An Exploratory Study of Varying Phenotypes of Posttraumatic Stress Among a Comorbid Substance Misuse Population

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Dedication

For all who have struggled to find recovery.

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Abstract

Objective: The purpose of this study was to explore differences in presentation of posttraumatic distress (PTD) that may represent different phenotypes, such as a possible cognitively-driven variant, in addition to those rooted in the prevailing conditioned-fear model. In conjunction, links to substance misuse and a purposeful selection bias for specific drugs-of-choice (DoC) based on phenotype variation were examined. **Method:** A convenience sample of inpatients in residential treatment for substance misuse who also endorsed posttraumatic distress following at least one previous traumatic experience (N = 177) completed self-report assessments and an in-person direct inquiry. **Results:** Hierarchical cluster analysis and ANOVA results partially supported our hypotheses and provided some evidence of a cognitive-focused phenotype, as well as a possible image/adrenergic-based phenotype. Subsequently, multinomial logistic regression determined that the hypothesized phenotypes were significantly linked to DoC selection, specifically (a) cognitive-focused phenotype predicted primary alcohol/benzodiazepine use (b) image/adrenergic-based phenotype predicted cannabinoid/opiate use, and (c) a traditional "mixed" PTD presentation predicted polysubstance use. **Conclusions:** Findings from this exploratory study offer additional validation to calls for continued examination of varying phenotypes, as well as a cognitively-driven model, of PTD additional to those based in conditioned-fear. Additionally, evidence was shown for a purpose-driven theory of substance misuse, hallmarked by an underlying maladaptive drive to select a DoC with the capacity to alleviate specific symptomatology (e.g., heavy alcohol use to alleviate excessive rumination and sleep disturbance).

Keywords: cognitive, adrenergic, posttraumatic distress, drug-of-choice, substance misuse

Table of Contents

CHAPTER 1: INTRODUCTION

When individuals perceive events as threatening, they may experience a variety of physiological effects stemming from the traumatic stress response, including increased adrenergic responding and cognitive hyper-attentiveness or even potential dissociation (see Sapolsky, 2004; van der Kolk et al., 2012). The various features of this internal process allow for the behavioral responding (e.g., fight-flight-freeze responses) needed to react to a potential threat. Following removal of the threat or when the situation resolves, the physiological effects of the traumatic stress response should then begin to gradually subside as the involved systems disengage and the individual enters the natural recovery cycle, which involves emotional and cognitive processing of the event (see Galovski et al., 2015). However, for some, rather than fully subsiding and resolving after the event has concluded, the process becomes stuck and natural recovery is halted (Donahue et al., 2012; Resick, 2001), leaving the body's traumatic stress response engaged. Continuation of this partial-activation mode often results in an exacerbation of physiological responses (e.g., intrusive reexperiencing, hyperarousal and -reactivity) and associated behaviors (e.g., hypervigilance, avoidance), along with increasing cognitive and affective distress, which together make up the symptomatology associated with posttraumatic stress disorder (PTSD). Why some individuals experience this lingering distress posttrauma, and others do not, is not fully clear; but, the development of PTSD has most often been attributed to a resulting conditioned fear response. Additionally, affective dysregulation or cognitive distortion coupled with disengagement style coping seems to play a significant role in maintaining PTSD (Galovksi et al., 2015; Pugh et al., 2015).

Recently, emerging evidence has suggested that other neurological pathways, in addition to the conditioned fear response, may be implicated in the development of PTSD (Gilpin & Weiner, 2017). Some researchers (e.g., Beck et al., 2011; Grunert et al., 2007) have described

individuals' distress based on non-fear-related cognitions (e.g., guilt, shame, anger), and others have noted physiological features suggestive of a possible cognitively-driven model of distress (e.g., Koenigs et al., 2008; Koenigs & Grafman, 2009, Lanius Vermetten, et al., 2010). Essentially, rumination of overly negative thoughts and beliefs about the trauma (i.e., cognitive distortions or stuck points) may be the driving factor for building internal conflict and continued posttraumatic distress (PTD). In these cases, associated fear-based adrenergic responding (e.g., intrusive reexperiencing, hyperarousal and hyperreactivity) may be a secondary consequence stemming from the distressing ruminations. Regardless of whether PTD results from conditioned fear or as a cognitively-driven reaction, co-occurrence or interplay between the affective/adrenergic and cognitive symptoms is likely. However, some individuals reporting significant distress subsequent to a variety of traumatic experiences (e.g., combat, childhood abuse, sexual assault, witnessing human carnage) may hypothetically experience distinctly greater severity in symptomatology related to one aspect over the other, endorsing either primarily imagery-based or ruminative thought-focused distress. Therefore, the current study first sought to identify whether some individuals reporting PTD endorsed greater severity of either image/adrenergic-based or cognitive-focused aspects of distress, rather than a more general evenly-mixed presentation with equivalent levels of both affective/adrenergic and cognitive distress.

In addition, PTD has been shown to be highly comorbid with substance misuse, with estimates indicating individuals diagnosed with PTSD are two to three times as likely to also have a substance use or alcohol use disorder (Gilpin & Weiner, 2017). Compelling evidence suggests that substance misuse may be purpose-driven and highly linked to "self-medicating" attempts at cognitive avoidance and emotional numbing (e.g., excessive use of alcohol to fall and

stay asleep) in those experiencing PTD (Bonn-Miller et al., 2007; Gilpin & Weiner, 2017; Jakupcak et al., 2010; Nishith et al., 2001). Hence, the choice of substance used, often referred to as drug-of-choice (DoC), also may rely heavily on symptomatologic presentation and whether greater image/adrenergic-based or cognitive-focused distress is more prevalent (i.e., arousal-dampening substances such as cannabinoids and/or opiates versus thought-suppressing substances such as alcohol). Thus, the current study also explored whether the type or pattern of distress endorsed was associated with a specific DoC, or a lack of a specific DoC. Increased knowledge and understanding of these differences may have significant implications for treatment of and recovery from both PTD and substance misuse.

Traditional Model of Posttraumatic Distress

Since the advent of the diagnosis, the American Psychiatric Association (APA, 1980) has identified PTSD as persistence of the fight-flight-freeze response long after either a potential or realized threat has passed. Through factor analysis, PTSD has been conceptualized as a multi-dimensional condition, symptomatically consisting of a variety of 17 to 20 individual signs of disturbance that fall under 3 to 4 main diagnostic “Criterions” (i.e., subgroups of symptoms), depending on the edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM; see APA, 1980; 1987; 1994; 2000; 2013; Buckley et al., 1998; Cox et al., 2008). Traditionally, these diagnostic Criterions have clustered individual symptomatic items based on intrusive reexperiencing, avoidance and numbing, cognitive and affective dysregulation, and/or hyperarousal and reactivity, with a required minimum number of items under each cluster endorsed at a sufficient level of severity to meet threshold for the diagnosis. Theories of PTSD and corresponding assessment measures have in turn been developed to further define and identify the disorder (e.g., Blake et al., 1995; Foa et al., 1989; Keane et al., 1988; King & King,

1994; King et al., 1998; Yehuda & LeDoux, 2007), with most based on an understanding of the underlying mechanisms involved in behavioral conditioning (i.e., conditioned fear model). However, with the recent inclusion of a newly refined symptom cluster, *Negative Alterations in Cognition and Mood* (i.e., Criterion D), the latest edition of the DSM (i.e., DSM-5; APA, 2013) seems to acknowledge the notion that PTSD may not be limited to a fear response, but may also involve dysregulation of other states, such as guilt and shame (Friedman et al., 2010; Lanius, Frewen, et al., 2010).

The prevailing model of posttraumatic stress has implicated dysfunction in the hypothalamic-pituitary-adrenal axis' negative feedback loop, centering on a conditioned-fear response, in which a hypersensitive and overactive amygdala is frequently triggered by resulting conditioned stimuli (Briscione et al., 2014; Friedman, 2015; Koenigs & Grafman, 2009; Koenigs et al., 2008; Sapolsky, 2004, Yehuda & LeDoux, 2007). Thus, an individual's traumatic stress response is engaged and then becomes figuratively stuck in a highly aroused and responsive state long after what would be clinically expected (Resick, 2001; Yehuda & LeDoux, 2007). As an additional byproduct of this over-responding, the amygdala imposes a severe inhibition of the prefrontal cortex, the brain's center for critical and abstract thought. This inhibition results in cognitive suppression and a significantly diminished capacity for the cognitive processing necessary to effectively counter amygdala hyperreactivity and extinguish the traumatic stress response via the properly-functioning feedback loop. In effect, the individual experiences a massive signal of threat when encountering conditioned stimuli while being simultaneously hindered in his or her ability to enlist the prefrontal cortex to accurately assess the validity of the perceived threat signal or effectively relay any discrepant assessment (Donahue et al., 2012).

Much PTSD research has focused on identifying effective therapeutic interventions, such

as empirically-based therapies that provide behavioral desensitization to conditioned stimuli and/or cognitive reprocessing of the event (e.g., Chard, 2005; Foa et al., 2000; Macdonald et al., 2016; McLean & Foa, 2014; Resick et al., 2008; Shapiro, 1989). Although some overlap exists between behavioral and cognitive approaches, more behaviorally-based approaches (e.g., Prolonged Exposure) primarily utilize imaginal and *in vivo* exposure techniques to flood the amygdala, with the goal of habituating to the threat stimuli and eventual extinction of the over-responding (Abramowitz, 2013; Foa et al., 1991; Hembree et al., 2003). Conversely, cognitively-focused approaches (e.g., Cognitive Processing Therapy, Cognitive-Behavioral Conjoint Therapy) attempt to initiate prefrontal cortex activation within the trauma context in order to engage and then cognitively suppress amygdala hyperactivity (Resick & Schnicke, 1992; Monson et al., 2005). These trauma-focused behavioral and cognitive approaches, rooted in the conditioned fear model, have been utilized interchangeably to treat PTSD with high efficacy and long-term effectiveness (see Ehlers et al., 2010; Powers et al., 2010; Resick et al., 2012; Rizvi et al., 2009; Stein et al., 2012).

However, evidence that the conditioned fear model may not account for all PTSD presentations, or what may be additional phenotypes, is increasing (see Grunert et al., 2007; Lanius et al., 2005; Lanius Vermetten, et al., 2010; Stein et al., 2012); and, although highly effective overall, these evidence-based approaches may not be equally effective for all PTD presentations (see Bradley et al., 2005; Grunert et al., 2003; Resick, Suvak, et al., 2012; Rizvi et al., 2009; Stein et al., 2012). For example, some evidence suggests that non-fear-based PTD may be less responsive to exposure techniques (Grunert et al., 2007; Lee et al., 2001). While exploring possible contributors to non-response following use of a behaviorally-based treatment (i.e., Prolonged Exposure) for PTSD with industrial accident victims, Grunert and colleagues

(2007) observed that non-responders primarily endorsed distress that was not immediately fear based (i.e., guilt/shame/anger-focused). Furthermore, following the addition of cognitive restructuring components into extended treatment, 18 of the 23 previous non-responders saw reduction in distress and no longer met diagnostic levels of PTSD. These observed differences in treatment response appear to be related to the variations in presentation of PTD. Therefore, a greater understanding of variation between possible differential phenotypes may assist with identifying the "treatment of best fit" for each case, thereby potentially significantly reducing non-response rates to first-line PTSD interventions (Seidler & Wagner, 2006).

Calls for Reconceptualization of PTD

Recently, some researchers have called for further efforts to explore other potential phenotypes of PTD, aside from the well-studied conditioned fear model (Friedman, 2015; Lee et al., 2001; Stein et al., 2012; Yehuda & LeDoux, 2007; Yehuda & McFarlane, 1995). Friedman (2015) has suggested that multiple diagnostic phenotypes of posttraumatic distress—each involving a unique pathogenesis, presentation, prognosis, and "treatment of best fit"—likely exists. The emerging premise is that posttraumatic stress may be similar in variability to our current view of mood disorders, necessitating further deconstruction of the current PTSD diagnostic criteria, potentially in favor of a spectrum of clinical phenotypes delineated by varying overlapping symptomatologies (Friedman, 2015; Lobo et al., 2015).

Support for Additional Phenotypes

While much of the symptomatology commonly associated with PTD does fall under what would traditionally be considered an anxiety disorder, many of its psychological and physiological signs and features (e.g., mood dysregulation, variations in neurologic activation and cortisol levels) serve to distinguish PTSD as more than a conditioned-fear based anxiety

disorder (Craske et al., 2009; Friedman, 2013; Friedman et al., 2011; Stein et al., 2011; Yehuda & LeDoux, 2007). In partial response to this growing awareness, the DSM-5 (APA, 2013) has removed PTSD from inclusion as an anxiety disorder as well as added a dissociative subtype into the diagnosis (Friedman, 2013; Regier et al., 2013). However, researchers continue to identify additional potential phenotypes (e.g., adrenergic, developmental trauma) not currently represented in the diagnosis (see Briscione et al., 2014; Raskind et al., 2014; Simpson et al., 2015; van der Kolk et al., 2009). Further, Friedman (2015) has suggested that PTSD aligns better along a distress-disorders subset of internalizing disorders (e.g., major depression, dysthymia, generalized anxiety disorder) or externalizing disorders (e.g., substance misuse, conduct disorders), rather than fear disorders (e.g., panic, various phobias). Friedman has also proposed that findings further support the argument that PTSD may at times present as primarily distressing rumination and anhedonia (i.e., potentially cognitively-driven) rather than simply pathological fear. Thus, if accurate, existing models based in conditioned fear would not account for all PTSD presentations.

Similarly, recent studies utilizing neuroimaging methods have identified an inverted neurological pathway present in some expressions of PTD with some subjects diagnosed with PTSD demonstrating increased prefrontal cortex and decreased amygdala activity, rather than the inverse (i.e., amygdala hyperactivity and prefrontal cortex hyporeactivity) expected in the conditioned fear model (Lanius, Vermetten, et al., 2010). Further support has come from Koenigs and colleagues' (2008; 2009) research involving combat veterans with brain injury, which found decreased likelihood of developing PTSD in some subjects with damage to the prefrontal cortex. These findings are contrary to what would be expected based on the conditioned fear model and may be suggestive of a cognitively-driven phenotype of PTD.

A Possible Cognitively-Driven Phenotype

As previously noted, some studies have indicated that prefrontal cortex hyperactivity, rather than hyporeactivity, may play a dominant role in certain cases of PTD (Koenigs et al., 2008; Koenigs & Grafman, 2009; Lanuis et al., 2010). This observed prefrontal cortex hyperactivity may be associated with greater distress centered around alterations in cognition and mood (e.g., guilt- or shame-focused ruminations, intrusive negative thoughts); while prefrontal cortex hyporeactivity, which is traditionally anticipated in PTSD, may be associated with greater distress centered on reexperiencing and hyperarousal. When an individual experiences a traumatic event, certain pre- and peri-traumatic facets of the event may conflict with the individual's pre-trauma view of self, others, and the world (Lee et al., 2001; Resick & Schnicke, 1992). In most cases, individuals are able to cognitively process these incongruencies during the natural recovery cycle, making adaptive accommodations for this new experience into their existing worldview without significant long-lasting distress (Donahue et al., 2012; Galovski et al., 2015). However, at times, individuals may have difficulty in making meaning of the event and successfully accommodating the new information, which may result in distorted maladaptive cognitions involving self-criticism and self-blame (e.g., "it was my fault"; "I am a bad person"). These self-deprecating thoughts may then provide the foundation for evolving beliefs that call into question one's own self-efficacy to evade future trauma, thus cognitively-driving an affective/adrenergic response (e.g., "I will continue to make poor decisions and invite threat and harm, which leaves me feeling unsafe"). Coupled with maladaptive coping strategies used to avoid cognitive and affective distress (e.g., substance misuse), overly negative cognitions (e.g., guilt, shame, blame) may continue unchallenged and contribute to increasingly greater distress (Held et al., 2011; Resick, 2001; Street et al., 2005). From this perspective, this study sought to

further examine possible variations in posttraumatic stress presentations that may be indicative of a cognitively-driven phenotype of PTD, marked by heavy rumination of strong negative cognitions that, when not avoided, acutely engage other more affective/adrenergic responses, such as increased emotional lability and reactivity.

A Purpose-Driven Theory of Substance Misuse

A growing body of research has shown substance misuse, which is often conceptualized as attempts at cognitive avoidance and emotional numbing (i.e., disengagement coping), to have a high degree of comorbidity with various mental health disorders (Bonn-Miller et al., 2007; Brotchie et al., 2004; Jakupcak et al., 2010; Najavits et al., 1997; Tull et al., 2010). This view of substance misuse as an avoidance and/or numbing strategy is particularly salient with trauma survivors (e.g., Bonn-Miller et al., 2010; Held et al., 2011; Held et al., 2015; Najavits et al., 1997). As a result, researchers have called for further exploration into the periods of transition from simple use into increased substance misuse and the possible underlying motivations (e.g., recent traumatic experience; physical traumatic injury), rather than focusing solely on the period after actual dependence (i.e., physical addiction) onset (Bonn-Miller et al., 2010; Swendsen et al., 2010). One large sample (N = 5001) longitudinal study, carried out during the 10-year follow-up to the National Comorbidity Survey (Swendsen et al., 2010), found that behavioral disorders and prior substance use were the greatest predictors of the transition from use to misuse and that PTSD specifically was associated with dependence on at least one substance. Additionally, Najavits and colleagues (1997) have cited comorbidity rates for substance misusers who have a dual diagnosis of PTSD to be as high as 59% in some samples.

Variations in PTD presentations (i.e., variations in phenotypes) also may account for differences in DoC, possibly associated with which brain regions are affected (Semple et al.,

2000). We hypothesized that PTD characterized by a high degree of cognitive hyperactivity, involving frequent rumination and intrusive thoughts (i.e., cognitive-focused), may be highly associated with use of substances, such as alcohol, that heavily impact and inhibit cognitive functioning. Alternatively, image/adrenergic-based PTD, hallmarked by heavy affective or adrenergic arousal around reexperiencing and intrusive imagery (i.e., visual memory), may prompt greater reliance on substances such as cannabinoids and opiates, which may better assist in emotional numbing.

Alcoholic beverages can significantly disrupt cognitive functioning, often bringing about a euphoric state while also dampening cognitive processing (Gould, 2010; Oscar-Berman, & Marinkovic, 2007). Studies have shown the frontal lobes, which encompass the prefrontal cortex, to be particularly susceptible to the effects of alcohol (Oscar-Berman & Hutner, 1993; Oscar-Berman & Marinkovic, 2003), leading to impairment in executive functioning as well as cognitive deficits in decision-making and impulse-control (Oscar-Berman & Marinkovic, 2007). Thus, alcohol's known capacity for dulling cognitive functioning may be especially appealing to PTSD sufferers experiencing greater rumination of cognitive-focused (e.g., guilt, shame, blame) distress and searching for a means of suppressing negative thoughts. Therefore, we hypothesized that endorsement of alcohol as DoC will be associated with primarily cognitive-focused distress and increased severity in the *Alterations in Cognitions and Mood* (Criterion D) symptoms, further outlined in the subsequent "Purpose of the present study" section.

Cannabinoids (e.g., marijuana, hashish) contain active-ingredients tetrahydrocannabinol (THC) and cannabidiol (CBD), chemicals that can alter mood, affect, basic senses, temporal processing, and cognitive functioning—particularly critical thinking, problem-solving, and memory (NIDA, n.d.). Additionally, cannabinoids have been shown to have properties that aid in

relaxation of cardiovascular tissue and vasodilation (Kunos et al., 2000). This vascular effect is similar to that of some medications that have been shown to reduce some of the hyperarousal aspects of PTSD (Raskind et al., 2014; Simpson et al., 2015). Furthermore, research has suggested that marijuana may ameliorate some aspects of PTD and reduce co-occurring depressive symptoms (Bremner et al., 1996; Passie et al., 2012). According to Passie and colleagues (2012), cannabinoids may temporarily reduce limbic-based affective and adrenergic responding, allowing some reprieve from intrusive memories, flashbacks, and nightmares. The authors also cited emerging evidence of cannabinoid effects on extinction of the conditioned fear response, which may hypothetically leave potential co-occurring distorted cognitions intact. Therefore, cannabinoids may be most attractive to those with heavy imagery-based distress or a more evenly-mixed presentation than for those with greater cognitive-focused distress. As such, we hypothesized that those with greater image/adrenergic-based or mixed PTD will more frequently report cannabinoids as their DoC. We also suspect that those with a more mixed symptomatology may endorse significant alcohol in addition to cannabinoid (i.e., polysubstance) use, which may be associated with lingering cognitive distress.

Opiates (e.g., heroin, oxycodone, hydrocodone, methadone) are morphine-based or -derived substances that act upon the brain's pain and pleasure centers (NIDA, n.d.). Opiates have become a significant health issue over the past decade, partially due to their capacity to temporarily alleviate some forms of psychological distress (Fareed et al., 2013; Tull et al., 2010). Fareed and colleagues (2013) conducted a review of the literature and found considerable links between PTD and opiate use. The authors hypothesized that the sedative properties in opiates might be especially appealing to those with PTSD for numbing affective/adrenergic hyperarousal. PTSD sufferers might also gravitate toward opiates in order to supplement

depleted beta-endorphin levels, the brain's natural emotional- and physical-pain reliever, which may become depleted due to frequent traumatic stress responding (Fareed et al., 2013). For these reasons, we hypothesized that opiates may also be especially appealing to, and be selected as DoC, by, those experiencing significant image/adrenergic-based PTD.

Stimulants (e.g., cocaine, amphetamines, methamphetamines) act on the brain's pleasure centers to produce short-term euphoria and extreme energy (NIDA, n.d.), which may initially be attractive for someone suffering from PTSD. However, certain effects may occur that counter this initial draw, such as greater ability to remember aspects of the trauma and increased anger and irritability (Semple et al., 2000). Furthermore, Tull and colleagues (2010) found that cocaine use showed no association with PTSD symptom clusters in a study examining relationships between PTSD and alcohol, cocaine, and heroin dependence. Therefore, we hypothesized that stimulants may be less appealing to those with PTSD, regardless of presentation or phenotype; and, greater stimulant use may be associated with lower PTD.

Purpose of the Present Study

For Aim 1, we sought to identify whether specific symptoms of PTD would cluster by severity to define multiple presentation types (i.e., phenotypes), including a cognitive-focused (cognitively-driven) and image/adrenergic-based phenotypes. We hypothesized first (Hypothesis 1) that some individuals who have experienced prior trauma resulting in at least some distress will present with PTD symptomatology hallmarked by significantly greater focus on individual symptoms related to cognitive distress (e.g., guilt, shame, blame) than on those related to imagery and adrenergic responding. Specifically, we predicted in Hypothesis 1 that individuals who identify as experiencing primarily cognitive-focused distress will endorse greater severity among some *Negative Alterations in Cognitions and Mood* (Criterion D) items—i.e., item D2

"strong negative beliefs about self, others, or the world," item D3 "excessive blaming of self or others," and item D4 "strong negative feelings"—and *Hyperarousal and Reactivity* (Criterion E) subgroup item E2 "Engaging in high risk behaviors." Conversely, we hypothesized that an image/adrenergic-based phenotype would also emerge, hallmarked by significantly greater focus on individual symptoms involving distressing imagery (e.g., visual memory, nightmares) and affective and adrenergic reactivity (e.g., hypervigilance). Specifically, we anticipated in Hypothesis 2 that individuals who identify as experiencing primarily image/adrenergic-based distress would endorse greater severity among some or all Intrusive Reexperiencing (Criterion B) items—particularly item B1 "intrusion by trauma-related memories"—and some Criterion E items—i.e., item E1 "irritability, angry outbursts, and aggression," item E3 "hypervigilance," item E4 "increased startle," and item E5 "difficulty concentrating." Finally, as an overall comparison group, we posited that individuals who identify as experiencing a more traditional (i.e., symptomatically broad) mixed symptomatology will not endorse greater severity of one cluster of PTD items (i.e., cognitive-focused versus image/adrenergic-based) over the other.

For Aim 2, we examined whether DoC may align with the proposed variations in PTD presentations, which may provide supporting evidence of potential differences in physiological mechanisms (e.g., cognitively-driven) underlying our hypothesized phenotypes. Specifically, our third hypothesis (Hypothesis 3) predicted that individuals who identify as experiencing greater cognitive-focused distress would indicate alcohol as their DoC. Furthermore, those endorsing higher image/adrenergic-based distress would indicate cannabinoids and/or opiates as DoC. Finally, those reporting a more symptomatically evenly-mixed presentation, with relatively similar levels of affective/adrenergic and cognitive distress, would indicate polysubstance use.

CHAPTER 2: METHOD

Participants

Study participants were recruited from a pool of adult (i.e., 18 years or older) residents in a private 30-day residential substance misuse treatment facility in the eastern United States. Of more than 200 who met initial prescreening criteria (i.e., prior report of at least one experience perceived by the resident as being traumatic) and agreed to take part, 177 residents endorsed continued distress and impairment subsequent to at least one event meeting current DSM-5 PTSD Criterion A (i.e., the index stressor) diagnostic requirements (i.e., APA, 2013) and responded to a sufficient portion of the assessments (i.e., at least 95%) to be included in final analysis. Over half the sample (65%) was male, with age ranging from 19 to 65 years (M = 38.0, SD = 10.6). The majority of participants were White (89%), followed by 4% Hispanic ethnicity, 3% Black, and 3% multiracial. With regard to sexual orientation, most participants identified as straight (92%), with 5% identifying as gay, and 3% bisexual. Over half (54%) of the participants had no previous treatment experience prior to this inpatient admission and, on average, were in either their second or third week of treatment (M = 13.3 days, SD = 7.2) at the time of study recruitment and participation. Finally, over half (54%) of the participants met threshold criteria for probable diagnosis of PTSD based on a PTSD screening measure described below, and all had a current diagnosis of at least one substance use disorder. As an additional safeguard against potential confounds in exploring variations in presentation of PTD, none of the participants had taken part in a specifically trauma-focused therapy prior to their entering the current period of treatment.

Measures

Demographics

Basic demographic information concerning age, biological sex, race/ethnicity, sexual

orientation, and employment status was collected during concurrent admissions records review at time of study interview.

Trauma History and Posttraumatic Distress

Participant trauma history, selection of index trauma, and current level of posttraumatic distress were obtained using the PTSD Checklist for DSM-5 (PCL-5) with Life Events Checklist for DSM-5 (LEC-5) and extended Criterion A (Weathers et al., 2013). This version of the PTSD Checklist is a compilation of preexisting measures and diagnostic criteria commonly used in assessment of trauma history and PTSD. The component measures have each been modified from former versions to reflect current DSM-5 guidelines and combined into a three-part assessment, as follows.

Part A. The updated version of the Life Events Checklist (LEC; Gray et al., 2004) for DSM-5 is a 17-item self-report measure that prompts respondents to identify any lifetime exposure to stressful life events. For each potentially traumatic event, respondents indicate their exposure to that event on a 6-item nominal scale (i.e., happened to me, witnessed it, learned about it, part of my job, not sure, doesn't apply). In a group of 108 undergraduate psychology students the original LEC demonstrated good convergence with the Traumatic Life Events Questionnaire (TLEQ; kappa = 0.55) and correlated with the PTSD Checklist – Civilian (PCL-C; reliability ranging from 0.34 to 0.48). The LEC also demonstrated good test-retest reliability over 7 days (all kappa statistics except one > 0.52). This study utilized the LEC-5 as a screening measure to ensure the inclusion criteria of "prior traumatic experience" was met.

Part B. The Criterion A (index stressor) portion is a 9-item questionnaire that initially directs respondents to select an index trauma (i.e., that which respondent considers to be the most currently distressing trauma) from items previously identified on the LEC-5. Then, focusing on

the selected trauma, respondents answer questions that assess whether the selected index trauma met DSM-5 Criterion A for PTSD.

Part C. The PCL-5 (Weathers et al., 2013) is a 20-item self-report measure that reflects the current DSM-5 PTSD diagnostic contained in Criterions B through E. The PCL-5 evaluates symptom frequency over the past month stemming from an identified prior trauma (i.e., the previously selected index trauma). Each item is scored on a 5-point scale ranging from 0 (*not at all*) to 4 (*extremely*), with total scores ranging from 0 to 80. Higher scores indicate greater frequency and severity of symptomology associated with posttraumatic distress as well as probable presence of PTSD. Although the PCL is not a diagnostic assessment, current guidance from the National Center for PTSD (U.S. Department of Veterans Affairs, 2019) has identified a total score of 33 as the cut-point for establishing a probable diagnosis of PTSD. Symptom severity (i.e., item scores > 1) and endorsement patterns (i.e., at least one Criterion B, one C, two D, and two E symptoms) can also be combined with the total score being at or above cut-off as an additional level of assurance of probable PTSD (Blevins et al., 2015; U.S. Department of Veterans Affairs, 2019). A review by Keen and colleagues (2008) of 21 studies using the original PCL, reported overall internal consistency alpha coefficients between .85 and .96, test-retest reliability between .96 (at 2 to 3 days) and .68 (at 12 to 14 days), and convergent validity with similar measures of PTSD symptomatology. Blanchard and colleagues (1996) have previously found the PCL to have high diagnostic efficiency at .90. This study utilized the PCL-5 as a primary outcome assessment to assess for a probable diagnosis of PTSD—discriminating between (a) subthreshold score < 33 and Criterion thresholds not met (b) subthreshold score < 33 with Criterion thresholds met (c) threshold score $\geq$ 33 and Criterion thresholds not met (d) threshold score $\geq$ 33 and Criterion thresholds met. The PCL was also used to determine

individual symptom severity, identify clustering of specific symptoms, and compare anticipated phenotypic groups. Cronbach's alpha for the PCL-5 in the current study was .94.

Trauma-Related Guilt

The Trauma-Related Guilt Inventory (TRGI; Kubany et al., 1996) is a 32-item self-report measure developed to assess posttraumatic thoughts and feelings of guilt. The TRGI includes three scales (i.e., Global Guilt Scale, Distress Scale, Guilt Cognitions Scale) and 3 subscales (i.e., Hindsight-Bias/Responsibility Subscale, Wrongdoing Subscale, Lack of Justification Subscale). Items are rated on a 5-point scale ranging from 1 (*never/not at all true*) to 5 (*always/extremely true*), then summed for total and/or subscale scores. Higher scores indicate greater levels of guilt. Psychometric testing involving almost 600 individuals, including university students, battered women, and Vietnam veterans, showed moderate to high (α = .66-.94) internal consistency. The TRGI has also shown high convergent validity with the PCL (Weathers et al., 1993) and the Mississippi Scale for Combat-Related Posttraumatic Stress Disorder (Keane et al., 1988), and test-retest reliability after two days ranged from 0.74 to 0.83. Cronbach's alpha for the TRGI in the current study was .95.

Trauma-Related Shame

The Trauma-Related Shame Inventory (TRSI; Oktadalen et al., 2014) is a 24-item measure that assesses degree of posttraumatic shame, with two scales distinguishing between internal-focused and external-focused shame, as well as cognitive (i.e., self-condemnation or perceived condemnation) and affective-behavioral subscales. Items are rated on a 4-point scale ranging from 0 (*not true of me*) to 3 (*completely true of me*), then summed. Higher scores indicate greater levels of shame. G-theory testing of the TRSI has shown dependability coefficients above .80, with high measure design dependability of .87 (Oktadalen et al., 2014).

The TRSI was also shown effective in distinguishing between shame and guilt, with a high generalizability coefficient (i.e., divergent validity, differential construct validity) of .82 when compared with the TRGI. Cronbach's alpha for the TRSI in the current study was .98.

Alcohol Misuse

The Alcohol Use Disorders Identification Test (AUDIT; Saunders et al., 1993) is a 10-item self-report measure, containing three domains (i.e., hazardous alcohol use, dependence symptoms, harmful alcohol use), commonly used to assess alcohol use and potential misuse. Items are scored on a 5-point scale (0 to 4)—with item-specific anchors to indicate frequency (i.e., *never*, *less than monthly*, *monthly*, *weekly*, *daily*) or quantity of use—with total scores ranging from 0 to 40. Saunders and colleagues (1993) reported good internal consistency among items sensitive to dependence ($\alpha = .93$) and harmful use ($\alpha = .81$). Babor and colleagues (2001) identified scores of 8 or above as indicative of likely harmful use and a combined score of 4 or more on questions 4-6 and a total score of 20 and above meets cut-off criteria for alcohol dependence. This study utilized the AUDIT to examine secondary aims of determining the possibility of associations between alcohol use and posttraumatic stress phenotype and symptom severity. Cronbach's alpha for the AUDIT in the current study was .93.

Substance Misuse (Non-Alcohol)

The Drug Use Disorders Identification Test (DUDIT; Berman et al. 2005) is an 11-item self-report measure, commonly used to assess non-alcohol related substance misuse and dependence. Items are scored on a 5-point scale (0 to 4) with item-specific anchors to indicate frequency (i.e., *never*, *less than monthly*, *monthly*, *weekly*, *daily*) or quantity of use. The DUDIT has been shown to have good internal consistency ($\alpha = .80$) with a 90% sensitivity for predicting dependence and 78% specificity (Berman et al., 2005). Berman and colleagues (2005) identified

males with a score of 6 or more, and females with a score of 2 or more, as likely positive for drug-related problems; and, either sex with scores of 25 or more are likely having heavy dependence. This study utilized the DUDIT to examine secondary aims of determining the possibility of associations between drug use and posttraumatic stress phenotype and symptom severity. Cronbach's alpha for the DUDIT in the current study was .94.

Procedures

Treatment center residents were prescreened via chart review carried out by the study principal investigator, who reviewed admissions records (i.e., biopsychosocial report) and assessments (i.e., intake PCL-5) for indication of past trauma. Individuals with a history of trauma were made aware of the study by treatment center staff, at which time the resident was asked if he or she would be interested in meeting with the principal investigator to learn about possible participation. If interested, the resident was scheduled for a recruitment briefing, conducted privately in an on-site individual therapy room, to be further informed about the study, requirements for participation, their right to decline to participate, as well as the right to confidentiality, should they choose to participate.

After reviewing the informed consent document and obtaining signed consent, eligibility was confirmed through participants' self-report of trauma history via the Life Events Checklist-5 to verify the accuracy of intake reports and identify any previously unreported trauma. Participants also verbally affirmed experiencing current distress related to at least one traumatic event and selected an index trauma (i.e., event that they considered to be the most traumatic and currently distressing). Participants then provided further details about their index event to verify that it met DSM-5 Criterion A (APA, 2013) guidelines for an experience that could potentially produce PTSD. To ensure measure fidelity, the principal investigator then verbally administered

the PCL-5 to establish the presence and severity of related PTD. Next, participants completed paper copies of the TRGI and TRSI to examine potential aspects of trauma-related guilt and shame. While participants completed these measures, the principal investigator accessed intake records and recorded archived demographic data and participants' responses to the AUDIT and DUDIT. Finally, participants engaged in a brief interview with the principal investigator. First, participants identified whether their experience of posttraumatic distress seemed to be more focused on negative cognitions (i.e., thoughts about their trauma), distressing imagery (i.e., visual memories of the trauma), or a mixed presentation with neither thoughts nor any visual memories being more distressing than the other. Next, participants reported their drug-of-choice (DoC), or all substances used if they were polysubstance users with no specific DoC. Participants then indicated whether they considered their substance use to be related to alleviating trauma-related distress. Finally, participants described how their chosen substance(s) "functionally" helped with trauma-related distress (e.g., "alcohol helped me to sleep"; "opiates numbed me").

Data Analysis

SPSS software (version 25.0) was used for all statistical analyses. Missing data patterns for all outcome variables were analyzed, showing that less than 1% of all individual item-level data points were missing. Cases were excluded from final analysis if interview questions were not responded to or if more than 5% of an outcome measure was left incomplete. Of the 185 participants initially enrolled, two cases (1%) did not endorse one of the three PTD phenotypes and six cases (3%) had at least one measure with data entirely missing and were excluded. Otherwise, in accordance with common practices (see Tabachnick & Fidell, 2007), mean substitution was utilized for four other cases with instances of missing item-level data of less than 5% of a measure.

Means, standard deviations, and correlations for variables of interest, as well as internal consistency reliability for study measures, were calculated. When appropriate, chi square tests were used to determine relationships for nominal variables. All variables were examined to determine whether basic assumptions for all planned statistical methods were met. Levene's test for homogeneity of variance (Levene's *W*) was utilized to verify that the assumption of homoscedasticity was not violated when using Fisher's ANOVA. Tukey's Honestly Significant Difference (HSD) Post-hoc Test was carried out for multiple comparisons. If Levene's statistic showed significance, demonstrating heteroscedasticity, Welch's unequal variances *t*-test (Welch's *t*) was utilized for analysis of variance, followed by Games-Howell Nonparametric Post-hoc Test for multiple comparisons.

Items from the PCL-5 were included in a hierarchical cluster analysis as an initial examination of Aim 1, which postulated variations in PTD presentation defined by clustering of specific symptoms. Grouping-type analyses are considered appropriate methods for detection and evaluation of hypothetical factor structures outlining various mental health diagnoses (Asmundson et al., 2000; Henry et al., 2005). Cluster analysis is considered to be a viable grouping technique when seeking to minimize between-group confounds while establishing within-group commonalities (Henry et al., 2005). Common guidelines for sample size estimates in various types of grouping analyses recommend 5 to 10 subjects per variable, necessitating a minimum of 100 participants, given 20 symptom variables in this study's model (i.e., PCL-5 items). However, a minimum of 150 subjects is recommended for sufficient power with highly correlated instrument items, which was the case for this study (Arrindell & van der Ende, 1985; Hutcheson & Sofroniou, 1999). In addition, to explore the hypothesized existence of varying phenotypes of PTD (Aim 1), participants were grouped based on self-assessment of their PTD

presentation (i.e., cognitive-focused, image/adrenergic-based, mixed) to form fixed variables for a series of one-way analysis of variance (ANOVA) to test for mean differences in PTD symptom severity (i.e., PCL-5 scores) and degree of trauma-related guilt and shame. Variations in severity of alcohol and non-alcohol substance misuse (i.e., AUDIT and DUDIT scores, respectively) were also examined. G*Power (Faul et al., 2009) estimated that ANOVA with alpha = .05 required n = 159 subjects to achieve power = .80 with a medium effect size (d = .25).

Finally, to explore Aim 2, a multinomial logistic regression was conducted to identify associations between substance use and PTD presentation. We sought to determine the probability of the categorical variables of PTD phenotypes' (i.e., cognitive-focused, image/adrenergic-based, mixed presentation) membership in the dependent categorical variables of DoC (i.e., alcohol/benzodiazepine, cannabinoids/opiate, polysubstance use). Polysubstance use was selected as the reference category.

CHAPTER 3: RESULTS

Preliminary Analysis

As sex differences are often found in severity and diagnosis of PTSD (e.g., Najavits et al., 1997; Olff, 2017), some preliminary analyses were conducted to determine whether differences existed among main variables in the current sample. Results from independent samples *t*-test indicated that female sex was associated with greater overall PTD, with females reporting significantly greater severity for PTSD symptomatology (Table 1) and more trauma-related shame than did males ($p < .001$). However, males reported more trauma-related guilt than did females ($p < .001$).

Additionally, an independent samples *t*-test indicated that females endorsed significantly higher levels of non-alcohol substance use than did males ($p < .001$). Conversely, males reported greater alcohol misuse than females ($p = .003$). While these preliminary analyses indicate the need to control for biological sex in analyses when possible, we were unable to do so because of vastly unequal sample sizes for phenotype groupings. For example, for the image/adrenergic-based group, male subsample size ($n = 25$) was five times that of female subsample size ($n = 5$).

Finally, means, standard deviations, and correlation coefficients for all outcome variables for the entire study sample were calculated (Table 2). PTSD total scores were highly correlated ($p < .001$) with both trauma-related shame and trauma-related guilt, which demonstrated a strong inverse relationship with both the PTD and trauma-related shame ($p < .001$). Additionally, non-alcohol substance misuse demonstrated a moderate positive correlation with PTD and trauma-related shame ($p < .001$), as well as a moderate negative correlation with trauma-related guilt ($p < .001$). Finally, alcohol misuse was negatively correlated with non-alcohol substance misuse ($p < .001$) but was not significantly correlated with PTD or trauma-related guilt or shame.

Hypothesized Phenotypes and Variations in PTD

To examine Aim 1 (i.e., exploring PTD presentation and the possible existence of varying phenotypes), we first conducted a hierarchical cluster analysis of PCL-5 items to explore whether the individual PTD variables would cluster into distinct groups aligned with our hypothesized cognitive-focused and image/adrenergic-based phenotypes. The nearest neighbor single-linkage method was first employed to identify any potential outliers among the variables. Posttraumatic nightmares (item B2) and flashbacks (item B3),which clustered together, avoidance of external trauma reminders (item C2), difficulty recalling important parts of the trauma (item D1), risk taking (item E2), and sleep disturbance (item E6) emerged as potential outliers (i.e., not closely related to other items and/or clusters) but were all retained for analysis in the final hierarchical cluster analysis, which employed Ward's minimum variance method with squared Euclidean distance measuring. Results indicated a four-cluster solution, appearing to partially align with Hypothesis 1 and 2 (Table 3) (Figure 1). Specifically, the items making up Cluster1 combined reexperiencing of certain trauma-related imagery and avoidance symptoms. Cluster 2 was comprised of PTD items related to cognitive distress and the negative effects of heavy rumination, with the notable inclusion of sleep disturbance (item E6), which has traditionally been viewed as a hyperarousal and reactivity item. Cluster 3 was made up solely by items related to adrenergic hyperreactivity, including risk taking (item E2), which was originally hypothesized to align with the cognitive-focused items. Finally, Cluster 4 items included posttraumatic nightmares (item B2), flashbacks (item B3), and difficulty remembering (item D1), all of which again appeared relatively distant from the other items and clusters.

Additionally, a series of one-way ANOVAs were conducted to detect significant variations in total severity of PTD and degree of distress for each Criterion, as well as for

trauma-related guilt and shame and degree of substance misuse, between participants grouped according to their self-selected PTD phenotype (see Table 4). Although no significant differences were found between the image/adrenergic-based PTD group and either the mixed [$t(162) = 8.57$, *ns*] or cognitive-focused groups [$t(162) = 0.40$, *ns*] for overall severity (i.e., total PCL-5 score), post hoc analysis detected significantly greater distress for the mixed PTD presentation group compared with the cognitive-focused PTD group [$t(162) = 8.18$, $p = .031$]. Furthermore, at the level of Criterions, mixed presentation showed significantly greater distress involving *Negative Alterations in Cognitions and Mood* (Criterion D) than image/adrenergic-based [$t(162) = 5.07$, $p = .025$]. Otherwise, no significant variability was found between phenotype groups for either of the two secondary measures of PTD (i.e., Trauma-Related Guilt Inventory and Trauma-Related Shame Inventory) or for either measure of degree of substance misuse (i.e., Alcohol Use Disorders Identification Test and Drug Use Disorders Identification Test).

Relationship between DoC and PTD

To establish the dependent variable for examining Aim 2 (i.e., associations between PTD factors and DoC selection) and testing Hypothesis 3 (i.e., PTD phenotype predicts DoC), participants identified their DoC, if any, during the assessment session. Two additional categories emerged (i.e., benzodiazepine, hallucinogen), beyond the four identified *a priori* (i.e., alcohol, cannabinoid/opiate, stimulant, polysubstance), based on participants' responses. Benzodiazepine use, in particular, was seen to coincide with primary alcohol use. Therefore, in considering these additions, we determined that benzodiazepines, as sedatives with similar effects on the brain's GABA system to that of alcohol, would be grouped with alcohol into one category. Otherwise, primary hallucinogen use remained as an independent category but was

excluded from any analyses with DoC (e.g., multinomial logistic regression, ANOVA) due to the minimal representation (i.e., only two cases) in this category. Cases involving primary stimulant use (n = 10) were similarly excluded due to small sample size.

In testing Hypothesis 3 (i.e., PTD phenotype predicts DoC), the multinomial logistic regression model was significant [χ^2 (4) = 24.33, $p < .001$]. Results indicated that individuals who reported cognitive-focused presentation compared to mixed were more likely to have alcohol/benzodiazepines as the DoC rather than polysubstance [Exp(B) = 3.96, $p < .001$]. Participants reporting either a cognitive-focused [Exp(B) = 4.85, $p < .05$] or image/adrenergic presentation [Exp(B) = 6.42, $p < .05$] compared to mixed were more likely to have cannabinoid/opiate as DoC rather than polysubstance (Table 5). Proportionally, nearly half (49%) of the mixed PTD group (n = 33) endorsed polysubstance use and accounted for 56% of the DoC's membership. Alternatively, membership in the cognitive-focused PTD group predicted primary alcohol/benzodiazepine use. Participants reporting cognitive-focused PTD were 366% more likely than the image-adrenergic PTD group and 296% more likely than the mixed PTD group to endorse alcohol/benzodiazepine over polysubstance use. The cognitive-focused group was also 385% more likely than the mixed group to endorse cannabinoid/opiate as DoC over polysubstance use. Two-thirds (66%) of cognitive-focused group members (n = 53) accounted for 62% of primary alcohol/benzodiazepine use group membership. Finally, membership in the image/adrenergic PTD group predicted primary cannabinoid/opiate use. Participants were 32% more likely than the alcohol/benzodiazepine group and 542% more likely than the mixed group to endorse cannabinoid/opiate as DoC over polysubstance. Those reporting image/adrenergic-based PTD displayed greater relative proportion of membership in the cannabinoid/opiate group, with 28% (n = 7) endorsing cannabinoid and/or opiate as DoC,

compared to only 12% of cognitive-focused and 6% of mixed presentation group members, and accounting for one-third (33%) of that DoC group's membership.

CHAPTER 4: DISCUSSION

The purpose of the present study was to explore whether distinctly varied presentations, or phenotypes, of PTD exist (Aim 1) and to examine how those variations might be associated with comorbid substance misuse (Aim 2). Specifically, we hypothesized that certain symptomatic patterns of PTD would generalize across individuals to form novel groups, defined by distinct presentations that would include a cognitive-focused and image/adrenergic-based phenotype, and that those phenotypic variations would predict selection of DoC.

Varying Phenotypes of PTD

Results from our multi-pronged approach at exploring Aim 1 generally supported this study's first two hypotheses proposing varied phenotypes. First, hierarchical cluster analysis exploring varied response patterns of the individual items of PTD symptomatology appeared to show the variables grouping into clusters (i.e., imagery-based and adrenergic distress with avoidance, ruminative distress, adrenergic distress, and imagery-based distress) that partially aligned with our proposed phenotypic variations (i.e., cognitive-focused distress versus image/adrenergic-based distress). Interestingly, in congruence with some findings from both clinical and non-clinical samples (e.g., Clancy et al., 2020), sleep disturbance (i.e., PCL-5 item E6), which was not addressed in our predictions, clustered alongside the items of ruminative distress rather than near item B2 "nightmares" or the items of adrenergic arousal. A recent meta-analysis of research exploring perseverative cognition and sleep disturbance in non-clinical populations (Clancy et al., 2020) reported findings that perseverative cognition (i.e., worry and rumination) was associated with diminished sleep quality, reduced sleep time, and extended onset latency. These findings and Brosschot and colleagues' Perseverative Cognitions Hypothesis (2006), which posits that perseverative cognition engages the traumatic stress

response system in much the same way as do physical stressors, align with our findings and further suggest that sleep disturbance would logically cluster with our cognitive-focused PTD factors. This interplay seems to suggest additional support for the possibility of variations in the physiological mechanisms underlying PTD (i.e., allowing for a cognitively-driven model of PTD). Alternatively, Bonn-Miller and colleagues (2010) found marijuana use, which the current study and prior research (i.e., Bremner et al., 1996) have found to be associated with arousal and reactivity symptoms, to also be linked to coping with trauma-related sleep disturbance. Additionally, Vandrey and colleagues (2014) reported all of our primary substances (i.e., alcohol, benzodiazepines, cannabis, and opiates) to be arousal-dampening with similar capabilities for decreasing sleep latency. However, this apparent contradiction may be indicative of overlap and/or confusion between experience of trauma-related nightmares (i.e., image/adrenergic-based), which is its own item on PTSD assessments, and nighttime rumination (i.e., cognitive-focused) as both equating to sleep disturbance, despite having vastly different underlying neurobiological substrates.

Second, participants' selection of PTD presentation (i.e., cognitive-focused, image/adrenergic-based, or mixed presentation) was informed by the respondents' subjective assessments of their own experiences of symptomatology. Although their choices were between only the three named phenotypes, the mixed presentation (i.e., a more evenly-mixed traditional PTD presentation) acted as a form of baseline contrast, allowing for somewhat of a "not applicable" answer to the underlying question of cognitive-focused versus image/adrenergic PTD. Essentially, we asked participants to select between PTD-as-usual or one of two presentations with varied symptomatologies. Therefore, given that 62% (n = 109) of participants reported experiencing PTD in line with one of the hypothesized variable presentations

(particularly the cognitive-focused phenotype) over the usual mixed presentation, we believe that stable variations in PTD presentation were present. This finding is particularly salient when cross-referenced with the cluster analysis results above.

Our hypotheses were also supported by significant differences found in PTD severity based on the participants' self-selected presentation of distress. Those reporting a mixed presentation demonstrated the greatest severity of distress, particularly when compared to cognitive-focused. Additionally, the mixed group presented with a more broad and evenly-dispersed symptomatology across the full spectrum of PTD factors; whereas, the image/adrenergic-based group demonstrated lower distress related to *Negative Alterations in Cognitions and Mood* (Criterion D) subgroup (i.e., areas of cognitive-focused distress), and the cognitive-focused group displayed lower distress on two items related to imagery-based distress (i.e., flashbacks and deficits in recall of aspects of trauma memories). These observations were viewed as a byproduct of mixed PTD presentation being, by definition, representative of a merger of both cognitive-focused and image/adrenergic-based distress. As such, the cumulative effect of a broader symptomatology impacting a greater scope of one's life may result in higher levels of distress. Similarly, assuming our purpose-driven theory of substance misuse, a broader symptomatology with multifaceted pattern of distress would prompt a multifaceted substance use strategy (i.e., polysubstance use).

PTD Phenotype's Link to DoC

A wealth of research has found various links between PTD and substance misuse (e.g., Gilpin & Weiner, 2017; Kearns et al., 2019; Ullman, 2016). For this study, in the case of DoCs and the predictive capability of our various phenotypes (i.e., H3), phenotype demonstrated significant links to DoC selection. We found that report of a mixed presentation of PTD

predicted polysubstance use, and determined that membership in the cognitive-focused PTD group predicted selection of alcohol and/or benzodiazepine as DoC. These findings seem to be supported by prior research by Brotchie and colleagues (2004) noting greater schema-level distortion (i.e., cognitive-focused distress) for alcohol users than for combined alcohol and opiate (i.e., polysubstance) users, as well as for opiate-only users. Primary cannabinoid and/or opiate use was predicted by membership in the image/adrenergic PTD, which was also supported by prior research (e.g., Bremner et al., 1996; Passie et al., 2012). In particular, Bremner and colleagues (1996) found marijuana use to be motivated by attempts to manage hyperarousal (i.e., adrenergic) features of PTD.

Limitations

Certain limitations to the current study should be considered. First, we enrolled an extremely homogenous sample of largely straight White males. This was an anticipated, frequently encountered, and generally unavoidable aspect of carrying out research in the local area, given the general demographic make-up of local inhabitants. Given noted culturally-based differences in index trauma (e.g., Alcantara et al., 2012) and peri- and post-traumatic responding (e.g., Alcantara et al., 2012; Ghafoori et al., 2012; Koo et al., 2016; Triffleman & Pole, 2010), current results may look different for racial and ethnic minority individuals. Similarly, significant variations in pre- and post-traumatic factors have been detected for sexual minority individuals (e.g., Roberts et al., 2012; Triffleman & Pole, 2010). Thus, our limited sample demographics likely significantly reduces generalizability of our study findings.

Furthermore, in line with what has been previously noted in the research literature concerning biological sex and trauma responses (e.g., Kessler et al., 1995; Najavits et al., 1997; Olff, 2017), our preliminary analyses showed significant differences in both PTD and comorbid

substance use based on participants' sex. Females reported significantly greater PTD severity and displayed more trauma-related shame than did males, as supported by prior work (e.g., Benetti-McQuoid & Bursik, 2005; Gilpin & Weiner, 2017). Females also endorsed greater misuse of substances other than alcohol, which is supported by findings from Danielson and colleagues' (2009) study of young adults; while, as noted by Gilpin and Weiner (2017), males demonstrated a greater propensity for misuse of alcohol. Interestingly, males also demonstrated greater trauma-related guilt (e.g., Benetti-McQuoid & Bursik, 2005), which was inversely related to both PTD and trauma-related shame. This finding may reflect the complicated relationship between shame, guilt, and trauma that accounts for mixed findings throughout the literature (see Crocker et al., 2016). However, as noted earlier, given the large disparity between the number of females to males, we were unable to control for sex in our analyses.

Additionally due to small sample size, we were unable to fully examine both of our hypothesized phenotypes as two of our primary comparison groups (i.e., the image/adrenergic-based PTD and primary cannabinoid/opiate use groups) had considerably lower membership than the others. Unlike the considerable variance anticipated between the mixed presentation group and both the cognitive-focused and image/adrenergic-based phenotypes, cognitive-focused and image/adrenergic-based groups were predicted to be closely related in terms of overall PTD severity, with anticipated significant variations between specific individual PTD items, and possibly rising to the level of significant variability at the PTSD Criterion level (e.g., *Negative Alterations in Cognitions and Mood* or *Hyperarousal and Reactivity* subgroups). However, despite recruiting enough participants for overall power, relatively few participants selected either image/adrenergic-based presentation of PTD or primary cannabinoid/opiate use as DoC. This severely reduced power and our ability to detect significance for these groups during certain

analyses. Therefore, observed nonsignificance between groups may be a result of limited statistical power rather than a validation of between-group equivalence. However, given that these two groups were hypothesized to be linked, the fact that we have seen relatively small numbers of participants endorsing membership in one or both may speak to the fitness of our model, in and of itself.

Another possible limitation is that, although all participants endorsed trauma and lingering distress, not all appeared to fully meet threshold for PTSD, according to the PCL-5. Trauma-related guilt and shame measures were included due to the realization that items on the PTSD Checklist might not be representative of all facets of PTD. Additionally, given that the PCL-5 is not considered to be a diagnostic tool, it is possible that some participants identified on this measure as being subthreshold, might appear as more symptomatic on a more sensitive diagnostic interview. Therefore, we defaulted to inclusivity at the possible risk of reducing our ability to observe more robust contrasts between groups. Had all been PTSD positive, our findings might have identified greater significance and connection among our hypothesized factors; or, possibly, an all PTSD population might have reduced or eliminated the findings we have noted.

Finally, considering biases commonly found in research, the principal investigator for this study also served as the study's interviewer/evaluator, which could have influenced participants (i.e., acquiescence bias) through various unintended verbal and non-verbal signals or actions. Additionally, given that trauma-related outcome measures (i.e., PCL-5, TRGI, TRSI) were administered prior to all participants' selection of presentation of PTD (i.e., phenotype), question-order bias may have impacted selection. We attempted to counter these possibilities by inclusion of open-ended questions during the interview portion of data collection to allow for

participants' subjective descriptions of experienced PTD and how their chosen DoC relates to alleviation of trauma-related distress. However, as we did not stagger the order of questioning, we cannot be certain that bias was not introduced.

Future Research Directions

As discussed in the previous section, multiple factors were not fully explored due to disparate or limited membership in the comparison groups. In particular, future research should focus on examining the variability inherent for factors such as race and ethnicity, biological sex, and sexual orientation. All of these factors, individually and intersectionally, may influence PTD presentation and DoC selection.

The existence of a cognitively-driven (i.e., prefrontal cortex hyperactivity triggering acute HPA-axis overresponding) phenotype of PTD was an underlying, but untested, hypothesis for this study. We were unable to test this hypothesis directly due to the significant cost and logistical limitations associated with neurological research (e.g., financial cost, access to neuro-imaging equipment). Therefore, future research avenues should involve actual brain imaging to explore level of prefrontal cortex activity occurring in relation to various phenotypes. As the current study findings are merely observed clues that may inferentially point to possible physiological mechanisms underlying variations in PTD presentations, neuroimaging methods are necessary to empirically determine the existence of any model of PTD in addition to the conditioned-fear model.

Similar to the self-medication hypothesis (see Khantzian, 1997), with the intersection between PTD and comorbid substance misuse, we have conceptualized many cases of substance misuse to be purposeful, albeit maladaptive, attempts to moderate the negatively-valenced effects of trauma rather than a hedonistic drive to chronic intoxication. Much of the prior research,

previously cited, reported purpose-driven links between a specific substance and an individual element of PTD, such as “marijuana use coping motives for sleep problems” (i.e., Bonn-Miller et al., 2010), or broadly examined the comorbidity between PTSD and opiate use disorder (e.g., Fareed et al., 2013). However, recent investigation by Kearns and colleagues (2019) appeared to support our proposal that combinations of substances (i.e., polysubstance) used, such as conjoint alcohol and marijuana use, may be attempts to purposefully self-medicate a broad symptomatology (e.g., mixed PTD presentation) with varied underlying physiological mechanisms simultaneously or intermittently engaged. The researchers (Kearns et al., 2019) also seemed to align with ours and others’ (e.g., Friedman, 2015) advocacy for continued exploration into these multifaceted diagnoses and complex relationships.

Finally, as noted previously, two of our main examination groups (i.e., image/adrenergic-based PTD and primary cannabinoid/opiate use) contained significantly reduced membership. While it is possible that most people who experience PTD experience either cognitive-focused or a mixed presentation, and that most are either primary alcohol/benzodiazepine or polysubstance users rather than only cannabinoid and or opiate use, continued exploration of our hypotheses with a much larger sample population and adequate subsample sizes is needed.

Clinical Implications

Further elucidation of varied internal structure and symptomatology inherent to PTD, and how these nuanced variations may differentially affect or be affected by other related factors and comorbidities, is important for both accurate assessment and effective referral to treatment of best fit (e.g., Ehlers et al., 2010; Grunert et al., 2007; Rizvi et al., 2009). In the case of PTD phenotypes and DoC, the predictive and diagnostic capacity is increased greatly by greater understanding of the interplay between these two data points. Knowledge that an individual

beginning to struggle post trauma with image/adrenergic-based distress may have increased susceptibility for cannabinoid and/or opiate misuse allows for a more targeted preventative approach. An understanding that observation of alcohol misuse may point to cognitive-focused (possibly prefrontal cortex/cognitively-driven) PTD, could allow for better treatment outcome via effective prediction and prescription of a trauma-focused treatment of best fit involving cognitive processing and restructuring. Alternatively, observation of image/adrenergic-based distress may better warrant and direct referral to an exposure-based intervention as treatment of best fit. In this capacity, a more individualized approach to therapeutic planning could prove to be a significant treatment multiplier, potentially drastically reducing non-response rates in an environment (e.g., trauma and substance misuse recovery) in which often the first opportunity to deliver a treatment may be the only opportunity afforded us.

Examination of various phenotypes involved in PTD may be of great importance for our understanding of the neurological mechanisms involved in how individuals respond both psychologically and physiologically to traumatic experiences. Therefore, this exploratory study conducted an initial examination as a first step at developing inferential foundations from which to build a greater knowledge base for all levels and facets of PTD.

Conclusions

Despite the limitations noted above, the hypothesized PTD phenotypes have shown considerable between-group variability among many of the PTD and substance misuse factors. In particular, analysis of variance and chi-square tests of independence detected distinct differences between groups, generally supporting our hypothesis of the existence of varied phenotypes of PTD, namely appearance of cognitive-focused and image/adrenergic-based phenotypes, additional to the traditional “mixed” presentation. Additionally, multinomial logistic regression

displayed significant predictive capability of phenotype in reference to DoC. Specifically, as hypothesized, variation in PTD phenotype was predictive of DoC selection, with (a) cognitive-focused phenotype related to alcohol/benzodiazepine as DoC (b) image/adrenergic-based phenotype related to cannabinoid/opiate as DoC, and (c) mixed presentation aligning with polysubstance use. Otherwise, variations in DoC endorsement (i.e., substances endorsed, rates of endorsement within each group) appeared to also mirror our hypotheses proposing varying phenotypes. While considerable work remains to validate these findings and further explore these interactions, this exploratory study adds to the literature by providing evidence for the existence of varying phenotypes of PTD and elucidating the links between PTD and DoC selection.

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Appendix

Table 1

Preliminary Analysis of Dependent Variables by Participants' Biological Sex (N = 177)

	Female (n = 62)		Male (n = 115)		t	p
Variables	*M*	*SD*	*M*	*SD*		
Posttraumatic distress	46.94	17.90	31.64	17.38	5.53	.000
Reexperiencing (Criterion B)	9.79	4.74	6.47	4.04	4.90	.000
Avoidance (Criterion C)	4.98	2.39	3.50	2.40	3.94	.000
Alteration in cognitions and mood (Criterion D)	17.15	7.18	12.13	7.69	4.24	.000
Hyperarousal (Criterion E)	15.02	6.71	9.55	6.00	5.55	.000
Trauma-related guilt	83.81	25.98	109.92	24.69	6.59	.000
Trauma-related shame[a]	32.66	22.55	16.34	17.88	--	--
Alcohol misuse[a]	14.61	13.77	20.70	10.60	--	--
Non-alcohol substance misuse[a]	22.05	16.85	8.43	12.64	--	--

Note. The following assessments were used to measure each of the constructs above: The PTSD Checklist for DSM-5 (PCL-5) for posttraumatic distress, Trauma-Related Guilt Inventory (TRGI), Trauma-Related Shame Inventory, Alcohol Use Disorders Identification Test (AUDIT), and Drug Use Disorders Identification Test (DUDIT) for non-alcohol substance misuse.

[a] Assumption of homoscedasticity not met by Levene's statistic, $p < .05$. Unequal variances t-test showed significance for the TRSI [$t(103) = 4.92$, $p < .001$], AUDIT [$t(101) = 3.27$, $p = .003$], and DUDIT [$t(99) = 5.57$, $p < .001$]

Table 2

Means, Standard Deviations, and Correlations among Dependent Variables

Variables	*M*	*SD*	1	2	3	4	5
1. Posttraumatic distress	37.00	18.98	--	--	--	--	--
2. Trauma-related guilt	100.77	28.02	-.74*	--	--	--	--
3. Trauma-related shame	22.06	21.08	.73*	-.82*	--	--	--
4. Alcohol misuse	18.56	12.13	-.13	.07	-.07	--	--
5. Non-alcohol substance misuse	13.20	15.63	.43*	-.41*	.36*	-.53*	--

Note. * $p < .001$. $N = 177$. The following assessments were used to measure each of the constructs above: The PTSD Checklist for DSM-5 (PCL-5) for posttraumatic distress, Trauma-Related Guilt Inventory (TRGI), Trauma-Related Shame Inventory (TRSI), Alcohol Use Disorders Identification Test (AUDIT), and Drug Use Disorders Identification Test (DUDIT) for non-alcohol substance misuse.

Table 3

Comparison of Hierarchical Cluster Analysis Four-Cluster Solution with Hypothesized Phenotype Variations

HCA Solution	Hypothesized Variations	
	H1 Cognitive-focused	H2 Image/Adrenergic-based
Cluster 1: Adrenergic Distress and Avoidance		Criterion B
B1. Intrusive memories		B1. Intrusive memories
B4. Emotional distress		
B5. Physical reactions		
C1. Internal avoidance		
C2. External avoidance		
Cluster 2: Ruminative Distress	Criterion D	
D3. Blaming	D3. Blaming	
D4. Negative feelings	D4. Negative feelings	
D2. Negative beliefs	D2. Negative beliefs	
D5. Anhedonia		
D6. Feeling distant		
D7. Difficulty w/ positive emotion		
E6. Sleep disturbance		
Cluster 3: Adrenergic Distress		Criterion E
E2. Risk taking	E2. Risk taking	
E5. Difficulty concentrating		E5. Difficulty concentrating
E3. Hypervigilance		E3. Hypervigilance
E4. Increased startle		E4. Increased startle
E1. Irritability		E1. Irritability
Cluster 4: Imagery-Based Distress		Criterion B
B2. Nightmares		
B3. Flashbacks		
D1. Difficulty remembering		

Note. $N = 177$. Individual items are identified by Criterion and individual item number (e.g., "B1" = item 1 of Criterion B from the PCL-5).

Table 4

Means, Standard Deviations, and ANOVA for Dependent Variables by Phenotype

	Cognitive (n = 80)		Image/ Adrenergic (n = 22)		Mixed (n = 63)		*F(2, 162)*	*p*
Variables	*M*	*SD*	*M*	*SD*	*M*	*SD*		
Posttraumatic distress	33.85	18.68	33.45	18.92	42.03	19.60	3.66	.028
Reexperiencing (Criterion B)	7.03	4.31	7.27	5.10	8.65	4.86	2.30	*ns*
Avoidance (Criterion C)	3.69	2.59	3.68	2.57	4.54	2.42	2.23	*ns*
Alteration in cognitions and mood (Criterion D)	12.96	7.70	10.82	7.61	15.89	7.93	4.35	.014
Hyperarousal (Criterion E)	10.18	6.84	11.68	6.67	12.95	6.80	2.95	*ns*
Trauma-related guilt	103.74	28.92	101.91	28.37	96.83	27.87	1.06	*ns*
Trauma-related shame	20.26	20.88	17.95	19.90	26.57	22.44	2.08	*ns*
Alcohol misuse	19.56	12.01	18.05	12.11	16.95	12.25	0.83	*ns*
Non-alcohol substance misuse	11.38	15.61	16.09	15.61	14.57	16.42	1.12	*ns*

Note. The following assessments were used to measure each of the constructs above: The PTSD Checklist for DSM-5 (PCL-5) for posttraumatic distress, Trauma-Related Guilt Inventory (TRGI), Trauma-Related Shame Inventory, Alcohol Use Disorders Identification Test (AUDIT), and Drug Use Disorders Identification Test (DUDIT) for non-alcohol substance misuse.

Table 5

Summary of Multinomial Logistic Regression Predicting Drug-of-Choice by Phenotype

Drug-of-Choice[a]		*B*	*SE*	Wald	*df*	*p*	e^B
Alcohol/ Benzodiazepine	Intercept	-0.24	.26	0.83	1	.363	
	Cognitive-Focused	1.38	.38	12.92	1	.000	3.96
	Image/ Adrenergic-based	-0.17	.59	0.08	1	.777	0.85
	Mixed	0[b]					
Cannabinoid/ Opiate	Intercept	-2.11	.53	15.89	1	.000	
	Cognitive-Focused	1.58	.66	5.68	1	.017	4.85
	Image/ Adrenergic-based	1.86	.73	6.47	1	.011	6.42
	Mixed	0[b]					

Note. R^2 = .16 (Nagelkerke). Model $\chi^2(4) = 24.33$, $p < .001$. e^B = exponentiated *B* (i.e., odds ratio).

[a] The reference category is: Polysubstance use.

[b] This parameter is set to zero because it is redundant.

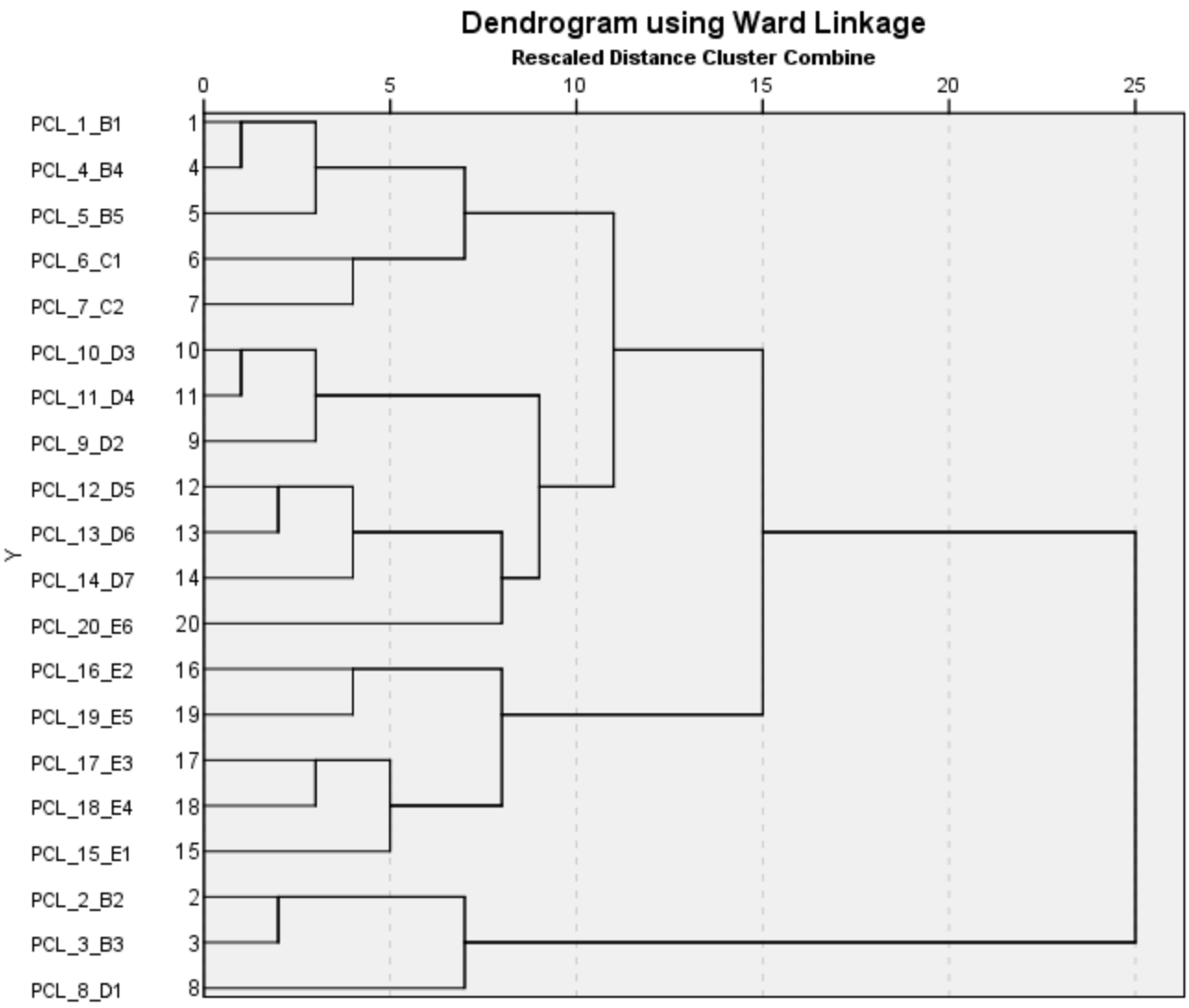

Figure 1: Hierarchical Relationship between Individual PCL-5 Items

Informed Consent Statement

Trauma-Related Symptoms among Individuals in Substance Use Treatment

Overall Study Principal Investigator (PI): D. Allen Donahue, MA
Co-Investigator University of Tennessee: Gina Owens, PhD

INTRODUCTION

You may be eligible to take part in a research study being conducted by researchers from the University of Tennessee – Knoxville (UTK). The study is being conducted at Cornerstone of Recovery (CoR) and will not add any time to your total time in treatment. This form gives you important information about this research study and your possible participation. Please take time to review this information carefully and talk to the researcher(s) about any questions you may have, to ensure that you understand what the study is about.

INFORMATION ABOUT PARTICIPANTS' INVOLVEMENT IN THE STUDY

You are being asked to take part in this study because you are currently receiving treatment for substance abuse (i.e., alcohol and or drugs), and you have also indicated having experienced at least one previous potentially traumatic event in your life. The purpose of this study is to look at distress that may occur after traumatic experiences. In particular, the research team is interested in how your thoughts, feelings, and behaviors might have been impacted and changed by your experiences, as well as how this impact may also be linked to substance abuse and which substances might be most appealing to you. With this goal, the research team hopes to recruit participants to help add to our knowledge and understanding of the various factors involved in posttraumatic distress and how trauma may be linked to substance abuse.

Your participation in this study will take approximately 90 minutes. If you consent to participate, you are consenting to the use of data you have already provided to CoR during your time in Assessment and Orientation (A&O), such as intake information (e.g., general demographics, trauma history) and assessment data from some of the questionnaires (i.e., PCL, AUDIT, DUDIT) you will be completing as part of CoR's normal battery of assessments. You also will be provided a study packet, which includes three questionnaires that take approximately 20-30 minutes to complete. Finally, you will take part in a brief interview with a member of the research team, during which you will be asked a few additional questions concerning how you have responded to trauma and about your substance use.

RISKS

Some discomfort is possible when thoughts, feelings, and memories of traumatic events are explored; however, as this study is being carried out at CoR, treatment staff are on hand should you experience distress and want therapeutic support while completing the study measures and brief interview. Otherwise, with the handling of medical and research records there may be the possibility of a breach of confidentiality. However, every effort is being made to protect your privacy; as such, your name will not be entered into the research documents. The research team is carefully trained on how to handle and protect your private information and strictly controls access to study data.

BENEFITS

There is no anticipation of direct benefit to you from taking part in this study. However, it is hoped that information gained from this study will improve assessment and treatment approaches for others struggling with substance abuse and posttraumatic distress.

CONFIDENTIALITY

Under federal privacy regulations, you have the right to determine who may review or use your personal health information (also called "protected health information" or PHI). This includes information that can identify you. For example, it can include your name, phone number, birthdate and medical record

number. The PHI that researchers will receive, review and use in this research study may include information such as:

- Demographic information;
- Information from assessments that you have completed during the initial intake phase; and,
- Information provided by you about your trauma history and past alcohol or substance use.

If you choose to be in this research study and sign this consent form, you are giving your permission to Cornerstone of Recovery to share your PHI with the research team at The University of Tennessee, and for the research team to review and use your PHI for the research study. Your PHI, which might identify you, may also be shared with or used by the Institutional Review Board (IRB) at The University of Tennessee, Knoxville. However, your PHI will only be used and/or given to others:

- To do the research described in this consent form;
- To study the results of the research; and,
- To see if the research was done correctly.

Your PHI will be used until the research ends and all required monitoring has been completed. You may withdraw or take away your permission to use and share your PHI at any time. You can do this by sending written notice to the researchers listed on this consent form. When you withdraw your permission, no new PHI will be shared or used after that date. However, information that has already been collected may still be used to complete the research. You have the right to see and copy your PHI that is shared or used in this research study. However, in order to complete the research, your access to this PHI may be temporarily suspended while the research is in progress. When the study is completed, you will be able to access this information.

If you do not permit use of your PHI you cannot participate in this research study. If you withdraw your permission for use of your PHI, you may not be able to stay in the study. However, your decision to permit, not permit, or withdraw your permission for use of your PHI will not affect your relationship with Cornerstone of Recovery or the services you receive in any way.

Otherwise, all information that is collected will be presented as anonymous group data, meaning that **you will NOT be personally identified**. However, complete confidentiality cannot be promised, because in some cases it may be necessary to provide information regarding your current health and well-being to your assigned CoR treatment program and staff. For example, if you indicate you have thoughts of harming yourself or others the researchers will want to immediately report this to your treatment team to make sure you receive the needed assistance in ensuring your safety.

FUTURE RESEARCH

Your information will not be used or shared with other researchers for future research, even if identifiers are removed.

CONTACT INFORMATION

If you have any questions or comments about this research project, please contact the PI, Allen Donahue at ddonahu7@vols.utk.edu or his faculty advisor, Dr. Gina Owens, at gowens4@utk.edu or 865-974-2204.

This research protocol has been reviewed and approved by the University of Tennessee Institutional Review Board. If you have questions about your rights as a participant, please contact the University of Tennessee Office of Research Compliance Officer at utkirb@utk.edu or 865-974-7697.

PARTICIPATION

Your decision to take part in this study is completely voluntary. No one has coerced or intimidated you into taking part in this study. You are participating because you want to. The Principal Investigator or another member of the research team has answered any and all questions you have about: this study, your participation in the study, and the procedures involved in the study.

You may change your mind and withdraw this consent at any time. If you do so, we will discontinue your participant in this study and it will not affect your eligibility for care or any other benefits to which you are entitled. Should you choose to withdraw, please discuss this with a study staff member.

CONSENT

I have read the above information. I have received a copy of this form. I agree to participate in this study.

Participant's Name (printed) _____

Participant's Signature _____ Date _____

Vita

Allen Donahue is a lifelong member of the military community. He was born at Fort Bragg, North Carolina, to CW5 (Ret.) Kenneth Allen “Doc” Donahue and Carmen W. Mendonsa-Donahue, both also “Army brats.” In early 1997, he enlisted in the United States Army Military Intelligence Corps and served honorably in various capacities until late 2005, along the way earning Associate of Arts degrees in Russian Language and Russian Area Studies from Monterey Peninsula College and The Defense Language Institute in Monterey, California. Following active military service, he remained in San Antonio, Texas, and completed Bachelor of Arts degrees in Psychology and Criminal Justice at The University of Texas at San Antonio in 2009. After graduation, he began working as a Research Associate and eventual Protocol Coordinator in behavioral science research with the STRONG STAR Multidisciplinary PTSD Research Consortium at The University of Texas Health Science Center at San Antonio while also working toward a Master of Arts in Community Counseling from The University of Texas at San Antonio. Upon completion of his Masters in 2012, Allen entered the Counseling Psychology Doctoral Program at The University of Tennessee in Knoxville in order to work and study with Dr. Gina Owens in her Military Stress and Health Research Lab. Allen earned a concurrent Master of Arts in Psychology in 2015, passed his comps in 2018, and successfully completed the pre-doctoral internship at the G.V. (Sonny) Montgomery VA Medical Center in Jackson, Mississippi in July of 2019. Therefore, completion of this work is the final step to earning the Doctor of Philosophy in Counseling Psychology and joining the Central Alabama Veterans Healthcare System as a Graduate Psychologist.