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Distress Tolerance: Associations with Trauma and Substance Cue Reactivity in Low-Income, Inner-City Adults with Substance Use Disorders and Posttraumatic Stress

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Abstract

Cue reactivity has great potential to advance our understanding of posttraumatic stress disorder (PTSD), substance use disorder (SUD), and PTSD/SUD comorbidity The present investigation examined distress tolerance (DT) with regard to trauma and substance cue reactivity. Participants included 58 low-income, inner-city adults (49.1% women; *M*age = 45.73, *SD* = 10.00) with substance dependence and at least four symptoms of PTSD. A script-driven cue reactivity paradigm was utilized. Four DT measures were administered, including the Distress Tolerance Scale, Mirror-Tracing Persistence Task (MTPT), Breath-Holding Task (BH), and Paced Auditory Serial Addition Task (PASAT). Lower DT, as indexed by MTPT duration, was significantly predictive of greater levels of self-reported substance cravings/urges in response to trauma cues, above and beyond covariates. Lower DTS scores predicted lower levels of self-reported control/safety ratings in response to substance cues. None of the DT indices was significantly predictive of heart rate variability. Clinical and research implications are discussed.

Keywords

cue reactivity; distress tolerance; trauma; posttraumatic stress; substance use; experimental paradigm

Posttraumatic stress disorder (PTSD) and substance use disorders (SUD) are complex psychiatric conditions that commonly co-occur (e.g., McCauley, Killeen, Gros, Brady, & Back, 2012). Subclinical PTSD also is prevalent and marked by similar levels of impairment as diagnostic PTSD in SUD populations (e.g., Norman, Tate, Anderson, & Brown, 2007). Comorbidity of PTSD (i.e., hereafter referring to both subclinical and diagnostic PTSD) and

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SUD is difficult-to-treat and marked by a more costly and chronic clinical course when compared to either disorder alone (e.g., McCauley et al., 2012; Mills, Teesson, Ross, & Peters, 2006; Norman et al., 2007; Schafer & Najavits, 2007; Vujanovic, Bonn-Miller, & Petry, 2016). Thus, to improve or develop treatments, it is important to understand psychological processes with potential to inform theoretical models and interventions for PTSD/SUD.

Cue reactivity is one such process with great potential to advance our understanding of PTSD, SUD, and PTSD/SUD comorbidity. In individuals with PTSD and/or SUD, cue reactivity is a phenomenon in which emotional, behavioral, and/or physiological responses are evoked by internal (e.g., emotions, memories) and external (e.g., images, smells) trauma- and/or substance-related cues (e.g., Carter & Tiffany, 1999; Coffey et al., 2002; Drobes & Tiffany, 1997; King, Smith, McNamara, Matthews, & Fridberg, 2015; Rohsenow, Niaura, Childress, Abrams, & Monti, 1990). Trauma-related cue reactivity, specifically, has been shown to reliably distinguish between PTSD and non-PTSD populations (Pineles et al., 2013), predict development of PTSD in the aftermath of trauma (e.g., Gutner et al., 2010; Kleim, Wilhelm, Glucksman, & Ehlers, 2010), predict PTSD treatment outcome (e.g., Norrholm et al., 2016), and correlate positively with PTSD symptom severity (e.g., Foa, Rothbaum, Riggs, & Murdock, 1991; Rabellino, Densmore, Frewen, Theberge, & Lanius, 2016; Shin, Rauch, & Pitman, 2006). In terms of substance-related cues, research demonstrates that substance cues reliably increase craving among substance users (e.g., Thomas, Drobes, & Deas, 2005), and substance cue reactivity is a well-established relapse risk factor for almost every known substance of abuse (e.g., Carter & Tiffany, 1999; Kosten et al., 2006; See, Fuchs, Ledford, & McLaughlin, 2003; Sinha, 2011). Among individuals with PTSD/SUD, both trauma and substance cues can evoke elevated cravings and changes in physiological responding (e.g., salivation), compared to neutral cues (e.g., Coffey et al., 2002; Coffey et al., 2010; Coffey, Stasiewicz, Hughes, & Brimo, 2006; Nosen et al., 2012; Waldrop, Back, Verduin, & Brady, 2007). Overall, this literature suggests that desensitization to trauma cues via intervention, for example, can have implications for not only PTSD symptom severity but also substance use cravings and urges (e.g., Coffey et al., 2006).

Given the clinical relevance of cue reactivity, it is imperative to identify and understand cognitive-affective factors that are malleable via cognitive-behavioral intervention and have potential to influence trauma- and substance-related cue reactivity. Distress tolerance (DT), defined as the perceived or actual ability to tolerate negative or aversive emotional or physical states (Leyro, Zvolensky, & Bernstein, 2010), is one such promising factor with demonstrated relevance to (a) PTSD (e.g., Banducci, Connolly, Vujanovic, Alvarez, & Bonn-Miller, 2017; Vujanovic, Litz, & Farris, 2015; Vujanovic, Rathnayaka, Amador, & Schmitz, 2016), (b) SUD (e.g., Ali, Seitz-Brown, & Daughters, 2015; Bornovalova, Gratz, Daughters, Hunt, & Lejuez, 2012; Daughters, Lejuez, Bornovalova, et al., 2005; Daughters, Lejuez, Kahler, Strong, & Brown, 2005), and (c) PTSD/SUD comorbidity (e.g., Banducci, Bujarski, Bonn-Miller, Patel, & Connolly, 2016; Tull, Gratz, Coffey, Weiss, & McDermott, 2013). An emerging literature has documented that DT is robustly and negatively (inversely) associated with PTSD symptomatology across studies of trauma-exposed, community recruited adults, military veterans, psychiatric inpatients, and substance dependent adults

(Banducci et al., 2016; Marshall-Berenz, Vujanovic, Bonn-Miller, Bernstein, & Zvolensky, 2010; Vujanovic, Bakhshaie, Martin, Reddy, & Anestis, 2017; Vujanovic, Dutcher, & Berenz, 2017; Vujanovic et al., 2015; Vujanovic, Rathnayaka, et al., 2016). Similarly, DT has demonstrated significant associations with SUD severity, initiation, and treatment outcomes (Ali et al., 2015; Richards, Daughters, Bornovalova, Brown, & Lejuez, 2011). Furthermore, DT has been positively associated with duration of abstinence attempts (Daughters, Lejuez, Kahler, et al., 2005; Farris et al., 2016) and shown to predict residential SUD treatment retention (Daughters, Lejuez, Bornovalova, et al., 2005). In recent years, a DT-based intervention for SUD has shown promise in improving DT in adults in residential SUD treatment (Bornovalova et al., 2012), although this trial did not document changes in SUD-related outcomes (e.g., craving).

Limited, mostly cross-sectional work has been conducted to examine the mechanistic role of DT in PTSD/SUD populations. In trauma-exposed, substance using, community adults, DT has been shown to partially mediate the association between subclinical PTSD and both cannabis and alcohol coping motives (Potter, Vujanovic, Marshall-Berenz, Bernstein, & Bonn-Miller, 2011; Vujanovic, Marshall-Berenz, & Zvolensky, 2011). Other work among community adults has established an indirect effect of DT on alcohol consumption via the PTSD hyperarousal cluster (e.g., sleep disturbance, hypervigilance), such that trauma-exposed adults with low DT and PTSD hyperarousal symptoms may be particularly likely to consume alcohol (Duranceau, Fetzner, & Carleton, 2014). Furthermore, DT may impact SUD residential treatment outcomes among adults with PTSD, as males (relative to females) with a current diagnosis of PTSD (relative to no PTSD) who were lower in DT completed significantly fewer SUD treatment sessions (Tull et al., 2013).

DT models of PTSD/SUD (e.g., Vujanovic et al., 2015) posit that lower abilities to tolerate negative emotional or physical states may exacerbate both PTSD symptoms and substance use due to an intensified motivation to avoid (i.e., self-medicate) the negative emotional or physiological states (e.g., PTSD symptoms, cravings) one perceives or experiences as challenging to withstand. Individuals with SUD and elevated PTSD symptoms who also manifest low DT may experience increased reactivity to trauma and substance cues. For example, among individuals with PTSD/SUD, those low DT might demonstrate elevated reactivity, including substance cravings/urges and lower perceived emotional control or safety, in response to trauma cues due to inability to effectively withstand the emotional distress evoked by trauma reminders. Similarly, those with low DT may manifest increased substance cravings/urges and lower perceived emotional control or safety in response to substance cues due to ineffective (perceived or actual) coping strategies when confronted with cues evocative of cravings and urges. Across substance or trauma cue contexts, such reactivity also may take the form of changes in physiological reactivity. Clinically, this enhanced reactivity to cues may intensify PTSD symptomatology and amplify substance use due to efforts to escape or reduce negative affective states. Thus, increased reactivity among those with lower DT ultimately may interfere with abstinence as well as PTSD and/or SUD treatment efforts. Therefore, it is imperative to advance our understanding of DT relations with trauma- and substance cue reactivity in individuals with PTSD/SUD.

Several gaps have been noted in the extant literature. First, the vast majority of work exploring relations between DT and PTSD/SUD has utilized a cross-sectional methodology, limiting our ability to understand longitudinal, temporal associations among the documented relations (Vujanovic et al., 2015). Laboratory-based studies are a necessary next step to enhance experimental and temporal control over the variables of interest. Second, no studies to date have examined DT with regard to trauma- and substance cue reactivity. Thus, experimental cue reactivity paradigms offer the potential to shed light on the covariation between DT and reactivity to trauma and substance cues over a defined time period. Finally, most extant studies on the role of DT in PTSD/SUD have been conducted among subclinical populations of substance users exposed to potentially traumatic events, thereby limiting our inferences about clinical populations. This is unfortunate, as the available literature curtails our understanding of clinical samples with PTSD/SUD.

Therefore, the overarching aim of the current investigation was to examine associations between DT and trauma- and substance cue-related reactivity in adults with substance dependence and four or more symptoms of PTSD in a rigorous experimental design. Due to the multi-faceted nature of DT in terms of discrepancies between self-report and behavioral indices (Leyro et al., 2010), this study included four indices of DT, one self-report measure and three DT behavioral tasks. It was hypothesized that lower DT, measured via self-report and behavioral indices, would significantly predict trauma- and substance cue reactivity, as indexed by higher self-reported cravings/urges and lower self-reported ratings of control/safety to both trauma and substance cues. Lower DT was also expected to be related to lower high frequency (HF) heart rate variability (HRV; i.e., beat-to-beat variation in heart rate and an index of autonomic control of cardiac rhythm) in response to trauma and substance cues in this sample, as past research has found associations between resting low HRV and both PTSD (e.g., Chalmers, Quintana, Abbott, & Kemp, 2014; Rebellino et al., 2017) and SUD (e.g., Quintana, McGregor, Guastella, Malhi, & Kemp, 2013). However, few studies have examined changes in HRV following stressors or cues in PTSD/SUD samples; relevant studies suggest reductions in HRV can be expected (e.g., Green et al., 2016; Minassian et al., 2014; Ray, Pyne, & Gevirtz, 2017). All effects were considered after controlling for theoretically-relevant covariates including gender, substance use severity (i.e., number of SUD diagnoses), and PTSD symptom severity (e.g., Ali et al., 2015; Fox et al., 2006; Potenza et al., 2012; Richards et al., 2011; Simpson, Stappenbeck, Varra, Moore, & Kaysen, 2012; Sonne, Back, Diaz Zuniga, Randall, & Brady, 2003).

Method

Participants

The sample was comprised of 58 adults (49.1% women; *M*age = 45.73, *SD* = 10.00) with substance dependence per the *Diagnostic and Statistical Manual – 4th Edition* (DSM-IV; American Psychiatric Association, 2000) and at least four symptoms of PTSD per the *DSM-5* (American Psychiatric Association, 2013). Please see Table 1 for sample characteristics. Inclusionary criteria were comprised of: being 18-65 years old and proficient in English; meeting criteria for current (past month) substance dependence; reporting history of trauma exposure per *DSM-5* PTSD Criterion A; endorsing at least four current (past

month) *DSM-5* PTSD symptoms; and seeking treatment for substance dependence and trauma-related symptoms. Exclusionary criteria included: exclusive (only) nicotine dependence, alcohol or opioid dependence requiring detoxification, current or past bipolar I disorder or major psychotic disorder, active (past 6 months) psychotic spectrum symptoms, major unstable medical conditions, current (past month) suicidal or homicidal ideation with intent or plan, pregnancy, or inability to provide consent.

Measures

Structured Clinical Interview for DSM-IV Axis I Disorders—(SCID-I; First, Spitzer, Gibbon, & Williams, 1996). The SCID-I is a well-established structured diagnostic interview designed to assess major *DSM-IV* Axis I disorders. For purposes of the present study, the SCID-I was used to establish study inclusionary/exclusionary criteria, as noted above, and to derive the covariate for the number of SUD diagnoses (i.e., *DSM-IV* alcohol/substance abuse or dependence).

Clinician Administered PTSD Scale for DSM-5—(CAPS-5; Weathers et al., 2013a). The CAPS-5 is a well-established, 30-item structured interview for the assessment of PTSD, which assesses the 20 *DSM-5* PTSD symptoms, rated on a 5-point Likert-style scale (0 = *absent* to 4 = *extreme/incapacitating*), as well as the duration of symptoms, subjective distress, and relevant impairments in functioning. Administration requires identification of an index traumatic life event, assessed via the Life Events Checklist for DSM-5 (LEC-5; Weathers et al., 2013b). Overall ratings range from 0-80. Symptom ratings equal to or greater than '2' are considered symptom 'threshold' for diagnosis. In the current study, the past-month time-frame was used and internal consistency of the CAPS-5 was good (Cronbach's α = .89).

Distress Tolerance Scale—(DTS; Simons & Gaher, 2005). The DTS is a 15-item self-report measure that evaluates the extent to which respondents believe they can experience and withstand distressing emotional states (Simons & Gaher, 2005). Respondents rate their responses to each item (e.g., *"I can't handle feeling distressed or upset"*) on a 5-point Likert-style scale (1 = *strongly agree* to 5 = *strongly disagree*). The DTS – Total Score ranges from 15-75, with higher scores indicating greater DT. The DTS has demonstrated good psychometric properties (Simons & Gaher, 2005). For the current study, the DTS – total score was used as an index of perceived psychological DT, consistent with relevant past literature (Vujanovic et al., 2013). The internal consistency of the DTS in the current study was good (Cronbach's α = .88).

Mirror-Tracing Persistence Task—(MTPT; Quinn, Brandon, & Copeland, 1996). The MTPT is a behavioral index of psychological DT. The task requires participants to use a computer mouse to trace objects on the computer screen, as if viewing them through a mirror. When the mouse moves outside of the lines, a buzzer sounds loudly. Participants are encouraged to try their best and are told they can discontinue at any time. DT is measured as the length of time (number of seconds) that participants engage in the task. The MTPT has been used as a measure of DT in past work with trauma-exposed samples (Marshall-Berenz et al., 2010).

Breath-Holding Task—(BH; Hajek, Belcher, & Stapleton, 1987). The BH task is a behavioral index of physical DT. The task requires participants to hold their breath as long as possible. DT is measured as the length of time (average of two trials; in seconds) that participants are able to hold their breath (Daughters, Lejuez, Bornovalova, et al., 2005; Hajek et al., 1987). The BH task has been used as an index of physical DT in past work with both SUD populations and populations exposed to potentially traumatic events (e.g., Berenz, Vujanovic, Coffey, & Zvolensky, 2012; Rohsenow et al., 2015).

Paced Auditory Serial Addition Task-Computerized Version—(PASAT-C; Lejuez, Kahler, & Brown, 2003). The PASAT-C, originally developed to assess cognitive functioning after head injury (Gronwall, 1977), was modified and computerized for use as a behavioral index of psychological DT. This task requires participants, over three levels (increasing in difficulty), to add numbers by continually summing the two most recently presented digits. Incorrect or missed responses result in a loud buzzing sound, and the latency between number of presentations decreases as level of difficulty increases. On the third level, participants are given the option to self-terminate the task by clicking a button on the computer screen. DT is measured as the latency to discontinue the task; and lower DT is indexed by shorter latency to discontinue the task.

Addiction Severity Index-Lite—(ASI-Lite; Cacciola, Alterman, McLellan, Lin, & Lynch, 2007; McLellan et al., 1992). The ASI-Lite is a well-established, multi-dimensional, interview-based measure for SUD that assesses the respondent's lifetime and past-month status across seven domains (e.g., alcohol and drug use, employment/self-support). The ASI-Lite was used to obtain descriptive data, such as sociodemographic information and substance use history.

Visual Analog Scale—(VAS; Coffey et al., 2010). The VAS ratings were used to index subjective responses to trauma and substance scripts, including self-reported level of substance craving, substance use urge, desire to avoid consuming substances, control, and safety. Ratings were conducted on a 100-point scale ("not at all" to "very much so") displayed on a computer screen. Participants also rated the vividness of the scripts in creating mental images.

Physiological Measures—Heart rate reactivity was measured using raw electrocardiogram data collected with disposable Ag/AgCL electrodes places in a modified Lead II placement on the chest. Signals were amplified by a Biopac ECG100C amplifier with a 35Hz low pass notch filter and 0.05Hz high pass filter, and digitized by a Biopac MP150 at a channel sampling rate of 1.000kHz. R-waves were detected using standard parameters for human resting heart rate in Biopac AcqKnowledge software, using auto threshold detection with a noise rejection interval of 5% of the peak-to-peak range, and windowing of 40 to 120BPM. Trained research assistants edited the resulting inter-beat intervals for irregular beats using CardioEdit software (Brain-Body Center, University of Illinois at Chicago, Chicago IL). The HF-HRV was quantified from these inter-beat interval sequences using CardioBatch software (Brain-Body Center, University of Illinois at Chicago, Chicago IL) and the moving polynomial method (Porges, 1985; Porges & Bohrer,

1990), with standard adult HF-HRV settings: 2Hz sample rate, frequency window of 0.12-0.40Hz, and 30s epoch length.

Procedure

This investigation represents a secondary analysis of data from the parent study (Vujanovic, Smith, Green, Lane, & Schmitz, 2018), a randomized clinical trial comparing the efficacy of a novel combined cognitive-behavioral therapy for PTSD/SUD with cognitive-behavioral therapy for SUD alone (ClinicalTrials.gov Identifier: NCT02461732). The data for the current investigation are based upon the intake and baseline sessions of the parent study only. The study was approved by all relevant institutional review boards and conducted in accord with the Declaration of Helsinki.

Recruitment—Adults interested in treatment of SUD and trauma-related symptoms were recruited via community-based and online strategies (e.g., newspaper ads; Craigslist). Interested individuals called the treatment research clinic and were screened for general eligibility (e.g., age, substance use, English proficiency) via telephone; potentially eligible individuals were then scheduled for an intake appointment.

Intake and Baseline Sessions—At the general intake appointment, individuals first provided verbal and written consent to be screened for eligibility. The intake appointment consisted of the administration of the SCID-I, ASI, and medical screening. No interested participants were excluded for unstable medical conditions. Participants who met general eligibility criteria for the parent treatment study were then asked to provide verbal and written consent, if they expressed interest in participating. The baseline session of the study, scheduled immediately after the intake appointment, was comprised of administration of the LEC-5 and CAPS-5, self-report questionnaires, behavioral computer tasks (e.g., MTPT, BH, PASAT-C), and the experimental laboratory paradigm (described below).

Laboratory Procedures—Following CAPS-5 administration and prior to the experimental laboratory paradigm, participants were asked to generate three 50-second script cues. Script cues (trauma, substance, neutral scripts) were composed from the first-person perspective. For purposes of standardization, scripts were limited in duration to 50 seconds each, audio-recorded by a standardized voice, and played back to participants via headphones during the laboratory session described below. Script cues were selected for this paradigm to create individualized, emotionally salient trauma and substance cues and account for the diverse experiences of participants. Script cues are a standard laboratory paradigm to measure trauma and substance cue reactivity (e.g., Tull, Berghoff, Wheeless, Cohen, & Gratz, 2017).

Participants completed each script cue with the help of study staff, who ensured that participants' scripts were appropriately vivid, detailed, and within the 50-second time-frame. First, each participant completed a neutral script cue (i.e., "Please think back to a specific, recent that you experienced a neutral event and tell me about the situation in detail"); situations associated with negative affect or distress were not permitted. Second, each participant completed a brief substance script cue (i.e., "Please think back to a specific,

recent time that you used drugs and/or alcohol and tell me about the situation in detail"). Third, each participant was asked to compose a brief trauma script cue (i.e., "Please think back to the most upsetting traumatic event and tell about the situation in detail"), describing the index PTSD Criterion A traumatic event.

Following script composure, participants completed self-report questionnaires and computer tasks. Participants then were introduced to the sound-attenuated controlled experiment room and seated at a desk, facing a computer screen equipped with keyboard and mouse. The experimenter control room was located in the same hallway. Each participant was informed of the laboratory procedures, introduced to the computerized questionnaire form (i.e., VAS), and fitted with physiological monitoring equipment for heart rate. Participants were led through the entire laboratory session via standardized, audio-recorded directions.

For pre-cue baseline monitoring, participants were instructed to sit quietly for 5 minutes. Physiological measures were recorded throughout the experimental session, with average HF-HRV calculated over each minute. Self-reported VAS ratings were gathered at one-minute intervals during this baseline. The cue reactivity paradigm was comprised of the presentation of three script cues (neutral, trauma, substance). The order of script presentation was randomized and counterbalanced across participants to minimize carryover effects. After each script cue presentation, participants were instructed to immediately provide VAS ratings. Then, the experimenter instructed the participant to sit quietly for 10 minutes to allow a return to pre-cue baseline physiological levels. The next script cue was then administered and the process repeated until all three script cues were presented.

Data Analytic Plan

All data analyses were conducted using SPSS version 24. First, in order to reduce the number of variables and assess the underlying dimensions of the six VAS ratings coded after each script, two exploratory factor analyses (EFA) using the principal components analysis (PCA) extraction method and promax rotation were conducted for these variables (separately for trauma and substance reactivity). For each EFA, the Bartlett's test of sphericity and the Kaiser-Meyer-Olkin (KMO) measure of sampling adequacy were conducted to evaluate the appropriateness of using EFA (Tabachnick & Fidell, 2007; Williams, Onsman, & Brown, 2010). Kaiser's criteria (eigenvalue > 1) and scree plot (Cattell, 1966) were used for decisions regarding the number of components to be retained. Variables with loadings above 0.6 were preserved for a given component. The extracted components for each condition were conceptually interpreted. Thereafter, the sum scores of retained items on each component were calculated. Finally, a residualized difference score was computed by regressing each of the aforementioned sum scores on the baseline levels of these scores (Zumbo, 1999). These residualized scores were used as outcome variables in the following regression models and represent a "pure" difference. score after variance associated with the baseline score is removed. Internal reliability of these 2-item variables was examined and expressed as the Spearman-Brown reliability estimate (Eisinga, Te Grotenhuis, & Pelzer, 2013). Please see Table 2. Physiological reactivity to the cues was also examined for possible data reduction, and as relatively quick returns to baseline were observed after cues, analyses were restricted to the first minute of HF-HRV collected

following each cue. Notably, analyses were also conducted with two additional outcomes, including (a) HF-HRV during the 50-second cue and (b) 5 HF-HRV during 5-minutes post-cue, and the pattern of results remained consistent.

Second, manipulation checks were conducted to ascertain that the trauma and substance cues elicited significant distress compared to the neutral cues. Difference scores were computed by subtracting participants' scores in response to trauma scripts versus neutral scripts and substance scripts versus neutral scripts for each of the outcome variables derived from EFA, respectively. Two similar difference scores were also calculated by subtracting the minute-one HF-HRV following (a) trauma script versus neutral script and (b) substance script versus neutral script. Separate one sample t-tests were conducted to examine the significant difference of these scores from zero, to provide evidence for the effectiveness of trauma and substance cues compared to the neutral cue.

Third, descriptive statistics and bivariate correlations were calculated for all study variables. Fourth, a series of hierarchical linear regression analyses was conducted with regard to each of the outcome variables (see Table 4). Three covariates were included at step one of each regression model: gender, number of SUD diagnoses, and PTSD symptom severity. It was decided *a priori* that outcome variables that did not demonstrate significant bivariate correlations with any predictors would not be included in regression analyses. The four DT measures (DTS, BH, MTPT, PASAT) were entered simultaneously at step two of the models. Please note that analyses also were conducted using alternative substance-related covariates, including frequency of past-month substance use, and the pattern of results remained consistent. Prior to analysis, all data were examined for univariate and multivariate outliers and normality. The level of missing data was acceptable at less than 10% (Peng, Harwell, Liou, & Ehman, 2006). The Little's MCAR test indicated patterns of missing data completely at random (Little, 1988). To account for missing data, multiple imputation with 25 imputations was conducted (Allison, 2000; Sterne et al., 2009). The False Discovery Rate (FDR) method was used to control for family-wise error rate (Benjamini & Hochberg, 1995). The FDR adjusted probabilities were calculated on all hypothesized associations using the Benjamini-Hochberg procedure for multiple testing correction (Reiner, Yekutieli, & Benjamini, 2003). Note that analyses were also conducted with only the subsample of participants (70.6%, $n = 41$) who met full diagnostic criteria for PTSD, and the pattern of results remained consistent.

Results

Exploratory Factor Analyses

Bartlett's tests of sphericity were significant for both trauma and substance cue reactivity models ($p < .05$). The Kaiser-Meyer-Olkin (KMO) measure of sampling adequacy was also higher than the expected cut off of 0.7. Based on tests of scree plots and size of eigenvalues, two components were retained in both the trauma and substance cue reactivity models. The VAS item "avoid consuming substance" was omitted from both models as it showed communalities of less than 0.2. The range of communalities for all other scales varied from 0.4 to 0.6 in the trauma cue reactivity model, and from 0.3 to 0.7 in the substance cue reactivity model. The rotated component matrices for both are shown in Table 2.

In both models, VAS ratings of substance use cravings and urges showed loadings of greater than 0.6 on component 1, and VAS ratings of control and safety showed loadings of greater than 0.6 on component 2. Based on these findings and in line with extant work (e.g., Suri & Vaidya, 2015; Ziaee, Fadardi, Cox, & Yazdi, 2016), component 1 was interpreted as self-reported substance cravings/urges to the corresponding cue (i.e. either the trauma or substance cues), and component 2 was interpreted as self-reported control/safety to the corresponding cues. In the next step, a score for each component was computed through summing the strongly loaded items of each component. These scores were used as outcome variables in analyses. The Spearman-Brown reliability estimate for these variables ranged from acceptable to excellent (α's = .70 to .91).

Manipulation Checks: One Sample T-Tests

Participants rated script vividness highly across trauma (M = 94.07, SD = 10.75), substance (M = 93.55, SD = 12.22), and neutral (M = 90.09, SD = 19.20) scripts; and no between-group differences in terms of script vividness ratings were noted (p's > .05). One sample t-tests (i.e., manipulation checks) demonstrated the four subjective indices of cue reactivity derived from EFA to be significantly different from zero (Mean difference = 45.6, SD = 50.5, p < .001; Mean difference = 39.5, SD = 46.6, p < .001; Mean difference = 27.8, SD = 51.2, p < .001; and Mean difference = 11.4, SD = 18.7, p = .01 for 'cravings/urges in response to trauma cues,' 'control/safety in response to trauma cues,' 'cravings/urges in response to substance cues,' and 'control/safety in response to substance cues', respectively). The difference scores for minute-one HF-HRV following (a) trauma scripts minus neutral scripts and (b) substance scripts minus neutral scripts were not significantly different from zero.

Descriptive Statistics and Bivariate Correlations

Please see Table 3 for a summary of descriptive statistics and bivariate correlations among all study variables. MTPT scores were significantly, negatively correlated with cravings/urges in response to trauma cues. DTS scores were significantly, positively correlated with control/safety in response to substance cues. None of the indices of HRV was significantly related to any of the other study variables.

Regression Analyses for Trauma Cue Reactivity

Please see Table 4. In terms of self-reported cravings/urges to use substances in response to trauma cues, the model accounted for 31% of variance, F(7, 50) = 6.14, p < .001. At step one, PTSD symptom severity was significantly predictive of the outcome variable. At step two of the model, MTPT performance significantly predicted cravings/urges to use substances; lower MTPT scores predicted higher levels of cravings/urges (Table 4). With regard to control/safety in response to trauma cues, the model accounted for 19% of variance, F(7, 50) = 3.11, p =.01. PTSD symptom severity significantly predicted self-reported control/safety ratings. At step two, none of the indices of DT was significantly predictive of the outcome variable.

Regression Analyses for Substance Cue Reactivity

Please see Table 4. The model accounted for 31% of variance in self-reported cravings/urges to use substances in response to substance cues, $F(7, 50) = 5.71$, $p < .001$. At step one, PTSD symptom severity significantly predicted the outcome variable. At step two of the model, none of the indices of DT was significantly predictive of cravings/urges. With regard to self-reported ratings of control/safety in response to substance cues, the model accounted for 38% of variance in, $F(7, 50) = 6.32$, $p < .001$. None of the covariates emerged as a significant correlate. DTS, but neither of the other DT measures, was significantly predictive of control/safety ratings.

Discussion

This investigation tested the hypothesis that lower levels of DT, measured via self-report and behavioral indices, would predict greater reactivity to trauma and substance cues. Reactivity to trauma and substance cues was defined by higher self-reported cravings/urges to use substances, lower self-reported control/safety ratings, and lower HF-HRV. Covariates included gender, substance use severity (i.e., number of SUD diagnoses), and PTSD symptom severity. Hypotheses were partially supported by results.

First, lower DT, as indexed by MTPT duration, was significantly predictive of greater levels of self-reported cravings/urges to use substances in response to trauma cues, above and beyond covariates. Individuals with lower ability to withstand negative emotional states may therefore be more likely to respond to trauma cues with substance cravings and urges (i.e., self-medication). Overall, this finding supports negative reinforcement models of substance use, in that individuals with elevated PTSD symptomatology and low DT may be especially likely to use substances as a means to avoid or escape from the distress elicited by trauma cues. However, no other DT indices were associated with substance use cravings/urges, even at the bivariate level. These findings run contrast to the body of literature documenting relations between the DTS and PTSD symptom severity (Vujanovic et al., 2015), suggesting that DT is differentially related to PTSD symptomatology versus reactivity to trauma cues. More replication and extension of this work is necessary.

Second, lower DTS scores predicted lower levels of self-reported control/safety ratings in response to substance cues. For individuals with SUD and elevated PTSD symptomatology, the perceived ability to withstand negative emotional states may influence perceptions of one’s ability to maintain control and safety upon being exposed to substance cues. This finding has potentially important implications for intervention, since improving beliefs relevant to one’s DT may lead to improvements in adaptive responding to substance cues. Notably, no other DT indices were significantly related to substance cue reactivity at the bivariate level. This may suggest that higher *perceived* DT is most relevant to feelings of control and safety following substance cues, but further replication with more generalizable samples, such as PTSD/SUD populations with more varied socioeconomic status and racial/ethnic composition, is necessary.

Third, neither BH nor PASAT-C durations were significantly associated with any type of cue reactivity in this study. In light of these findings, it is noteworthy to consider that BH

duration in this sample (M = 62.19, SD = 33.20) was significantly higher than that found in individuals in SUD residential treatment (M = 30.12, SD = 13.77; Daughters, Lejuez, Bornovalova, et al., 2005) and general trauma-exposed, psychiatric inpatients (M = 38.84, SD = 21.97; Vujanovic, Dutcher, et al., 2017). This might suggest that (a) this sample manifested significantly higher rates of physical discomfort tolerance than other comparable samples; or (b) the methodology employed for recording BH duration in this sample did not produce adequate monitoring and standardization. For example, nose clips were not employed in this study during the BH task, and therefore, it is possible that participants took breaths prior to discontinuing the task and without notice by research staff. Future work in this domain should take extra precautions to ensure the standardization of this procedure, since physical discomfort tolerance is highly relevant to substance use maintenance due to the often physiologically uncomfortable nature of cravings and withdrawal symptoms (Goldstein & Volkow, 2002; Koob & Le Moal, 1997; West & Gossop, 1994). In contrast, the PASAT-C duration in this sample (M = 123.67, SD = 175.51) was significantly lower than that of individuals in residential SUD treatment (M = 208.71, SD = 165.22; Daughters, Lejuez, Bornovalova, et al., 2005) or community-recruited individuals who use substances (M = 367.2, SD = 118.2; Gorka, Ali, & Daughters, 2012). That psychological DT of this low-income, inner-city, predominantly African American sample was especially low may be due to chronic stress resulting from lack of access to resources (e.g., Joseph, Matthews, & Myers, 2014), greater exposure to neighborhood stress and violence (e.g., Evans & English, 2002), and societal discrimination (Rodriguez-Seijas, Stohl, Hasin, & Eaton, 2015). Additional work in this domain of inquiry is imperative, since both DT and cue reactivity manifest within broad-based sociocultural contexts that impact etiological and maintenance processes as well as intervention efficacy.

Notably, HF-HRV was not significantly associated with any study variables at the bivariate level. This is inconsistent with past literature documenting associations of lower HF-HRV and both PTSD and SUD (e.g., Chalmers et al., 2014; Green et al., 2016; Minassian et al., 2014; Quintana, et al., 2013; Ray et al., 2017; Rebellino et al., 2017). However, no published studies to date have examined cue-elicited HRV in PTSD/SUD samples. Without additional physiological data, we are unable to draw broad-based inferences about these findings. Replication of this work is necessary with multiple physiological indices.

A few additional considerations are worthy of note. Since manipulation checks indicated that the trauma cues produced larger effects on self-reported reactivity than the substance cues, it is possible that this difference in strength of manipulation impacted the results. Furthermore, the correlation between self-reported cravings/urges in response to trauma cues and cravings/urges in response to substance cues was moderately high (r = .64). The low levels of discrimination between trauma and substance cues in eliciting substance cravings/urges is noteworthy. This may be reflective of the SUD severity of the sample, in that participants tended to respond with higher levels of cravings/urges regardless of type of cue.

Although not the primary foci of the present study, several other findings are worthy of note. First, DTS was significantly correlated with BH duration (r = .39), despite the fact that most studies of DT do not find convergence between behavioral and self-report measures of the construct (Anestis, Bender, Selby, Ribeiro, & Joiner, 2011; Marshall-Berenz et al., 2010;

McHugh et al., 2011). The moderate magnitude of these associations may lend further support to their validity in measuring a higher order DT construct. Second, PTSD symptom severity was significantly, negatively correlated with DTS ($r = -.35$) and MTPT duration ($r = -.33$), but not with BH or PASAT-C duration. This attests to the distinct aspects of DT indexed by various measures (Leyro et al., 2010) and underscores the importance of advancing our understanding of how these facets translate to cognitive-behavioral treatment targets. Third, PTSD symptom severity was significantly associated with cravings/urges in response to trauma cues ($r = .48$), control/safety ratings in response to trauma cues ($r = -.59$), and cravings/urges in response to substance cues ($r = .52$). However, substance use severity at baseline was not related to any cue reactivity variables.

Several study limitations are worthy of note. First, the sample size was relatively small ($N = 58$). The sample size of this study may have limited the statistical power to identify small effects, and future research should replicate and extend this work with larger samples. Given the concerns regarding the clinical significance of small effects in social sciences (Light, Singer, & Willett, 1990; Stevens, 2002), the medium to large effect sizes documented in this study may lend greater confidence to the findings and attenuate concerns regarding generalizability (Brooks & Barcikowski, 2012). Second, the sample was comprised of predominantly African American, low-income, inner-city adults seeking treatment for SUD and trauma symptoms. In addition, the majority of participants (63%) met criteria for cocaine dependence. The underrepresented, clinically severe nature of the sample is certainly a strength of this study, but it also limits generalizability to less severe substance-using populations of greater socioeconomic advantage. Replication and extension of this work with less severe samples is therefore pertinent. Third, treatment-seeking participants with diverse types of SUD were recruited for this study, and most participants met criteria for multiple SUD ($M = 1.74$, $SD = .87$). It will be important for future work to examine relations between DT and substance cue reactivity among non-treatment seeking samples and among individuals using specific substance classes to evaluate whether DT is differentially related to cue reactivity across different types of populations or substances. Fourth, the study did not employ a standard negative affect or craving measure in assessing self-report cue reactivity. Rather, a series of one-item VAS measures was employed, as consistent with past work (e.g., Coffey et al., 2010). Future studies might consider employing standardized measures pre- and post-cue presentation. Fifth, the study was underpowered to test moderation, and yet, a natural extension of this work is to explore the moderating role of DT in the association between cue exposure type (trauma vs. substance) and reactivity. For example, future work might explore whether DT moderates the association between trauma cue presentation and cravings/urges to use substances, such that individuals with low DT might be most likely to report cravings/urges to use in the presence of trauma cues due to ineffective strategies to modulate negative emotions triggered by the cues. Finally, this study utilized only one index of physiological reactivity (i.e., HF-HRV). It is important for future work to include various measures of physiological reactivity (e.g., skin conductance, respiration) to better understand relations between DT and physiological responsivity to trauma and substance cues.

Overall, this study was the first to evaluate associations between DT and trauma- and substance cue reactivity. The clinical sample was comprised of a socioeconomically

disadvantaged population with SUD and elevated PTSD symptomatology. Results indicate that lower levels of behaviorally indexed DT were associated with greater substance cravings/urges in response to trauma cues and lower levels of perceived DT were associated with lower self-reporting control/safety ratings in response to substance cues. This preliminary work suggests that interventions targeting DT may be useful in improving trauma and substance cue reactivity in individuals with PTSD/SUD, thus potentially improving treatment outcomes.

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Table 1

Participant Characteristics

Variable	Mean(SD) or % (*n*)
Race/Ethnicity[1]	
Black/African American	77.7% (45)
White	17.2% (10)
Hispanic	5.2% (3)
Marital Status[1]	
Single/Never Married	43.3% (26)
Divorced	21.6% (13)
Married	13.3% (8)
Separated	13.3% (8)
Widowed	6.6% (4)
Socioeconomic Status[1]	
Less Than High School	15.5% (9)
High School/GED	32.5% (19)
Some College	34.5% (20)
College Degree	13.8% (8)
Not reported	3.4%(2)
Number of days with paid work (past 30 days) [1]	6.3 (*SD* = 9.6)
Monthly income (past 30 days) [1]	$378.4 (*SD* = $736.9)
Trauma Event Exposures[2]	
Number of Traumatic Event Exposures Types	8.3(4.3)
Assault with a weapon	82.7%(48)
Physical assault	79.3%(46)
Natural disaster	77.5%(45)
Transportation accident	75.8%(44)
Sexual assault	72.4%(42)
Toxic substance exposure	67.2%(39)
Life threatening illness or injury	60.3%(35)
Fire or explosion	60.3%(35)
Human suffering	60.3%(35)
Serious accident	55.1%(32)
Witnessed sudden violent death	51.7%(30)
Sudden accidental death	51.7%(30)
Other unwanted sexual experiences	51.7%(30)
Other stressful event or experience	51.7%(30)
Captivity	37.9%(22)
Causing serious injury to someone else	36.2%(21)
Combat war-zone exposure	36.2%(21)
Axis I Diagnoses (DSM-IV)[3]	

Variable	Mean(SD) or % (*n*)
Mean Number of Diagnoses	2.97 (*SD* = 1.20)
Mean Number of Substance abuse/dependence	1.74 (*SD* = .87)
Cocaine Dependence	62.0%(36)
PTSD[4]	70.6%(41)
Alcohol Abuse/Dependence	41.3%(24)
Cannabis Dependence	32.7%(19)
Major Depressive Disorder	22.4%(13)
Substance Induced Mood Disorder	8.6%(5)
Sedative Dependence	5.1%(3)
Stimulus Dependence	5.1%(3)
Opioid Dependence	5.1%(3)
Panic Disorder	5.1%(3)
Hallucinogen/PCP Dependence	1.7%(1)
Other substance Dependence	1.7%(1)
Obsessive Compulsive Disorder	1.7%(1)
Bing Eating Disorder	1.7%(1)
Substance Use[1]	
Tobacco Use, Past Month (yes/no)	53.4% (31)
Cannabis Use, Past Month (yes/no)	44.8% (26)
Alcohol Use Days, Past Month	10.2 (10.5)
Drug Use, Past Month (yes/no)	67.2% (39)
Drug Use Problem Days, Past Month	16.6 (12.1)
Positive Urine Toxicology	70.6%(41)
Primary Substance[5]	
Stimulants	60.3% (35)
Cannabis	20.6% (12)
Alcohol	15.5% (9)
PCP	3.4% (2)

Note.

[1]Data derived from Addiction Severity Index;

[2]Data derived from Life Events Checklist;

[3]Data derived from Structured Clinical Interview for DSM-IV Axis I Disorder;

[4]Data derived from the Clinician Administered PTSD Scale for DSM-5.

[5]Data derived from the Brief Substance Craving Scale.

Table 2

Rotated Component Matrices

VAS[1] Items	Component (trauma vs. neutral script)[2]		Component (substance vs. neutral script)[2]		
	1	2	1	2	
	2.6	1.1	2.3	1.0	Initial Eigenvalues
Craving	0.92		0.95		
Substance Use Urge	0.93		0.95		
Control		0.88		0.79	
Safety		0.89		0.86	

Note.

[1]Variables refer to the Visual Analogue Scale (VAS) ratings (0-100 scale), provided immediately following each script cue exposure.

[2]Components represent residualized difference scores when ratings to neutral script cues were subtracted from trauma or substance script cue ratings. Residualized scores represent the difference after variance associated with the baseline score is removed. The VAS item, "avoid consuming substance," was omitted from both models as it showed communalities of less than 0.2.

Table 3

Descriptive Statistics and Bivariate Correlations between Study Variables (n=58).[£]

Variable	1	2	3	4	5	6	7	8	9	10	11	12	13
1. Gender (female)[^a1]	1												
2. Number SUD[a2]	−.18	1											
3. PTSD symptom severity[a3]	.32*	−.07	1										
4. DTS[b4]	−.15	.13	−.35*	1									
5. BH Duration[b5]	−.36	.12	−.04	.39**	1								
6. MT Persistence[b6]	−.17	.05	−.33*	.01	.23	1							
7. PASAT Persistence[b7]	−.12	.05	−.05	−.16	−.12	.34	1						
8. Cravings/Urges-Trauma Cue[c8]	.08	−.18	.37*	−.05	−.02	−.45*	−.07	1					
9. Control/Safety-Trauma Cue[c9]	−.23	.04	−.59*	.04	.06	.15	.17	−.52***	1				
10. Cravings/Urges-Substance Cue[c10]	.20	−.15	.34*	−.08	.08	−.04	−.02	.64***	−.47**	1			
11. Control/Safety-Substance Cue[c11]	.13	.04	−.05	.39*	.07	−.02	.01	.02	.52***	−.37*	1		
12. HRV-Trauma Cue[c12]	−.07	−.09	−.001	−.04	.02	.14	.02	−.03	−.12	−.01	−.14	1	
13. HRV-Substance Cue[c13]	−.04	−.18	.11	−.19	−.02	.09	.07	−.04	−.07	.16	.08	.48**	1
M(SD) or %	49.1%	1.7 (.84)	33.81 (15.01)	37.92 (11.77)	62.19 (33.20)	87.42 (73.06)	123.67 (175.51)	45.54[d] (50.10)	−39.53 (46.62)[d]	28.71 (51.28)[d]	−11.4 (18.72)[d]	−.04 (1.16)[d]	.01 (.71)[d]
Observed Range	0-1	1-6	3-61	15-58	15.13-167.01	.07-304.80	2-600	−31-169[d]	−169-29[d]	−61-183[d]	−98-63[d]	−3.17-4.14[d]	−3.27-1.28[d]

** $p < .01$,

* $p < .05$; *Note.*

[a] Covariate;

[b] Predictor;

[c] Outcome Variable.

[^] Point-biserial correlation/categorical variable.

[a1] Gender (0 = male; 1 = female);

[a2] Number of Substance abuse/dependence diagnoses (N.SUD);

[a3] PTSD symptoms severity total score (CAPS-5);

[b4] DTS=Distress Tolerance Scale (Total Score);

[b5] BH=Breath-Holding (duration in seconds);

[b6] MT=Mirror-Tracing Task (persistence in seconds);

[b7] PASAT=Paced Auditory Serial Addition Task (persistence in seconds);

[c8] Substance cravings/urges in response to trauma cues;

[c9] Control/safety ratings in response to trauma cues;

[c10] Substance cravings/urges in response to substance cues;

[c11] Control/safety ratings in response to substance cues;

[c12] HRV=High frequency heart rate variability in response to trauma cues;

[c13] HRV=High frequency heart rate variability in response to substance cues.

[d] Values represent residualized difference scores between active (trauma or substance) and neutral conditions.

[f] All significance levels are based on 95% Bootstrapped confidence intervals of correlation coefficients (1000 resamples).

Table 4

Linear Regression Results: Trauma and Substance Cue-Related Reactivity

Trauma Cues: Self-Reported Cravings/Urges£					
	Δ *Adjusted* R^2	**b**	**SE**	**sr^2**	***p***
Step 1	**.23**				**.001**
Gender		11.91	15.45	.01	.44
Number of SUD[1]		−15.12	10.09	.05	.13
PTSD severity[2]		1.09	.66	.09	.10
Step 2	**.13**				**.02**
DTS (total score)[3]		−.24	.76	.001	.75
BH (in seconds)[4]		.36	.26	.03	.17
MTPT (in seconds)[5]		−.27	.10	.12	.03
PASAT (in seconds)[6]		.06	.05	.03	.24
Trauma Cues: Self-Reported Safety/Control£					
	Δ *Adjusted* R^2	**B**	**SE**	**sr^2**	***p***
Step 1	**.17**				**.01**
Gender		1.76	17.51	.0002	.92
Number of SUD		−6.69	8.97	.01	.45
PTSD severity		−1.19	.58	.14	.07
Step 2	**.02**				**.28**
DTS (total score)		−.084	.81	.0003	.91
BH (in seconds)		−.013	.27	.0001	.96
MTPT (in seconds)		−.025	.13	.001	.85
PASAT (in seconds)		.023	.05	.01	.65
Substance Cues: Self-Reported Cravings/Urges£					
	Δ *Adjusted* R^2	**B**	**SE**	**sr^2**	***p***
Step 1	**.28**				**<.001**

Trauma Cues: Self-Reported Cravings/Urges£					
	Δ *Adjusted* R^2	**b**	**SE**	**sr^2**	***p***
Gender		−25.59	15.43	.06	.11
Number of SUD		−14.67	9.10	.05	.10
PTSD severity		1.50	.50	.19	.01
Step 2	**.03**				**.28**
DTS (total score)		−.14	.67	.001	.83
BH (in seconds)		.34	.24	.03	.16
MTPT (in seconds)		−.067	.13	.01	.62
PASAT (in seconds)		.023	.04	.01	.57
Substance Cues: Self-Reported Control/Safety£					
	Δ *Adjusted* R^2	**B**	**SE**	**sr^2**	***p***
Step 1	**.18**				**.002**
Gender		23.27	9.98	.15	.08
Number of SUD		3.46	5.86	.01	.35
PTSD severity		−.45	.37	.04	.22
Step 2	**.20**				**.004**
DTS (total score)		1.01	.40	.13	.03
BH (in seconds)		.08	.16	.01	.62
MTPT (in seconds)		−.05	.06	.01	.33
PASAT (in seconds)		.04	.02	.05	.20

Note. p-values of regression coefficients have been reported following FDR correction for family-wise error rate.

[1]Number of Substance abuse/dependence diagnoses (N.SUD);

[2]PTSD symptoms severity total score (CAPS-5);

[3]DTS=Distress Tolerance Scale (Total Score);

[4]BH=Breath-Holding (duration in seconds);

[5]MTPT=Mirror-Tracing Persistence Task (persistence in seconds);

[6]PASAT=Paced Auditory Serial Addition Task (persistence in seconds).

[f] All outcome variables across the regression models are residualized difference scores.