to the hypothesized relationship between emotional numbing symptoms and crack/cocaine dependence (and the findings of Najavits et al., 2003), findings of an absence of a relationship between PTS symptoms and crack/cocaine dependence are consistent with past studies indicating stronger relationships between PTS symptoms and substances that have an anxiolytic/depressant effect than between PTS symptoms and cocaine use (e.g., Saladin et al., 1995). Likewise, whereas alcohol, heroin, marijuana, opiates, and benzodiazepines were identified as being helpful in reducing severity of PTS symptoms among Vietnam veterans, cocaine was not (Bremner et al., 1996). Given that cocaine use results in heightened central nervous system activity, it would be expected that individuals experiencing PTS symptoms such as hyperarousal may be motivated to avoid substances that would further increase arousal. Indeed, Bremner et al. (1996) found that cocaine use was associated with a worsening of PTS symptoms among Vietnam veterans. Yet, there is previous research demonstrating that cocaine dependent individuals report a functional relationship between their PTS symptoms and cocaine use, indicating that an increase in their cocaine use corresponds with a worsening of their PTS symptoms (Back et al., 2006). This suggests that crack/cocaine use may not be motivated by the experience of PTS symptoms *per se*, but instead by the desire to escape unpleasant emotions (e.g., shame, guilt, sadness) associated with the experience of PTS symptoms (see Waldrop et al., 2007). Of course, it is also possible that cocaine use among trauma-exposed individuals with PTS symptoms may be motivated by factors other than expectancies focused on the alleviation of distress. One factor worth investigating in future studies may be impulsivity, as separate lines of research demonstrate that both individuals with PTSD (Joseph, Dalgleish, Thrasher, & Yule, 1997; Kotler, Iancu, Efroni, & Amir, 2001) and cocaine dependent (vs. heroin dependent) individuals (Bornovalova, Daughters, Hernandez, Richards, & Lejuez, 2005) exhibit heightened levels of impulsivity.

Findings of an absence of a relationship between PTS symptom clusters and alcohol dependence are also contrary to findings of previous studies (Bremner et al., 1996; McFall et al., 1992; Saladin et al., 1995; Stewart et al., 1999). However, as noted previously, not all studies have found support for the self-medication function of alcohol dependence in relation to PTS symptoms (e.g., Shipherd et al., 2005; Sullivan & Holt, 2008; Ullman, Filipas, Townsend, & Starzynski, 2005), suggesting that further research is needed to better understand the conditions under which alcohol may function to self-medicate PTS symptoms. Laboratory-based studies examining the self-medication function of alcohol use in response to the experience of negative affect has found that the use of alcohol to cope with distress may depend on a number of factors. For example, Colder (2001) exposed a sample of college drinkers to a negative mood induction (aversive pictures) and examined associations between their physiological and self-reported emotional responses to the induction and the use of alcohol for coping reasons. Participants' self-reported emotional reactivity to the pictures was not found to be associated with alcohol coping motives. However, changes in physiological response (i.e., respiratory sinus arrhythmia and electrodermal responses) to the mood induction were associated with coping motives. In another study, Swendsen et al. (2000) found through experience sampling that self-medication through alcohol may be more likely to occur among men as compared to women. Consequently, future research on the self-medication function of alcohol among PTE-exposed individuals should examine gender differences in the use of alcohol to self-medicate PTS symptoms, as well as the role of physiological responding (as opposed to simply self-reported responding) to trauma-related cues in self-medication motives and/or alcohol craving. Of course, given that all participants with alcohol dependence in this study were also dependent on crack/cocaine and/or heroin, it is also possible that alcohol may serve less of a self-medication function in the context of illicit drug use.

Although promising, results must be considered in light of limitations present. First and foremost, the data were cross-sectional and correlational in nature. As such, it is impossible to determine the true temporal ordering of the relationships examined. Indeed, findings of a significant positive association between heroin dependence and hyperarousal symptoms may not be indicative of self-medication, but rather a “rebound effect” wherein substance use results in an increase in symptoms (Blume, Schmaling, & Marlatt, 2000). For example, Tomlinson, Tate, Anderson, McCarthy, and Brown (2006) found evidence that psychiatric symptoms generally worsen following a relapse to alcohol use. Likewise, it is important when examining the self-medication hypothesis to determine that participants' index traumatic event preceded their substance use. As mentioned previously, no participants reported that their index traumatic event (from which their PTS symptoms stemmed) occurred within the past year, ensuring that this traumatic event indeed preceded their past-year substance use (consistent with a self-medication hypothesis; Brady et al., 2004; Chilcoat & Breslau, 1998). Of course, the fact that this study examined participants' substance dependence following exposure to their index traumatic event does not preclude the possibility that participants were using those substances at similar levels prior to their index traumatic event as well, and that this substance use increased their risk for PTE exposure and the subsequent development of PTS symptoms (Cottler et al., 1992). However, even if participants were dependent on heroin prior to the development of their PTS symptoms, this does not necessarily mean that the presence of these symptoms did not increase their reliance on heroin even more, or that heroin was not used to manage the hyperarousal symptoms. That is, the function of heroin use may change following the experience of a PTE, such that heroin use originally motivated by positive reinforcement (e.g., “getting high”) may now be motivated by negative reinforcement (i.e., the alleviation of PTS symptoms). Nonetheless, prospective, longitudinal studies are needed to provide a more complete understanding of the nature of the relationship between traumatic exposure, PTS symptoms, and heroin dependence. In particular, these studies would benefit from examining the duration between traumatic exposure, the development of PTS symptoms, and the use of substances as a way of coping. In addition, it would be important for future studies to assess

for motivations underlying substance use in order to provide further support for the self-medication function of substance use in the context of certain PTS symptoms.

Another limitation concerns our sample, and the fact that participants were assessed in the last week of their 30-day stay at a residential treatment center. Although this likely limited the extent to which withdrawal symptoms may have affected participants' ability to accurately complete the self-report measures, participants' PTS symptoms may also have been less severe at the time of assessment than they were prior to their entry into the treatment facility. As a result, findings from this study may have underestimated the strength of the relationships between particular PTS symptom clusters and heroin, alcohol, and/or crack/cocaine dependence. However, the fact that participants in this study were receiving standard substance use treatment that did not focus directly on any difficulties stemming from traumatic exposure (see also Najavits, Sullivan, Schmitz, Weiss, & Lee, 2004) limits the extent to which this may be expected to have influenced the findings. Nonetheless, given evidence that the severity of PTS symptoms may naturally decline over the course of a period of abstinence (Coffey, Schumacher, Brady, & Cotton, 2007), future studies of the relationship between PTS symptoms and substance dependence should examine these relationships early in a period of abstinence or in the context of current substance use. In doing so, it is possible that other relationships between particular PTS symptom clusters and crack/cocaine, alcohol, and/or heroin dependence may emerge.

Moreover, participants' traumatic exposure and PTS symptoms were assessed through self-report measures. With regard to the latter, although the convergent validity of the PCL with well-established interview-based PTSD measures has been established (e.g., Palmieri et al., 2007), it is not possible to determine whether or not participants actually met criteria for PTSD. However, PCL scores within this sample were definitely elevated. The mean PCL score for this sample was 35.40, which is close to the suggested cut-off score for substance using populations (i.e., 38; Harrington & Newman, 2007). In addition, whereas effects may have been stronger among a sample of individuals diagnosed with PTSD through a clinical interview, there is evidence that even subthreshold PTS symptoms may be associated with levels of difficulty and distress comparable to full PTSD (Stein, Walker, Hazen, & Forde, 1997). Further, in regard to our assessment of participants' traumatic exposure, there was evidence that the index traumatic event identified by participants may have been sufficient to meet DSM-IV Criterion A for a PTSD diagnosis, given that (on average) participants reported that their index traumatic event was associated with moderate to extreme levels of life threat and distress at the time of occurrence. In addition, participants were asked to reference this index event in reporting on the severity of their PTS symptoms; consequently, participants' PTS symptoms were connected to the most traumatic event they identified.

Furthermore, in examining the self-medication model, we focused only on the outcome of substance dependence. It is possible that a greater number of significant associations may have emerged had we investigated a broader range of substance-relevant outcomes (e.g., frequency and duration of substance use). Future studies would benefit from examining a broader range of substance use outcomes. Finally, findings were obtained in a primarily African-American inner-city sample of substance users. As a result, findings may not be generalizable to other samples of substance users. 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 traumatic exposure.

Despite limitations, these preliminary findings provide evidence of a unique relationship between certain PTS symptoms and heroin dependence, consistent with the self-medication hypothesis of substance use and suggesting the need to further examine the relationship between PTS symptoms and heroin dependence. In addition, these findings suggest the need

to concurrently target both PTS symptoms and substance dependence in treatment. In fact, given high rates of comorbidity between anxiety disorders and substance use, researchers have suggested that co-occurring anxiety and SUDs may be better conceptualized as a single disorder in need of its own specialized treatments (Morissette, Tull, Gulliver, Kamholz, & Zimering, 2007; Tull et al., 2008), especially in regard to the co-occurrence of PTSD and SUDs (Brady, Dansky, Back, Foa, & Carroll, 2001; Coffey, Schumacher, Brimo, & Brady, 2005; Najavits et al., 2004). Combined treatments for PTSD and SUDs have been developed, such as *Seeking Safety* (Najavits, 2002; Najavits, Weiss, & Liese, 1996) and *Concurrent Treatment of PTSD and Cocaine Dependence* (CTPCD; Back, Dansky, Carroll, Foa, & Brady, 2001); however, given that individuals experiencing difficulties with PTSD symptoms may utilize substances as a way of escaping or alleviating distress, development of treatments that further emphasize the teaching of effective emotion regulation skills may also hold promise.

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Table 1
Distribution of Crack/Cocaine, Heroin, and Alcohol Dependence across Participants

	No Alcohol Dependence		Alcohol Dependence	
	No Heroin Dependence	Heroin Dependence	No Heroin Dependence	Heroin Dependence
No Cocaine Dependence	12	4	5	1
Cocaine Dependence	10	4	9	3

Table 2
Reported Rates of Exposure to Different PTEs and for Index Traumatic Events

	Overall Rate		Index Event Rate	
TEQ Item	**%**	**n**	**%**	**n**
Having been a victim of a violent crime (e.g., rape, robbery, assault).	79.2	38	54.2	26
Having been in serious danger of losing your life or being seriously injured.	64.6	31	35.4	17
Having witnessed someone who was mutilated, seriously injured, or violently killed.	39.6	19	4.2	2
Having been in a relationship where you were abused physically.	29.2	14	4.2	2
Having had unwanted sexual experiences that involved the threat or use of force.	18.8	9	2.1	1

Table 3

Logistic Regression Analysis Examining the Relationship between PTS Symptoms and Crack/Cocaine Dependence

	B	Wald test	OR	95% CI
Step 1				
Heroin Dependence	-3.42	5.00*	0.03	0.00-0.66
Depression Severity[a]	0.01	0.04	1.01	0.92-1.10
Age	0.27	13.57**	1.31	1.13-1.51
Step 2				
Heroin Dependence	-4.09	5.31*	0.02	0.00-0.54
Depression Severity	-0.01	0.01	0.99	0.88-1.13
Age	0.31	12.37**	1.37	1.15-1.63
Re-experiencing Symptoms[b]	0.35	1.93	1.42	0.87-2.32
Avoidance Symptoms[c]	-0.31	0.79	0.74	0.38-1.44
Emotional Numbing Symptoms[d]	-0.38	1.66	0.68	0.38-1.22
Hyperarousal Symptoms[e]	0.37	1.24	1.44	0.76-2.76

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

[a] Mean = 20.75, *SD* = 13.51;

[b] Mean = 10.54, *SD* = 3.95;

[c] Mean = 8.29, *SD* = 3.20;

[d] Mean = 6.31, *SD* = 3.03;

[e] Mean = 10.25, *SD* = 3.47.

* $p < .05$.

** $p < .01$.

Table 4

Logistic Regression Analysis Examining the Relationship between PTS Symptoms and Heroin Dependence

	B	Wald test	OR	95% CI
Step 1				
Crack/Cocaine Dependence	-3.16	4.45*	0.04	0.00-0.80
Depression Severity	0.01	0.13	1.01	0.95-1.07
Age	0.22	8.53**	1.24	1.07-1.43
Step 2				
Crack/Cocaine Dependence	-2.18	1.66	0.11	0.00-3.11
Depression Severity	0.02	0.06	1.02	0.89-1.16
Age	0.28	8.46**	1.32	1.10-1.60
Re-experiencing Symptoms	0.09	0.19	1.09	0.74-1.62
Avoidance Symptoms	-1.41	4.86*	0.24	0.07-0.86
Emotional Numbing Symptoms	0.71	2.74	2.03	0.88-4.69
Hyperarousal Symptoms	0.71	4.47*	2.04	1.05-3.95

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

* $p < .05$.

** $p < .01$.

Table 5

Logistic Regression Analysis Examining the Relationship between PTS Symptoms and Alcohol Dependence

	B	Wald test	OR	95% CI
Step 1				
Crack/Cocaine Dependence	0.93	2.08	2.53	0.72-8.96
Heroin Dependence	-0.27	0.13	0.77	0.19-3.18
Depression Severity	0.04	2.71	1.04	0.99-1.09
Step 2				
Crack/Cocaine Dependence	1.18	2.33	3.26	0.71-14.87
Heroin Dependence	0.71	0.61	2.03	0.35-11.97
Depression Severity	0.06	1.83	1.06	0.97-1.15
Re-experiencing Symptoms	0.22	2.04	1.25	0.92-1.68
Avoidance Symptoms	0.46	2.75	1.58	0.92-2.73
Emotional Numbing Symptoms	-0.22	1.00	0.80	0.52-1.23
Hyperarousal Symptoms	-0.47	3.30	0.63	0.38-1.04

Note. OR = Odds Ratio; CI = Confidence Interval. The overall model was significant, χ^2 (7) = 14.22, $p < .05$, and correctly classified 66.7% of alcohol dependent participants and 86.7% of participants without alcohol dependence.

* $p < .05$.

** $p < .01$.