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RUNNING HEAD: PTSD Emotion LPA

Latent Emotion Profiles of PTSD and Specific Emotions Predicting Differential Therapy
Outcomes in a Dismantling Study of Cognitive Processing Therapy

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Data availability statement: The data that these findings are based upon are available on request by emailing Dr. Brian Smith (Brian.Smith12@va.gov) at the Women's Health Science Division, National Center for PTSD. They are not publicly available by VA research policy and for privacy-based/ethical concerns.

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Abstract

Posttraumatic stress disorder (PTSD) is theoretically maintained by avoidance of emotions elicited from trauma-related beliefs. Whether PTSD symptom profiles and specific emotions predict treatment response is unknown. This secondary data analysis examined: a) whether individuals with PTSD can be sub-classified based on symptom clusters and specific emotions, and b) if these subgroups predict differential responses to cognitive versus exposure-based PTSD interventions. Women with physical or sexual assault-related PTSD were randomized to CPT (cognitive processing therapy elements only), CPT with written accounts (CPT+A), or written accounts (WA) only (n=150). Participants completed baseline measures of PTSD, state anxiety, internalized anger, externalized anger, shame, and guilt, and weekly PTSD measures during and 6 months after treatment. Latent profile analyses revealed four subgroups: *low symptoms and emotions; moderate-high reexperiencing, low internalized emotions* (i.e., moderate-high reexperiencing, moderate avoidance/hyperarousal/guilt, low shame/internalized anger/anxiety); *low reexperiencing, moderate emotions* (i.e., low re-experiencing, moderate avoidance/hyperarousal/guilt, moderate other emotions); and *high symptoms and emotions* (high symptoms and emotions except moderate externalized anger). The high symptom and emotion subgroup experienced greater PTSD symptom improvements in cognitive conditions than WA. Other groups did not exhibit differential change across conditions. Cognitive interventions may be well-suited for severe PTSD with high self-directed emotions.

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1. Introduction

One third to half of participants continue to meet diagnostic criteria for posttraumatic stress disorder (PTSD) following gold standard treatments such as Cognitive Processing Therapy (CPT; Asmundson et al., 2019; Resick et al., 2008; Resick et al., 2016, Resick et al., 2017). Tailoring interventions by identifying meaningful subgroups of PTSD presentations that are more or less likely to benefit from specific PTSD interventions may optimize treatment outcomes.

Latent profile analysis (LPA) can identify subgroups of people based on specific characteristics. LPAs of PTSD symptoms generally suggest that subgroups are stratified based on severity (e.g., Bondjers et al., 2018; Campbell et al., 2020; Chung et al., 2008; Durham et al., 2020; Hebenstreit et al. 2014, 2015; Murphy et al., 2019; Rahman et al., 2018; Sripada et al., 2020; Steenkamp et al., 2012). However, individuals with PTSD may not only vary based on their symptomatology, but also the specific emotions that they experience. Although PTSD was originally conceptualized as a fear-based disorder (Foa & Kozak, 1986), it involves disruptions in many negative emotions (i.e., emotions that involve both some subjective distress and unpleasant experience; Watson et al., 1988), such as sadness, shame, guilt, anger, and fear (Badour et al., 2017; Dalgleish & Power, 2004). Each of these emotions entail their own cognitive, behavioral, and experiential elements (Lench et al., 2011), which may obstruct or facilitate treatment success, or require distinct treatment elements to target (Badour et al., 2017; Brewin et al., 1996; Dalgleish & Power, 2004). Indeed, several basic emotion scientists show that distinct appraisals lead to distinct emotions states (e.g., perceptions of threat leading to fear, perceptions of loss leading to sadness; e.g., Smith & Lazarus, 1993; Smith et al., 1993). Accordingly, Dalgleish and Power (2004) suggest that PTSD involves “emotion-specific

components,” wherein the specific appraisals of traumatic content lead to distinct emotional states, which may require customized treatment approaches. Resick and colleagues (2016) similarly emphasize the role of appraisals in leading to divergent emotional experiences, with implications for PTSD recovery or lack thereof. Specifically, these authors suggest that some negative emotions can arise directly in response to trauma (i.e., “natural emotions”) or as a result of unhelpful beliefs (i.e., “manufactured emotions”). Manufactured emotions are theorized to impede PTSD recovery by obstructing natural emotions (Resick et al., 2016). Together, these theories suggest that PTSD presentations may vary substantially based on the specific emotions that occur with it, and that such specific emotions may help to meaningfully organize the heterogeneity of PTSD.

LPA studies have examined subtypes of PTSD symptoms and specific emotions, and have mostly identified subclasses that correspond to symptom severity (e.g., Contractor et al., 2015; Jongedijk et al., 2019, 2020). However, these LPAs included anxiety but not other emotions (e.g., anger at self, shame, guilt). LPA studies that have investigated additional specific emotions and PTSD symptoms suggest that anger (Armour et al., 2014) and guilt (Smigelsky et al., 2019) also covary with PTSD symptom severity. However, another study that conducted an LPA with guilt, anger, PTSD symptoms, complex PTSD (cPTSD) symptoms, and borderline personality disorder (BPD) symptoms in a community sample of trauma-exposed individuals revealed four classes: two classes exhibited low and high rates of endorsement of all symptoms including anger and guilt, respectively, and two others had high and moderate PTSD symptoms, respectively, but low anger and guilt (Saraiya et al., 2021). This study suggests that, at least in some samples, anger and guilt may not fully covary with PTSD severity. However, emotions other than anger and guilt (e.g., shame, anxiety) were not examined.

Finally, one study examined a wide range of peritraumatic emotions in trauma-exposed undergraduate students and suggested that three subgroups stratified on the basis of having generally high, moderate, and low emotions, while three additional subgroups exhibited unique patterns of emotions: low shame and guilt and elevated sadness; high anger and guilt but low shame and disgust; and low guilt. Generally, subgroups with higher anger, shame, and guilt had higher PTSD severity (Lancaster & Larsen, 2016). Although that study focused on peri- rather than post-traumatic emotions, findings again suggest that specific emotions may uniquely covary among trauma survivors, and that anger, shame, and guilt may be particularly related to PTSD severity. However, because PTSD symptoms were not included in this LPA, their covariance with these specific emotions remains unclear. This study also did not include different forms of anger that vary based on whether it is directed at self (i.e., internalized) or others (i.e., externalized). Further, both the Lancaster and Larsen (2016) and Saraiya et al. (2021) studies were not conducted in individuals with diagnoses of PTSD, which dilutes conclusions that can be drawn about whether anger and guilt covary with PTSD symptoms. LPAs in a sample diagnosed with PTSD with a broader range of emotions— including internalized and externalized anger— are required to better understand whether specific emotions may help to organize the considerable heterogeneity of PTSD.

Given the theorized role of different emotions in maintaining PTSD (e.g., Dalgleish and Power, 2004; Resick et al., 2016), individuals with PTSD profiles with specific emotions may also exhibit unique treatment responses. Indeed, higher anger and guilt-related beliefs at baseline predict poorer responses to PTSD interventions (Kaczurkin et al., 2016; Scher et al., 2017). However, the extent to which specific *subgroups* of emotions and PTSD symptoms inform differential PTSD treatment response is unknown. Identifying such meaningful subgroups of

emotions and PTSD could inform treatment matching by identifying individuals with certain constellations of emotions that may be more or less suited to certain interventions.

Evidence-based PTSD treatments vary in the extent to which they directly target trauma-related beliefs that theoretically maintain some specific emotions but not others. For example, CPT (Resick et al., 2008; 2016) with and without a written account of the traumatic event (CPT+A and CPT, respectively) focuses largely on directly challenging trauma-related beliefs to alleviate the emotions that arise from them. Conversely, exposure-based treatments, such as Prolonged Exposure (PE; Foa et al., 2019) and Written Exposure Therapy (Sloan et al., 2012) focus predominantly on modifying trauma-related beliefs indirectly through inhibitory learning via extended repetitions of the trauma-related memories and cues.

Exposure-related theory and research suggests that a primary mechanism through which exposure results in reductions in distress is through the development of new, non-threat-based associations and the enhancement of their retrievability (e.g., discovering that remembering a painful memory is not, in fact, dangerous; Craske et al., 2008; 2014). By this logic, exposure interventions may be particularly effective in reducing anxiety/fear-based profiles of PTSD because remembering traumatic memories in the absence of actual danger is likely to violate the threat-based appraisals that lead to these specific emotions (e.g., Smith & Lazarus, 1993; Smith et al., 1993). Other emotions such as shame, guilt, internalized anger, or exaggerated or inappropriate anger at others (which may lead to externalized anger) may be elicited by appraisals that are less readily corrected through exposure to traumatic stimuli alone without additional cognitive intervention (e.g., Ellsworth & Tong, 2006; Smith & Lazarus, 1993; Smith et al., 1993; Tracy & Robins, 2006). Indeed, given that cognitive interventions directly target trauma-related beliefs, one may postulate that they are better suited for presentations that involve

emotions other than fear/anxiety, because the appraisals that drive these emotions may or may not be automatically violated through exposure alone. However, research has shown that, as individual emotions, guilt and anger do not differentially moderate outcomes across distinct PTSD interventions (Kaczurkin et al., 2016; McLean et al., 2019; Scher et al., 2017; Rizvi et al., 2009). Moreover, cognitive and exposure-based interventions do not exert differential effects on emotions such as anger, anxiety, and shame, and guilt (Resick et al., 2008) although, in one study, a cognitive PTSD intervention resulted in greater improvements in some cognitive elements of guilt than exposure-based one (Resick et al., 2002). However, no research has examined whether individuals with specific combinations of these emotions and PTSD symptoms exhibit unique responses to PTSD interventions, possibly due to an interaction between the mechanisms of action of the intervention and the types of emotions they experience.

To address these gaps in the literature, we used secondary data from a dismantling study of CPT to identify: a) whether there are distinct subgroups of PTSD symptom clusters and emotional experiencing (i.e., anxiety, internalized anger, externalized anger, shame, guilt), and b) whether these subgroups predict distinct responses to cognitive (CPT), written narrative-based (i.e., written narrative accounts; WA), or combined cognitive and written narrative-based interventions (CPT+A; Resick et al., 2008; 2016). The exploratory nature of LPA makes postulating hypotheses about specific emotion profiles unfeasible. However, based on basic emotion research (e.g., Lazarus, 1991; Smith et al., 1993; Smith & Lazarus, 1993; Tangney et al., 2007), we predicted that individuals with elevations in emotions other than anxiety (i.e., guilt, shame, anger at self, anger at others) would benefit more from cognitive (i.e., CPT and CPT+A) than written narrative-based interventions.

2. Materials and Methods

Data analyzed in the present study were collected as part of a three-arm dismantling randomized controlled trial comparing what was previously “full CPT” (now CPT+A; Resick et al., 2008; 2016) with two of its components: cognitive therapy only (now standard CPT) or written accounts (WA). Study procedures are described in detail elsewhere (Resick et al., 2008) and are presented here in brief. All procedures were approved by University of Missouri-St. Louis and VA Boston Healthcare System Institutional Review Boards. This secondary analysis was not pre-registered, although the parent trial was registered at clinicaltrials.gov (NCT00245232).

2.1. Participants

Women who experienced a sexual or physical assault; met criteria for PTSD; were at least 3-months posttrauma; and had a stabilized medication regimen (if any) were recruited from the St. Louis Metropolitan area using word of mouth, referrals from survivor-focused agencies, community clinics and clinicians, and advertisements in print media and fliers. Exclusion criteria included: intent to engage in suicidal behavior (i.e., suicidal intent); substance/alcohol dependence; current psychosis; illiteracy; or currently being stalked or in an abusive relationship. Of the 256 women assessed, 162 were eligible and randomized. Twelve women were later withdrawn for meeting exclusion criteria. Ultimately, the intent-to-treat sample consisted of 150 women randomized to CPT+A ($n = 53$), CPT ($n = 47$), or WA ($n = 50$).

The mean age of the sample was 35.4 years ($SD = 12.4$) and the mean number of education years was 13.8 ($SD = 2.8$). As reported in the parent manuscript, income was significantly lower in the CPT+A group than the other groups (79% with income < \$20,000/year in the CPT+A group, versus 42% and 46% in WA and CPT, respectively; Resick et al., 2008). The majority of the sample was White (62%) or African American (34%), with 4% from other

racial groups. On average, participants' most distressing (index) traumatic events occurred 14.6 years earlier ($SD = 14.4$), and 41% of individuals reported taking psychotropic medication. These variables did not differ across treatment conditions. The most common comorbidities assessed were major depressive disorder (50%) and panic disorder (20%).

2.2. Measures

2.2.1. Diagnostic and demographic assessments. The presence of a PTSD diagnosis was assessed at baseline via the Clinician Administered PTSD Scale (CAPS; Blake et al., 1995) based on the *Diagnostic and Statistical Manual of Mental Disorders-IV-TR* (DSM-IV; APA, 2001). Assessors had 100% agreement (Resick et al., 2008).

The Structured Clinical Interview for DSM-IV Axis I Disorders-Patient Edition (SCID; First et al., 1995) was used to assess for panic, major depressive, psychotic, substance use disorder, and suicidal ideation. Assessors had 90% agreement ($kappa = .87$; Resick et al., 2008).

2.2.2. LPA variables. To aid in the interpretability of subgroups, PTSD symptom clusters rather than individual symptoms were included in the LPA along with anxiety, internalized-anger, externalized-anger, shame, and guilt. PTSD symptom clusters were measured via the *Posttraumatic Diagnostic Scale* (PDS; Foa, 1995), a 49-item self-report scale assessing the frequency of PTSD symptoms per DSM-IV (APA, 2001) over the past 30 days. Items are rated from 0 to 3 can be summed into re-experiencing (5 items; $\alpha = .82$), hyperarousal (5 items; $\alpha = .63$), and avoidance (7 items; $\alpha = .75$) clusters and a total PTSD score (17 items; $\alpha = .88$). Baseline subscale scores were entered into the LPA, and total weekly PDS scores after baseline were included as the primary outcome variable. State anxiety was assessed with the state subscale from *State-Trait Anxiety Inventory* (STAI; Spielberger, 1970; $\alpha = .95$).

The state subscale of the STAI is a 20-item subscale that examines the extent to which people experience a range of anxiety-related words (e.g., tense, worried) in the present moment. Anger was assessed with the 8-item Anger In ($\alpha = .79$; internalized/suppressed anger) and 8-item Anger Out ($\alpha = .81$; externalized anger) subscales from *State-Trait Anger Inventory* (STAXI; Spielberger, 1988). These subscales ask participants to indicate the extent to which they hold in or suppress anger (e.g., “I boil inside, but I don’t show it”), or express anger towards others in a verbally or physically aggressive way (e.g., “I make sarcastic remarks to others”), respectively. Shame was assessed via the 25-item *Experiences of Shame Scale* (ESS; Andrews et al., 2002; $\alpha = .96$), which asks participants to what extent they have a range of shame-related experiences (e.g., “Have you felt ashamed of any of your personal habits?”) over the past year. Items on the STAI, STAXI, and ESS are all rated from 1 to 4. Guilt was assessed via the 4-item Global Guilt scale ($\alpha = .89$) of the *Trauma-Related Guilt Inventory* (TRGI; Kubany et al., 1996). The Global Guilt subscale of the TRGI provides information regarding the overall intensity of general guilt, with items rated from 0 to 4.

2.3. Treatment Conditions

Conditions are described more fully in Resick et al. (2008). All treatment conditions were manualized and involved 12 therapy hours delivered over 6 weeks. CPT+A involves learning about PTSD and systematically identifying and challenging unhelpful beliefs that may be maintaining it. Socratic dialogue and a progressive series of worksheets are used to challenge potentially unhelpful trauma-related beliefs. Clients are also asked to write a detailed account of the index trauma and read this account daily as homework between two early sessions of therapy. CPT is the same as CPT+A but without the written account component. WA involved two, 1-hour sessions focused on psychoeducation regarding PTSD, providing a treatment rationale,

instruction on how to write accounts of their trauma and Subjective units of distress (SUDs) ratings. Subsequent sessions were 2 hours and involved participants reviewing homework from the prior week, writing accounts of their trauma, and then debriefing with a therapist. SUD were monitored before and after these exercises, and participants were asked to read their written accounts aloud daily between sessions and record their SUDs before and after each reading. Cognitive-focused interventions were proscribed. As described elsewhere (Resick et al., 2008) treatment fidelity was good with clinician adherence and competence above 80% for all conditions.

2.3.1. Procedure. Following collection of informed consent and assessments, eligible participants were randomly assigned to one of the three conditions by the data manager. All emotion measures described above were completed at baseline. The PDS was administered at baseline, weekly during the 6 weeks of treatment, at a 2-week posttreatment point, and at a 6-month follow-up, yielding eight assessments.

2.4. Data Analyses

Descriptive analyses were conducted in SPSS version 27. Latent profile analyses (LPA) were conducted in MPLUS version 8 (Muthén & Muthén, 2017). LPA is an exploratory, person-centered approach to identifying groups within a sample (Ruscio & Ruscio, 2004). Muthén (2004) cautions that inter-item correlations above .6 may indicate excessive collinearity with indicators. We therefore first examined correlations between the emotions and PTSD subscales to assess for multicollinearity. Following recommended procedures (Nylund et al., 2007; Masyn, 2013), we estimated models with the robust maximum likelihood (MLR) estimator, successively increasing latent subgroups until additional subgroups did not improve model fit. We assumed a missing at random missing data pattern based on the assumption that missing data (i.e., drop out)

is likely related to observed data. Although this is not a fully testable assumption, MLR estimators are robust to its violation (Li & Stuart, 2019).

Model fit was assessed in accordance with the procedures outlined by Nylund et al (2007). Successive models were compared based on a combination of the adjusted Lo-Mendell-Rubin likelihood ratio test (LMR; Lo et al., 2001), Akaike Information Criteria (AIC; Akaike, 1987), Bayesian Information Criterion (BIC; Schwartz, 1978), sample size-adjusted Bayesian Information Criterion (SSABIC; Sclove, 1987), and entropy statistic (Ramaswamy et al., 1993). A non-significant LMR indicates that an additional subgroup does not add value to the model. A lower relative value on the AIC and SSABIC indicates superior model fit. Entropy represents the degree of certainty that participants are correctly classified into a subgroup and range from 0 to 1 (1 indicating a higher degree of certainty). Along with these criteria, theoretical consistency, interpretability (i.e., adequate number of people in each subgroup), and parsimony (i.e., non-overlapping class membership, fewest number of classes necessary to adequately describe the population) were also used to differentiate multiple fitting models (Nylund et al., 2007).

To estimate the trajectory of change in PTSD across and after treatment, we conducted an unconditional latent growth curve model of PTSD using the full maximum likelihood estimator, from session 2 to 6-month follow-up. We did not include pre-treatment PTSD in the growth model because it was included in the latent subgroups. In the parent study, Resick et al., (2008) found that a linear trajectory offered the best fit to the data. However, PTSD is known to have a curvilinear change course (Nishith et al., 2002). In the current study, growth models were estimated using structural equation modeling (SEM) which allows greater flexibility in modeling time than regression-based approaches. Consistent with the parent study, time was coded categorically as session number, but the timescale was freely estimated for maximum flexibility

in modelling time. To set the scale, the first two time points (i.e., sessions 2 and 4) were set to zero and one, respectively, and the last (i.e., 6-month follow up) was set to seven. Residual variances were freely estimated and allowed to correlate with adjacent timepoints. The model was evaluated using the AIC, BIC, chi-square goodness of fit test (χ^2), Comparative Fit Index (CFI; Bentler, 1990), and Root Mean Square Error of Approximation (RMSEA; Steiger & Lind, 1980). CFI values greater than .95 and RMSEA values of .01 and .05 represent excellent and good fit, respectively (MacCallum, et al, 1996).

We then conducted a mixture model using Nylund-Gibson et al's (2019) *manual three-step* approach wherein growth parameters from the unconditional model are estimated for each latent subgroup. To test whether trajectories of PTSD severity differed across subgroups, we conducted pairwise comparisons of the intercepts and slopes using the Wald test for parameter constraints. For each family of comparisons (i.e., intercept comparisons and slope comparisons) we corrected for multiple comparisons with the Benjamini-Hochberg procedure, which adjusted significance thresholds based on a False Discovery Rate (i.e., the likelihood of incorrectly rejecting a null hypothesis) of $Q = .10$ (Benjamini & Hochberg, 1995). Standardized within-group Hedges' g effect sizes of change in PTSD from session 2 to 6-month follow up were calculated for each latent subgroup as model-estimated change from session 2 to 6-month follow up, divided by the session 2 standard deviation (Feingold, 2009), with a *Hedges g* correction for small samples (Hedges, 1981). Finally, we added a time by treatment interaction to each latent subgroup trajectory which compared differential change in PTSD in the two cognitive conditions versus WA. For this interaction, CPT and CPT+A were both dummy coded as 1 and WA was coded as 0. Within-group Hedges' g effect sizes of change in PTSD were calculated for each treatment condition in the case of significant interactions.

3. Results

There was < 10% missing data for baseline measures in the LPA. Attrition of PTSD scores across treatment ranged from 23% (session 2) to 47% (session 12). Means, standard deviations, and correlations within and between baseline emotion and PTSD scores are in Table 1. There were no differences in baseline emotions or PTSD severity across treatment conditions. Subgroup membership did not differ by treatment condition and there was also no differential attrition across treatment groups or latent subgroups. Only one inter-item correlation approximated exceeding the recommended cutoff of .60 (Muthén, 2004; Hypervigilance and Avoidance = .60), suggesting collinearity was not problematic.

Latent profile models were specified for 2-6 subgroup solutions (See Table 2 for fit indices). The 6-subgroup solution had a non-replicated log likelihood estimate suggesting instability. The 5-subgroup solution demonstrated the best fit based on the lowest AIC and SSABIC (but not BIC) and the highest entropy. However, the fifth subgroup contained only four participants suggesting the 5-subgroup solution may have been overidentified. Therefore, the 4-subgroup solution was retained as the most parsimonious model. The profile plot of the 4-subgroup solution is in Figure 1. Means for emotion and PTSD subscales for each class are shown in Table 3. To help systematize subgroup naming, we use the term “low” to refer to emotion mean scores that approached $z = 0.5$ SD below the overall mean score for that symptom cluster/emotion, “moderate” to refer to symptom clusters/emotions that approached the overall mean score for the sample ($z = 0$) up to $z = 0.5$ SD above the mean, and “high” to refer to anything that fell above $z = 0.5$ SD of the mean. 19% of the sample was in Subgroup 1 (low symptom cluster severity and low emotions; LL; $n = 29$), which had the lowest endorsement probabilities for the sample across most symptom clusters and emotion subscales. 22% of the

sample was in Subgroup 2 ($n = 33$) and 37% of the sample was in Subgroup 3 ($n = 56$), both of which were characterized by moderate severity in several symptom clusters (avoidance and hyperarousal) and emotions (externalized anger and guilt), but differed in levels of re-experiencing and several internalized emotions (shame, internalized anger, and anxiety). Specifically, whereas Subgroup 2 (moderate-high re-experiencing symptom cluster severity, low internalized emotions; MHL) endorsed moderate to high severity in the re-experiencing symptom cluster (as well as moderate avoidance and hyperarousal), this subgroup had generally lower scores on several internalized emotions (shame, internalized anger, and anxiety) and moderately severe externalized anger and guilt relative to other classes. In contrast, Subgroup 3 (low re-experiencing symptom cluster severity, moderate emotions; LM) endorsed low severity in the re-experiencing symptom cluster (as well as moderate avoidance and hyperarousal) and generally moderate emotions. Finally, 21% of the sample was in Subgroup 4 (high symptom cluster severity and high emotions; HH; $n = 32$), which involved elevations in all symptom clusters and specific emotions, with the exception of moderate externalized anger comparable to Subgroup 3. Good discrimination between subgroups was evident based on average latent subgroup probabilities $> 90\%$ for all classes.

Table 4 presents the growth estimates for the unconditional growth model and the conditional model of subgroup predicting change in PTSD during treatment. The unconditional model had good fit ($\chi^2 = 32.80$, $p < .05$, RMSEA = .07, CFI = .98) and suggested a curvilinear form to the data with PTSD declining over treatment and leveling off during the posttreatment and follow-up. As shown in Figure 2, all four latent subgroups significantly predicted initial status and change in PTSD. Benjamini-Hochberg adjusted pairwise comparisons revealed differences between all four subgroups on session 2 PTSD severity (adjusted $ps < .05$). Subgroup

4 (HH) exhibited a significantly greater decrease in PTSD over treatment than Subgroup 3 (LM; $W = 6.98$, adjusted $p = .049$), and Subgroup 1 (LL; $W = 6.27$, adjusted $p = .037$), but not Subgroup 2 (HML; $W = .371$, adjusted $p = .651$). There were no differences in change in PTSD between the other subgroups. There was a large effect size change in PTSD from session 2 to 6 month follow up for each subgroup ($g = 1.62$ for Subgroup 3 to $g = 2.12$ for Subgroup 4). Finally, a time by treatment condition interaction emerged for Subgroup 4 (HH), but not the other subgroups. Individuals in Subgroup 4 who received WA demonstrated less decrease in PTSD than those who received the cognitive therapies (WA vs. CPT+A or CPT: $B = 1.20$; 95% CI = .05 – 2.36, $SE = .59$, $p = .042$), although all three treatment conditions showed decreases in PTSD from session 2 to 6 month follow up with large effect size changes in the cognitive conditions (CPT $g = 1.63$, CPT+A $g = 2.33$) and a medium effect size for WA ($g = 0.48$).

4. Discussion

This study used LPA to identify if PTSD symptoms and specific emotions can organize PTSD heterogeneity and predict differential responses to PTSD interventions. Four subgroups of PTSD symptom cluster/emotion profiles emerged in this sample: low symptom cluster severity and low emotions (LL); moderate-high re-experiencing symptom cluster severity, low internalized emotions (MHL); low reexperiencing symptom cluster severity, moderate emotions (LM); and high symptom cluster severity and high emotions (HH). Consistent with prior LPA studies, these subgroups generally stratify based on severity (e.g., Bondjers et al., 2018; Campbell et al., 2020; Contractor et al., 2015; Sripada et al., 2020). It is important to note that, although the subgroups had varying levels of PTSD severity, even the LL group was sufficiently symptomatic to warrant a diagnosis of PTSD and subgroup names are therefore relative to each

other rather than reflective of absolute values of severity. However, several symptom clusters and emotions differentiated the subgroups beyond PTSD severity.

First, classes characterized by more moderate severity symptom clusters and emotions (i.e., MHL, LM) differed in re-experiencing symptoms and all emotions other than guilt. That is, in our sample, two subgroups of “moderately symptomatic” participants emerged: those who reported lower relative re-experiencing symptom cluster severity and higher relative specific emotions, and those who endorsed higher relative re-experiencing symptom cluster severity and lower relative specific emotions. While our data do not allow us to firmly speculate on the mechanisms that might explain these profiles, they may reflect how people experience their PTSD upon self-report. That is, in this sample of treatment-seeking participants who meet diagnostic status for PTSD, among those with moderate (i.e., mean level) PTSD, some individuals may be more prone to experience distress specifically in response to trauma cues and reminders (i.e., elevated re-experiencing but lower specific emotions), whereas others may be prone to experience more general distress rather than emotional responses to trauma cues specifically (i.e., lower re-experiencing but higher specific emotions). Notably, these groups had roughly equivalent levels of avoidance and hyperarousal PTSD symptoms, suggesting that variability in PTSD presentations across these groups is specific to re-experiencing and the specific emotions that they experience, rather than pervasive across PTSD symptoms. Clinically, this may highlight the importance of assessing additional trauma-related emotions like anxiety, shame, and anger, particularly for individuals who endorse relatively lower “classic” PTSD symptoms such as re-experiencing, because some individual’s distress may not be captured by PTSD symptomology per se. Indeed, such findings bolster the valuable addition of alterations in a range of negative emotions beyond fear in the Diagnostic and Statistical Manual of Mental

Disorders fifth edition relative to the fourth (APA, 2001; 2022). However, because our study was not able to test this hypothesis directly, additional research is needed to probe whether there is indeed a relationship between elevations in re-experiencing symptoms and decreases in specific emotions when comparing these two classes. Diverging from Smigelsky et al. (2019), our results suggest that these subgroups involved comparable guilt, suggesting that general guilt may not differentiate PTSD profiles, at least at moderate severity. One reason for this may be that we used the global guilt scale which is a measure of general guilt rather than guilt cognitions, which are specific trauma-related forms of guilt.

In contrast, the HH subgroup was the most symptomatic subgroup with elevations in all symptoms and emotions relative to the overall sample and all four subgroups. One exception was externalized anger which, though still elevated in comparison to the overall sample, was comparable to the LM subgroup. Thus, in comparison to the other subgroups, the distinguishing feature of this HH subgroup in comparison to the rest of the sample appears to be self-focused emotions and PTSD symptoms. Our HH class is partially consistent with prior research linking high PTSD symptoms to higher rates of anger (Durham et al., 2020), and previous LPA studies that show anger as differentiating subgroups (Saraiya et al., 2021; Armour et al., 2014). Prior studies that showed linkages between high PTSD symptoms and anger had mixed gender samples (Durham et al., 2020; Saraiya et al., 2021), and men with highly symptomatic PTSD may be particularly likely to also have high externalized anger.

The present sample was exclusively women, and the particularly symptomatic and emotional subgroups in this context may not exhibit such variability in externalized anger. However, although externalized anger didn't differentiate the HH and LM classes, both of whom had elevations in other emotions, it was still higher in these classes than the other two. Indeed,

among the two profiles with higher emotions (HH and LM), those with higher re-experiencing (or intrusive) symptoms experienced elevated internalized, but not externalized, anger than those with lower re-experiencing symptoms. One explanation for this may be that elevated intrusions may frequently remind participants of their trauma and their perceived role in it, leading to elevated internalized anger. This hypothesis warrants direct examination in further studies. It is also important to note that both the high and moderate emotion subgroups exhibited elevated externalized anger relative to low emotion subgroups, suggesting that externalized anger is still elevated in this group albeit less so than internalized anger. This suggests that both forms of anger differentiate the HH class from the low emotion subgroups, but internalized anger may be particularly potent in this group.

4.1. Emotion Profiles and PTSD Treatment

All four subgroups showed clinically significant improvements in PTSD symptom severity over treatment. However, only the HH group differed from the other subgroups in its rate of change in PTSD symptoms with greater reductions in PTSD symptoms than the LM and LL groups, but not the MHL group. Trajectories of change in PTSD did not differ across other groups. These findings are consistent with some research suggesting that higher baseline PTSD severity predicts greater improvements during PTSD treatments (e.g., Forbes et al., 2003), but inconsistent with other findings demonstrating the opposite (e.g., Speckens et al., 2006; van Minnen et al., 2002; Resick et al., 2021; Zang et al., 2021). Our findings may help to clarify this mixed literature by suggesting that elevations in both PTSD symptoms *and* emotions at baseline may predict greater responses to treatment, whereas elevations in PTSD symptoms alone at baseline may not exert such clear effects. Beyond this, our results suggest that emotion profiles generally do not differentially predict who responds to PTSD interventions.

Consistent with our hypothesis, while all four subgroups showed sustained improvements at follow-ups, only individuals in the HH subgroup showed a differential response across treatment conditions. Specifically, individuals in this subgroup had greater improvement when assigned to the cognitive conditions compared to the WA condition. Previous studies have shown poorer treatment outcomes associated with WA (Stein et al., 2012). To the contrary, our study suggests that most individuals with PTSD respond comparably to WA and cognitive interventions. However, a specific subgroup of individuals with PTSD, those with high emotions and PTSD symptoms, may not. It is unclear why this specific subgroup does not benefit to the same extent as others from WA. Perhaps, consistent with our hypothesis, cognitive interventions more directly target appraisals that lead to emotions other than anxiety/fear such as self-blame than written-based exposure interventions, resulting in smaller reduction in PTSD for individuals who have elevated emotions that are elicited by them. It is notable that research shows that cognitive change is a key mechanism of exposure-based interventions such as Prolonged Exposure (Cooper et al., 2017). However, these interventions involve in-vivo and verbal imaginal exposure rather than solely written exposure, as well as processing those exposures with the therapist (Foa et al., 2019), and it is possible that such approaches elicit cognitive change more directly. In addition, theorists suggest that some emotional responses may be related to sensory information processing systems whereas others are linked to verbal, cognitively-mediated, ones (Brewin et al., 1996). Cognitive interventions may be particularly suited to address PTSD for individuals with high levels of emotions linked to verbal, cognitive processes, whereas individuals with high levels of sensory-based emotions (e.g., fear) may respond comparably to both cognitive and written exposure interventions. It is also important to note that the HH group represents a minority of women in the sample (21%). Consequently,

although *some* individuals with PTSD may need a particular focus on cognitive interventions, many may benefit equally from exposure or cognitive approaches.

4.2. Limitations and Future Directions

There are a number of limitations of this study. First, the use of DSM-IV (APA, 2001) PTSD criteria and a female sample exposed to physical or sexual assault limit generalizability of our findings. Our findings warrant replication studies with DSM-5-TR criteria (APA, 2022) and a sample diverse to gender identity and a broader range of trauma exposure. Moreover, the majority of our sample was composed of individuals identifying as White or African American and key variables assessing other axes of diversity (e.g., disability status, sexual orientation, religion) were not measured. Replication studies should therefore expand their demographic measurement and representation across a range of axes of diversity. As well, sample sizes for the latent subgroups were small, so growth curve and moderation analyses may have been underpowered. The latent subgroups warrant validation to confirm that all distinct subgroups have been identified, which the sample size of the current study was not powered to do. As such, it is possible that meaningful population-level subgroups are not represented in this sample. In addition, at points we have suggested that specific emotions may differ depending on whether they arise from trauma-related beliefs or are a natural response to trauma. However, several emotions may fall into either category depending on the specific trauma experienced and its appraisal, and our data do not allow us to identify which is which. Future efforts to organize PTSD heterogeneity on the basis of specific emotions may benefit from a fine-grained analysis that incorporates trauma-related beliefs and the emotions that are linked to them.

Likewise, our study did not examine key emotions such as disgust, sadness, grief, or fear (which is distinct from anxiety; Craske et al., 2009). Future studies would benefit from a fuller

range of precisely-measured emotions. Related, measuring trauma-specific emotions may provide a more meaningful and useful way of organizing PTSD presentations than global or generalized emotions, and future researchers are therefore advised to attempt to replicate study findings with measures of specific emotions that are in reference to the trauma that occurred and its sequelae. Finally, study analyses were not preregistered, which can introduce bias to the analysis and reporting of results.

4.3. Conclusions

Despite these limitations, this study bears important clinical implications. First, that those with high PTSD severity and emotions exhibit greater PTSD severity reductions suggests that providers can offer PTSD treatments even to those with highly emotional or severe presentations. Second, a subgroup of individuals who exhibit high PTSD symptoms in combination with high, self-focused emotions may be particularly well suited to receive cognitive PTSD interventions such as CPT and CPT+A rather than written exposure interventions. However, in the majority of instances, when PTSD severity is low or moderate and such internally-focused emotions are less pronounced, clients may benefit from either intervention type. In this case, presenting clients with an array of treatment options is advisable because treatment outcomes may be optimized when people receive their preferred treatment (Simiola et al., 2015; Zoellner et al., 2009).

This is the first study to characterize subgroups of PTSD based on a range of specific emotions within a treatment-seeking population and provides important information about how profiles of PTSD-related emotions impact treatment responses.

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Table 1*Pearson's Correlations, Means, and Standard Deviations of Pretreatment Emotion and PTSD Subscales for Overall Sample*

	Shame	Anger In	Anger Out	State Anxiety	Guilt	Reexp	Avoidance	Hyper
Shame	59.19 (17.99)							
Anger In	.400***	19.29 (4.61)						
Anger Out	.162*	.360***	14.92 (4.02)					
State Anxiety	.498***	.436***	.148	50.94 (12.76)				
Guilt	.364***	.186*	.081	.260**	2.17 (1.14)			
PTSD Reexp	.172*	.109	-.063	.254**	.260**	7.58 (3.67)		
PTSD Avoid	.417***	.448***	.217**	.395**	.232**	.421***	11.70 (4.76)	
PTSD Hyper	.290***	.345***	.238**	.405***	.145	.550***	.603***	9.73 (3.12)

Notes. Pearson's correlations are shown in rows, means and standard deviations (in parenthesis) are shown on the diagonals,. Reexp = Reexperiencing. Avoid = Avoidance. Hyper = Hypervigilence.

* $p < .05$, ** $p < .01$, *** $p < .001$.

Table 2*Fit Indices for Competing Unconditional Latent Profile Models*

	AIC	BIC	SSABIC	LMR	<i>p</i>	Entropy
2 classes	6931.09	7006.36	6927.24	180.11	<.001	.746
3 classes	6868.08	6970.44	6862.84	81.01	<.001	.806
4 classes	6832.18	6961.64	6825.55	53.90	<.001	.844
5 classes	6817.69	6974.25	6809.68	32.49	.013	.857
6 classes	LOG LIKELIHOOD NOT REPLICATED					

Notes. AIC = Akaike information criterion. BIC = Bayesian information criterion. SSABIC = sample size adjusted Bayesian information criterion. LMR = Adjusted Lo Mendall Rubin Likelihood Ratio Test.

Table 3*Model Derived Means for Emotion and PTSD Subscales for the Overall Sample and Latent**Classes*

n/%	Shame	Anger In	Anger Out	State Anxiety	Guilt	Reexp	Avoid	Arousal
Overall	59.19	19.29	14.92	50.94	2.17	7.58	11.70	9.73
Class 1: ML (29/19%)	47.51	15.55	13.30	42.94	1.76	4.76	5.71	5.02
Class 2: ML (33/22%)	48.06	16.28	13.63	42.24	2.05	9.70	11.15	10.09
Class 3: LM (56/37%)	63.70	21.06	16.13	53.17	2.01	5.01	12.61	9.66
Class 4: HH (32/21%)	73.08	22.66	15.66	62.85	2.92	12.22	16.13	12.82

Reexp = Reexperiencing. Avoid = Avoidance. Arousal = Hyperarousal.

ACCEPTED

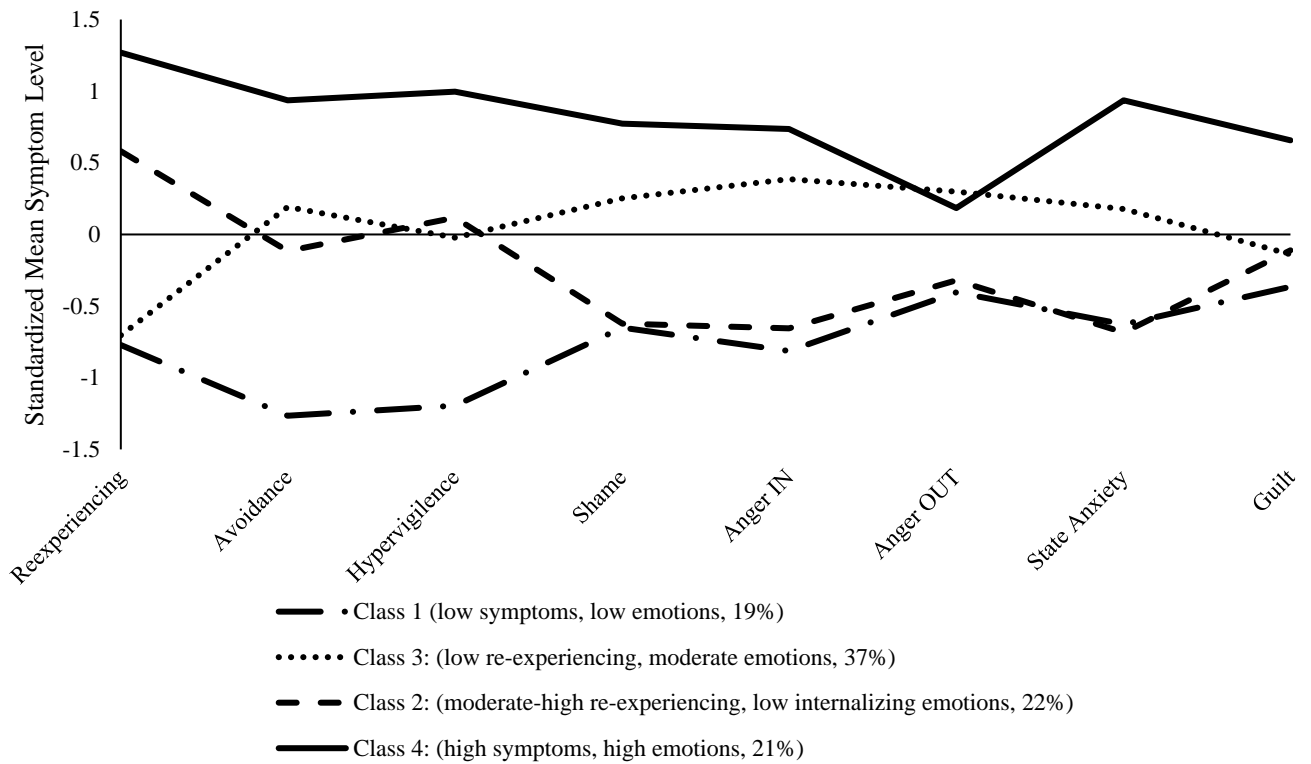
Table 4*Conditional Mixture Model of Class Membership Predicting Change in PTSD Severity Over Treatment*

Model Estimates	Initial Status	95% CI	S.E.	Slope	95% CI	S.E.	Hedges' g^a
Overall (classes not included)	28.46 ***	26.81 - 30.11	.844	-2.03	-2.36 - -1.70	.169	1.51
Class 1: LL	19.60***	16.18 - 23.01	1.74	-1.63***	-2.21 - -1.05	.297	1.69
Class 2: ML	31.88***	28.18 - 35.57	1.88	-2.40***	-3.04 - -1.75	.329	1.74
Class 3: LM	26.89***	24.51 - 29.28	1.22	-1.68***	-2.15 - -1.21	.241	1.62
Class 4: HH	36.80***	33.62 - 39.97	1.62	-2.66***	-3.27 - -2.06	.309	2.12

Note. ^aHedges' g calculated from session 2 to 6-month follow up for each latent class. Overall model fit: AIC = 5767.65, BIC = 5840.83, $\chi^2 = 32.80$, $df = 19$, $p < .05$, RMSEA = .073, CFI = .977. LL = Low symptoms, low emotions, ML = moderate reexperiencing, low emotions, LM = low reexperiencing, moderate emotions, HH = high symptoms and emotions
 $*p < .05$, $**p < .01$, $***p < .001$.

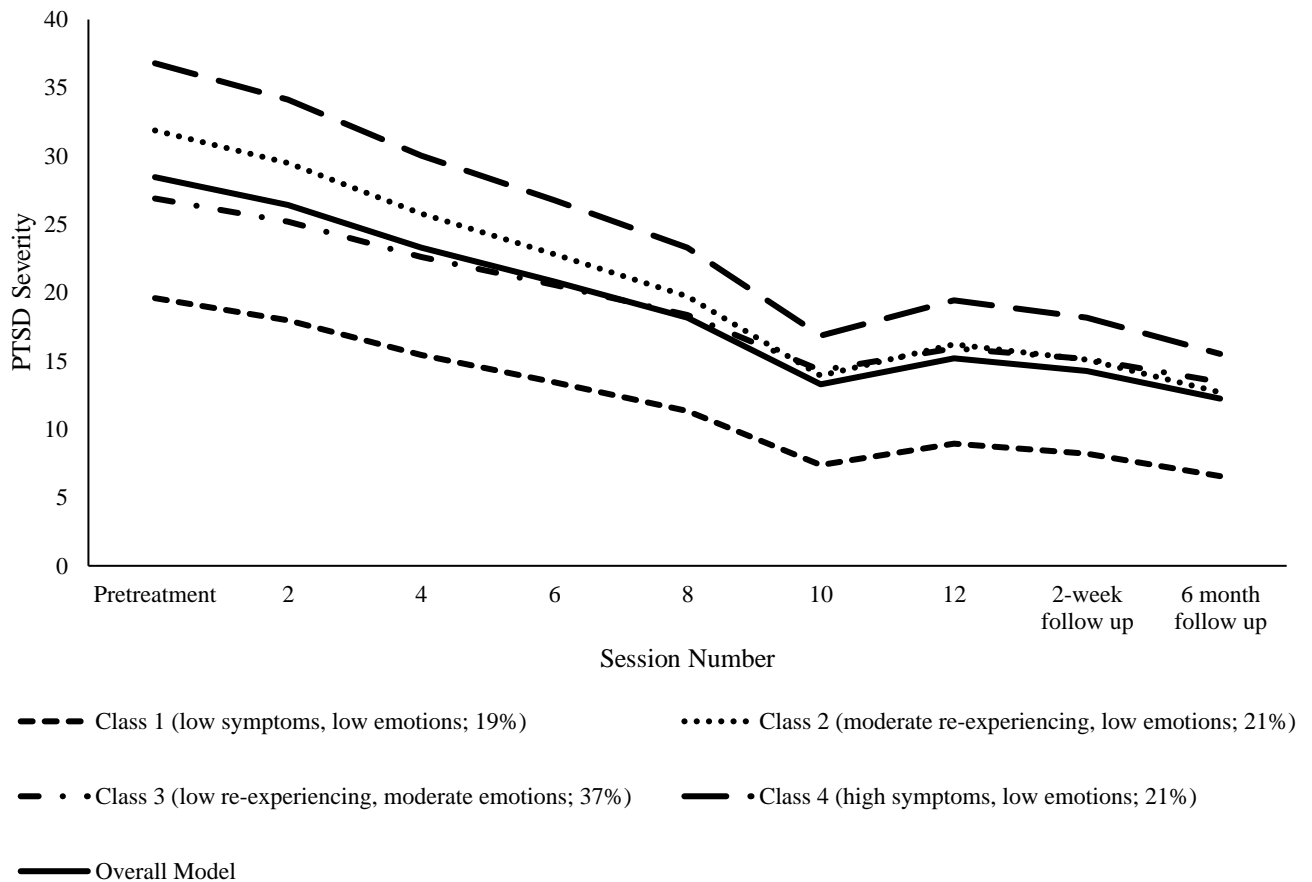
Figure 1

Four-Class Unconditional Latent Profile Plot of Emotion and PTSD Subscales



Note. Means presented as standardized z-scores.

ACCEPTED

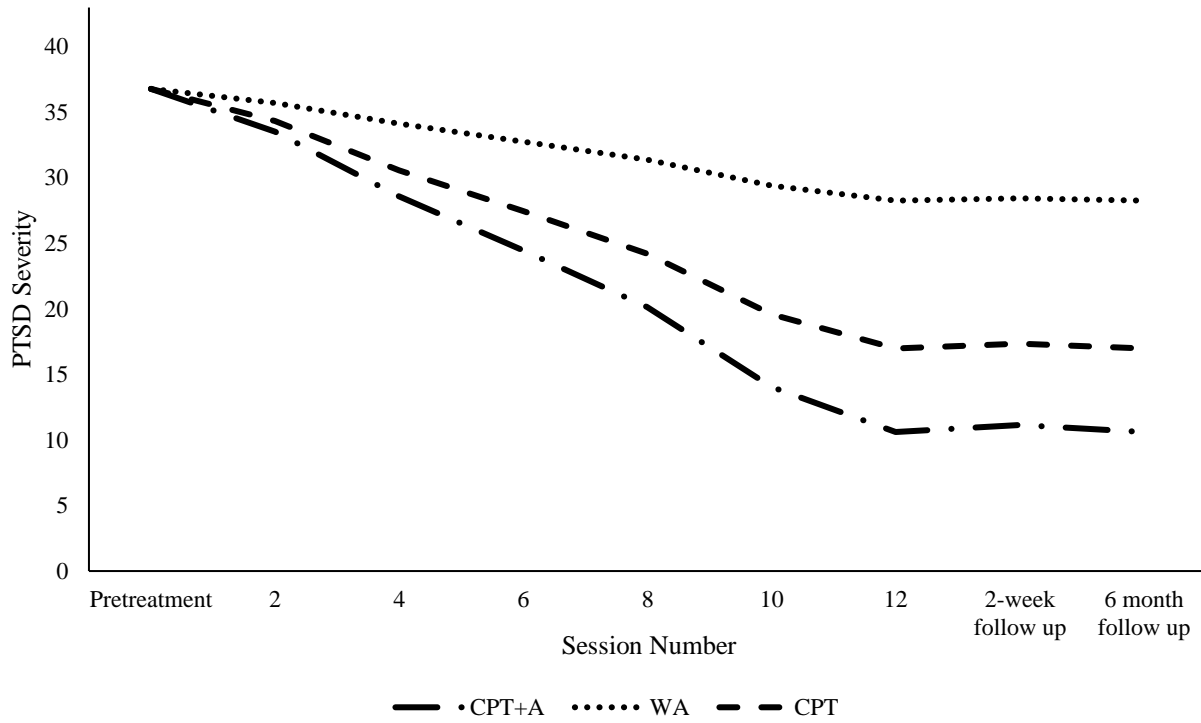
Figure 2*PTSD Symptom Change for 4-Class Model*

Note. Time freely estimated with first two time points set to 0 and 1, and last time point set to 7.

Overall Model: $B = -2.03$, $SE = .169$, $p < .001$; Class 1 (low symptoms and emotions): $B = -1.63$, $SE = .297$, $p < .001$; Class 2 (moderate re-experiencing, low emotions): $B = -2.40$, $SE = .229$, $p < .001$; Class 3 (low re-experiencing, moderate emotions): $B = -1.68$, $SE = .241$, $p < .001$; Class 4 (high symptoms and emotions): $B = -2.66$, $SE = .309$, $p < .001$.

Figure 3

Treatment Condition by Time Interaction for Class 4 (high symptoms and emotions)



Note. Time freely estimated with first two time points set to 0 and 1, and last time point set to 7.

CPT+A $g = 2.33$, CPT $g = 1.63$, WA $g = 0.48$, $B = 1.20$, $SE = .590$, $p = .042$.