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RESEARCH ARTICLE

The association between posttraumatic stress disorder symptom severity and distress tolerance in traumatic stress treatment

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Abstract

Distress tolerance, or the perceived ability to tolerate negative emotional states, is often associated with posttraumatic stress disorder (PTSD) such that higher distress tolerance is generally associated with less severe PTSD symptom levels. As distress tolerance is often considered a risk and maintenance factor in distress disorders, examining the association between changes in distress tolerance and changes in PTSD symptoms may have clinical relevance. The present study examined the associations between PTSD symptom severity and distress tolerance across three assessment points over 12 weeks among 212 patients receiving outpatient psychotherapy services. Using random-intercept cross-lagged panel modeling (RI-CLPM), concurrent and prospective associations between PTSD and distress tolerance were examined. PTSD symptoms at Time 1 and Time 2 significantly predicted distress tolerance at Time 2, $\beta = -.296$, and Time 3, $\beta = -.395$, respectively. Distress tolerance did not predict subsequent PTSD symptom severity. Exploratory analyses examined distress tolerance and four PTSD symptom clusters over time. Patterns of results differed across clusters, though it was consistent that only PTSD symptom clusters predicted subsequent distress tolerance and not vice versa. The results support the interrelationship of changes in psychopathology and emotional distress tolerance and indicate that distress tolerance may be an important factor in symptom remission during PTSD treatment.

Distress tolerance is a transdiagnostic construct with important implications for the development and maintenance of psychopathology (Leyro et al., 2010), and it is often described as the actual or perceived ability to tolerate aversive or distressing physical or emotional states (Vujanovic et al., 2022). Although distress tolerance can be examined behaviorally (i.e., physical distress tolerance), much of the empirical literature has examined the self-reported perceived capacity to withstand negative or aversive emotions, sometimes referred to as emotional distress tolerance (Simons & Gaher, 2005; Zvolensky et al., 2010), and self-reported emotional distress tolerance and behavioral distress tolerance are often uncorrelated or minimally correlated (Leyro et al., 2010). Emotional distress tolerance can be multidimensional, involving the experiences of attentional absorption by the emotion, tolerating or withstanding the emotional state, assessing the emotion as acceptable, and regulating the emotion as needed (Simons & Gaher, 2005). Distress tolerance is associated with a broad range of psychological concerns, including distress disorders (i.e., generalized anxiety disorder [GAD], major depressive disorder [MDD], and posttraumatic stress disorder [PTSD]), borderline personality disorder, obsessive–compulsive disorder, disordered eating behaviors, problematic substance use, and nonsuicidal self-injury and suicidal behavior (Akbari et al., 2022; Anestis et al., 2013; Conway et al., 2021; Garner et al., 2018; Macatee et al., 2020; Mattingley et al., 2022). In general, there is a negative correlation between distress tolerance and psychopathology such that individuals with more severe mental health–related symptoms report lower perceived tolerance for negative emotional states (Leyro et al., 2010). Distress tolerance has also been conceptualized as a trait-like measure, though it may be context-dependent. Some investigations have found consistency in self-reported distress tolerance over time (Cummings et al., 2013; Kiselica et al., 2014), and other prospective research has indicated that distress tolerance is amenable to change over time during treatment (see Heiland & Veilleux, 2023, for a review).

Given its consistent associations with both internalizing and externalizing psychopathology, distress tolerance is often investigated as a transdiagnostic developmental and maintenance factor of psychological disorders. Macatee and colleagues (2020) found that distress tolerance was correlated within relatives even after accounting for neuroticism, suggesting it may be a general risk factor in families. Distress tolerance has also been found to be negatively associated with negative affectivity (Kiselica et al., 2015; Leyro et al., 2010; Vujanovic et al., 2013). Some researchers have also suggested that individuals with a low tolerance for negative emotional states may be especially prone to engage in avoidance when presented with these aversive emotional experiences (Zvolensky et al., 2010), which is an important maintenance factor in anxiety and distress disorders. Similarly, internalizing distress disorders, such as GAD, MDD, and PTSD, share a common core of negative affectivity or generalized distress (Kotov et al., 2021; Watson, 2009). Because of the significant role of negative affect and generalized distress in PTSD (Byllesby & Palmieri, 2023), it is imperative to examine the role of distress tolerance in the context of trauma-related symptoms and their treatment.

More specifically, distress tolerance is theoretically associated with trauma exposure and posttraumatic stress symptoms. Researchers have posited that distress tolerance is a risk and maintenance factor for PTSD, and Vujanovic et al. (2022) discussed the possible theoretical pathways for this association. One rationale is that lower distress tolerance may be a predisposing factor for the development of PTSD following a potentially traumatic event (PTE) such that lower tolerance for aversive emotions may lead to increased avoidance behaviors (Lynch & Mizon, 2011; Vujanovic & Zegel, 2020). Emotional, behavioral, and cognitive avoidance are central features of PTSD and may maintain PTSD symptoms over time, consistent with the emotional processing theory of PTSD (Foa & Kozak, 1986). The emotional processing theory further considers that emotional engagement is a necessary condition for recovery. Vujanovic and Zegel (2020) also hypothesized that experiencing a PTE could alter one's perception of their ability to tolerate distress, possibly by updating previously held beliefs about emotions or one's self-efficacy in an emotional or stressful situation. The authors also proposed that the association could be bidirectional or transactional. For example, after experiencing a PTE, an individual may experience acute stress symptoms that influence their perceived tolerance of emotional experiences, which may then further influence posttraumatic stress symptoms. However, there is little empirical research that has examined changes in distress tolerance from pre- to posttrauma or how these changes interact with psychopathology in the immediate aftermath of a potentially traumatic experience.

Empirical research has generally supported these theoretical associations and found that distress tolerance, often measured using the Distress Tolerance Scale (DTS; Simons & Gaher, 2005), is consistently associated with PTSD symptom severity. Additionally, across studies, distress tolerance has been negatively correlated with both self-report (Simons et al., 2021; Vujanovic et al., 2013, 2016) and interview-based measures of PTSD symptoms (Marshall-Berenz et al., 2010; Vujanovic et al., 2022). Similarly, it has been associated with PTSD symptom severity in community samples (Marshall-Berenz et al., 2010; Vujanovic et al., 2013), veteran samples (Simons et al., 2021; Vinci et al., 2016), and samples of trauma-exposed individuals with problematic substance use (Vujanovic et al., 2016, 2022). A recent meta-analysis found that across 56 studies, there was an overall effect size (r) for the association between distress tolerance and PTSD of −.335; however, the effect size was stronger for self-reported emotional distress tolerance ($r = -.422$) compared to behavioral distress tolerance ($r = -.064$; Akbari et al., 2022).

In addition to overall PTSD symptom severity, researchers have also examined the associations between distress tolerance and the four PTSD symptom clusters outlined in the *Diagnostic and Statistical Manual of Mental Disorders* (5th ed., text rev.; *DSM-5-TR*; American Psychiatric Association [APA], 2022), namely, intrusions, avoidance, negative alterations in cognition and mood (NACM), and alterations in arousal and reactivity (AAR). To meet the criteria for a PTSD diagnosis, individuals need to experience at least one intrusion symptom (e.g., intrusive distressing memories), one avoidance symptom (e.g., avoidance of internal trauma-related stimuli, such as thoughts, emotions, or physical sensations), two NACM symptoms (e.g., persistent negative emotional state), and two AAR symptoms (e.g., hyperarousal). Many studies have found that all four PTSD symptom clusters are also negatively associated with distress tolerance (Banducci et al., 2017; Ennis et al., 2022; Vujanovic et al., 2016). However, Vinci et al. (2016) found that after controlling for covariates, distress tolerance was associated with intrusions and hyperarousal but not emotional numbing (i.e., NACM) or avoidance symptoms. These results are similar to Fetzner et al. (2014), who found that distress tolerance was associated with intrusions and avoidance, but not emotional numbing or hyperarousal, after controlling for time since trauma, number of traumatic events experienced, sex, and depression. In contrast, a meta-analysis that examined the associations between PTSD symptom clusters and distress tolerance found significant negative correlations with all four PTSD symptom clusters; moreover, in mixed-effects subgroup analyses, no differences were found in the effect sizes of these associations between the four symptom clusters (Akbari et al., 2022).

As distress tolerance may be a risk factor for the development and maintenance of PTSD symptoms, some researchers have theorized it to be a mechanism of symptom change during treatment (Sripada et al., 2016). Therefore, the construct has also been examined longitudinally and in treatment-seeking samples. For example, Hashoul-Andary et al. (2016) found that emotional distress intolerance 3 months after PTE exposure prospectively predicted symptom levels assessed 6 months posttrauma in a community sample of adults. Additionally, pretreatment distress tolerance was found to predict posttreatment PTSD symptom severity in a sample of veterans seeking treatment for PTSD and substance use disorder (Levy et al., 2018). Banducci et al. (2017) also found that changes in veterans' distress tolerance during residential treatment predicted both overall PTSD symptoms and all four PTSD symptom clusters at discharge. Similarly, improvements in distress tolerance during treatment were found to predict lower PTSD symptoms at 3-month follow-up in a community sample of adults (Boffa et al., 2018). Finally, in treatment-seeking adults with PTSD symptoms and problematic substance use, baseline distress tolerance predicted subsequent PTSD symptom severity such that lower tolerance at baseline was associated with higher symptom levels at the end of treatment (Vujanovic et al., 2022). These findings are insightful with regard to understanding that initial distress tolerance may predict posttreatment PTSD symptoms; however, these studies only included two assessment points, typically pre- and posttreatment assessments. Generally, at least three assessment points are required to adequately model linear effects (McCormick et al., 2023); thus, a pre–post design may not be able to capture linear effects. Similarly, these previous investigations did not examine bidirectional associations between these constructs, often only specifying PTSD symptoms as the outcome of interest.

The present study intended to confirm and extend previous findings regarding the association between distress tolerance and PTSD over time. In addition to replicating previous findings on the associations between PTSD and distress tolerance cross-sectionally in clinical samples, the current study aimed to examine these interrelationships prospectively over the course of treatment in a civilian treatment-seeking outpatient sample. The present analyses examined the interrelationships between PTSD symptom severity and distress tolerance using random-intercept cross-lagged panel modeling (RI-CLPM), allowing for prospective associations to be examined while controlling for PTSD symptoms and distress tolerance at the preceding time point. We anticipated that PTSD symptoms at a given assessment point would predict subsequent PTSD symptoms and hypothesized that distress tolerance at a given assessment point would predict subsequent perceived tolerance of distress. We also expected that distress tolerance at a given assessment would predict subsequent PTSD symptom severity, which is consistent with previous findings. Additional exploratory analyses explored the same cross-lagged models for the four PTSD symptom clusters and distress tolerance, but no hypotheses were made regarding differences across symptom clusters.

METHOD

Participants and procedure

Participants were treatment-seeking outpatients at a hospital-embedded specialty traumatic stress clinic. Data were collected as part of routine clinical care. Participants completed a battery of self-report questionnaires at their first intake appointment and then again at 6 weeks and 12 weeks after their intake as part of the standard clinical progress monitoring and outcome assessment. Institutional Review Board (IRB) approval was waived because the data were deidentified, and, therefore, the study did not constitute human subjects research per institutional policy. Participants were included in the present study if they were deemed appropriate for traumatic stress behavioral health services and completed the primary measures for at least two of the three assessment points concurrently. Participants received a variety of evidence-based individual and group treatments, and inclusion was not restricted to a specific treatment modality (e.g., cognitive processing therapy [Resick et al., 2017] or prolonged exposure [Foa et al., 2019]).

The average participant age was 40.2 years (*SD* = 12.3, range: 17–68 years), and most participants identified as female (65.0%). In total, 17.2% of the sample identified as Black or African American, 82.8% identified as White, and 4.0% identified as American Indian or Native American. Most individuals indicated they were single or had never married (30.3%), married (29.3%), or divorced (18.7%). Roughly one third of participants (37.8%) reported they were employed full-time, and 26.5% reported being unemployed. The most commonly reported PTEs were being beaten up or attacked (66.0%); an unwanted sexual experience or sexual assault (64.4%); the death of a family member or close friend due to accident, homicide, or suicide (51.0%); or seeing someone seriously injured or killed (47.2%).

Measures

PTSD symptom severity

The PTSD Checklist for *DSM-5* (PCL-5; Weathers et al., 2013) is a 20-item self-report measure of PTSD symptom severity, with each item representing one symptom of the *DSM-5* diagnostic criteria of PTSD. Participants were asked to rate each item on a 5-point scale ranging from 0 (*not at all*) to 4 (*extremely*). The PCL-5 has demonstrated good psychometric properties, including excellent internal reliability (r = .94–.96) and good convergent validity with clinical interviews of PTSD symptoms (Blevins et al., 2015; Bovin et al., 2016). A PCL-5 total score of 33 or higher is generally used as a threshold for probable PTSD (Bovin et al., 2016). Internal reliability in the present sample was good at all three time points, with Cronbach's alpha values of .92, .95, and .95 at Time 1 (T1), Time 2 (T2), and Time 3 (T3), respectively.

Distress tolerance

The DTS (Simons & Gaher, 2005) is a 15-item self-report measure of emotional distress tolerance. Each item is rated on a 5-point scale ranging from 1 (*strongly agree*) to 5 (*strongly disagree*), with one item reverse-coded. The DTS includes four subscales: Tolerance, Absorption, Appraisal, and Regulation; for the present study, the primary analyses used only the total score (i.e., all subscale scores summed). Higher scores are indicative of better tolerance for perceived distress. The DTS has demonstrated good test–retest reliability (Simons & Gaher, 2005). Internal reliability was good at all three assessment points, with Cronbach's alpha values of .92, .94, and .94 at T1, T2, and T3, respectively.

Data analysis

Preliminary analyses and descriptive statistics were completed in SPSS (Version 28). Data were screened for missingness and normality. Participants were included if they completed the study measures for at least two time points: intake (T1), 6 weeks postintake (T2), and 12 weeks postintake (T3). Of the 212 participants, 121 completed the study measures at two time points, and 91 completed the measures at all three points. The primary analyses were conducted using *Mplus* (Version 8) software (Muthén & Muthén, 2017). To examine the associations between PTSD symptom severity and self-reported distress tolerance across time, RI-CLPM with free intercepts (Hamaker et al., 2015; Mund & Nestler, 2019) was specified. RI-CLPM was used because it allows for the separation of within-person and between-person change to be accounted for in the model, and traditional cross-lagged panel modeling assumes that individuals fluctuate around a group mean, conflating individual within-person change and between-person change. In RI-CLPM, within-person change is emphasized, and autoregressive paths represent the amount of within-person carryover effect (Hamaker et al., 2015). In RI-CLPM, cross-lagged paths represent how much the deviations in one variable's expected score are predicted from the previous deviations from one's expected score on the other variable while accounting for structural change and previous scores. This model specified free intercepts because constraining the intercepts implies there are no stable between-person differences (Hamaker et al., 2015), and there were differences in symptom severity across the sample. Total scores for both measures at all three assessment points were initially entered as observed variables. In line with Mund and Nestler (2019), a latent factor for each variable at each time point was estimated, and error variances were fixed to zero. For the RI-CLPM, maximum likelihood (ML) estimation procedures were used. Excellent model fit was determined by examining several fit indices, and the model fit was considered good based on a comparative fit index (CFI) value of .95 or higher, Tucker–Lewis Index (TLI) value of .95 or higher, and root mean scare error of approximation (RMSEA) value of .06 or lower (Hu & Bentler, 1999). In addition, Akaike information criterion (AIC) and Bayesian information criterion (BIC) values were calculated and reported, with lower BIC values indicating better model fit. Standardized model estimates were interpreted.

Four initial path models were examined to find the best-fitting and most parsimonious model. First, the RI-CLPM was examined with no equality constraints on the autoregressive and cross-lagged paths. Then, the model was respecified to constrain the autoregressive paths to

TABLE 1 Bivariate correlations between PTSD Checklist for *DSM-5* (PCL-5) and Distress Tolerance Scale (DTS) scores over time

Variable	T1 PCL-5	T2 PCL-5	T3 PCL-5	T1 DTS	T2 DTS	T3 DTS	Latent M[a]	Observed M[b]	Observed SD
T1 PCL-5	–	.734	.597	−.534	−.488	−.431	48.32	47.90	16.80
T2 PCL-5		–	.796	−.530	−.588	−.625	40.24	40.54	18.82
T3 PCL-5			–	−.470	−.506	−.641	37.28	37.70	18.71
T1 DTS				–	.668	.733	38.83	39.01	13.43
T2 DTS					–	.709	41.20	40.94	14.63
T3 DTS						–	42.48	42.32	14.81

Note: N = 212, Time (T) 1: n = 199, T2: n = 174, T3: n = 142. T1 represents intake, T2 represents 6-week follow-up, and T3 represents 12-week follow-up. All correlations were significant at $p < .001$. PTSD = posttraumatic stress disorder; *DSM-5* = *Diagnostic and Statistical Manual of Mental Disorders*.
[a]Mean of latent variable used in path model.
[b]Observed mean score.

be equal over time while the cross-lagged paths were not constrained. The third model examined the model if the cross-lagged paths were constrained over time and the autoregressive paths were not. Finally, both the autoregressive and cross-lagged paths were constrained to equality. In addition to examining the association between PTSD symptom severity and distress tolerance over time, four additional exploratory RI-CLPMs were examined, one for each of the *DSM-5-TR* PTSD symptom clusters (i.e., intrusions, avoidance, NACM, and AAR).

RESULTS

Observed PCL-5 and DTS mean scores, with standard deviations, are presented in Table 1. Using a PCL-5 cutoff score of 33 or higher at T1, 78.8% of the sample met the criteria for a probable PTSD diagnosis, and 52.4% and 41.0% of the sample met this threshold at T2 and T3, respectively. Using a reliable change index of 15 or higher (Marx et al., 2022), 31.6% of the sample achieved reliable change by T3. PCL-5 scores decreased significantly from T1 to T2, $t(154) = 7.58$, $p < .001$, representing a medium effect size, $d = 0.61$, 95% confidence interval (CI) [0.44, 0.78]. Similarly, PCL-5 scores significantly decreased from T2 to T3, $t(103) = 2.46$, $p = .016$, but the effect size was small, $d = 0.24$, 95% CI [0.05, 0.44]. The effect size for the overall change from T1 to T3 was medium in magnitude, $d = 0.71$, 95% CI [0.51, 0.90]. Distress tolerance significantly improved between T1 and T2, $t(160) = -2.48$, $p = .014$, with a small effect size, $d = -0.20$, 95% CI [-0.35, -0.04]. The difference in DTS scores between T2 and T3 did not significantly differ, and the effect size for the overall change from T1 to T3 was small, $d = -0.36$, 95% CI [-0.55, -0.17]. These results were consistent when examining the Tolerance subscale of the DTS, which includes only three items related to perceived tolerance in the presence of emotional distress. DTS Tolerance subscale scores improved from T1 to T2, with a small effect, $d = -0.23$, 95% CI [-0.39, -0.07], with no statistically significant change between T2 and T3 and an overall small-to-medium effect from T1 to T3, $d = -0.34$, 95% CI [-0.52, -0.16]. Bivariate correlations for the DTS and PCL-5 are presented in Table 1.

Next, the RI-CLPMs were specified. Model fit indices for the initial models are presented in Table 2. After a review of the model fit indices, the model with both autoregressive and cross-lagged paths constrained was retained because it had the lowest Bayesian information criterion (BIC) value and was the most parsimonious. Table 3 shows the path model's standardized and unstandardized coefficients. The PCL-5 had significant autoregressive paths from T1 to T2, $\beta = .464$, $p < .001$, and from T2 to T3, $\beta = .541$, $p < .001$; however, the DTS autoregressive paths were not significant. In addition, PCL-5 scores at T1 were associated with DTS scores at T2, $\beta = -.296$, $p = .023$, and PCL-5 scores at T2 were associated with DTS scores at T3, $\beta = -.395$, $p = .008$. There were no significant cross-lagged effects for DTS scores predicting subsequent PCL-5 scores.

Next, the models were rerun using PCL-5 cluster-specific subscale scores and distress tolerance total scores, using the model with autoregressive and cross-lagged paths constrained. The model examining intrusive symptoms and distress tolerance over time demonstrated good model fit, $\chi^2(6, N = 212) = 11.05$, $p = .087$, CFI = .989, TLI = .973, RMSEA = .063, 90% CI [.000, .121]. Autoregressive paths for intrusions were significant such that T1 intrusions predicted T2 intrusions, $\beta = .382$, $p = .001$, and T2 intrusions predicted T3 intrusions, $\beta = .470$, $p = .001$. The DTS autoregressive paths were not significant. The only significant cross-lagged effect was that T2 intrusions predicted DTS scores at T3, $\beta = -.292$, $p = .039$.

The model examining avoidance symptoms and distress tolerance demonstrated excellent model fit, $\chi^2(6, N = 212) = 10.65$, $p = .100$, CFI = .989, TLI = .972, RMSEA = .060, 90% CI [.000, .119]. Similar to intrusions, only the avoidance autoregressive paths were significant: T1 avoidance was associated with T2 avoidance, $\beta = .406$, $p = .007$, and T2 avoidance was associated with T3 avoidance, $\beta = .404$,

TABLE 2 Model fit indices for random intercept cross-lagged path models

Model	χ^2 ($N = 212$)	df	CFI	TLI	RMSEA	90% CI	BIC	AIC
No equality constraints	0.44	2	1.000	1.000	.000	[.000, .085]	8,101.30	8,017.39
Auto-regressive paths constrained	3.66	4	1.000	1.000	.000	[.000, .100]	8,093.80	8,016.60
Cross-lagged paths constrained	4.22	4	1.000	.999	.016	[.000, .106]	8,094.39	8,017.17
Both cross-lagged and auto-regressive paths constrained	7.72	6	.997	.993	.037	[.000, .102]	8,087.15	8,016.66

Note: No chi-square values were statistically significant. df = degrees of freedom; CFI = comparative fit index; TLI = Tucker–Lewis index; RMSEA = root mean square error of approximation; CI = confidence interval; BIC = Bayesian information criterion; AIC = Akaike information criterion.

$p = .013$. For the cross-lagged effects, T1 avoidance significantly predicted T2 DTS score, $\beta = -.271$, $p = .023$, and T2 avoidance predicted T3 DTS score, $\beta = -.325$, $p = .019$.

The RI-CLPM for NACM symptoms met the criteria for excellent model fit, $\chi^2(6, N = 212) = 6.37$, $p = .383$, CFI = .999, TLI = .998, RMSEA = .017, 90% CI [.000, .092]. In this model, the only significant path was the autoregressive path for NACM symptoms at T1 and T2, $\beta = .256$, $p = .045$; the autoregressive path for NACM symptoms from T2 to T3 was nonsignificant, $\beta = .301$, $p = .054$. No cross-lagged paths were statistically significant.

Finally, the model examining AAR symptoms and distress tolerance demonstrated adequate model fit, $\chi^2(6, N = 212) = 12.56$, $p = .050$, CFI = .987, TLI = .968, RMSEA = .072, 90% CI [.000, .128]. For the autoregressive paths, only T1 AAR symptoms significantly predicted T2 AAR symptoms, $\beta = .332$, $p = .047$. For cross-lagged paths, T1 AAR symptoms were associated with T2 DTS score, $\beta = -.219$, $p = .045$), and T2 AAR symptoms were associated with T3 DTS score, $\beta = -.269$, $p = .048$.

DISCUSSION

The current study examined the change in PTSD symptoms and perceived distress tolerance across three time points in individuals receiving psychotherapy in an outpatient traumatic stress specialty clinic. First, PTSD symptom severity decreased over the 12-week data collection period, exhibiting a medium-to-large effect overall. Distress tolerance also improved from T1 (i.e., intake) to T2 (i.e., 6-week follow-up), indicating that distress tolerance is amenable to change during intervention. The change in DTS score overall from T1 to T3 (i.e., 12-week follow-up) exhibited a small-to-medium effect size. PTSD and distress tolerance were significantly negatively correlated at all three assessment points, and these findings are consistent with previous studies examining PTSD symptom severity and emotional distress tolerance in clinical samples (e.g., Boffa et al., 2018).

The primary analyses examined the interrelations between PTSD symptoms and distress tolerance prospectively. As hypothesized, autoregressive effects for PTSD symptom severity were significant such that PTSD at a given time point predicted subsequent PTSD symptoms. In RI-CLPM, positive autoregressive paths indicate that occasions when an individual scores above their expected score are likely to be followed by occasions when they continue to score above their expected score (Hamaker et al., 2015). Thus, increased PTSD symptom severity scores at one assessment point were associated with increased symptom severity at subsequent time points. Contrary to the hypotheses, the autoregressive effects for the DTS were not statistically significant, though DTS observed scores were strongly correlated across time (rs = .668–.733). This may be related to the RI-CLPM structure, which examines temporal deviations from expected scores (Hamaker et al., 2015). Although emotional distress tolerance is often described as a trait-like construct (Leyro et al., 2010), in the early course of therapy, individuals in the present study may have varied more from their expected score or there may have been less of a carryover effect.

The findings also showed that total PTSD symptoms at T1 and T2 predicted future distress tolerance at T2 and T3, respectively. This extends previous research by establishing that there are possible bidirectional associations between PTSD symptoms and distress tolerance (Vujanovic & Zegel, 2020), and this seems especially true at the early stages of receiving behavioral health services. Many previous clinical studies of PTSD and distress tolerance only examined two assessment points, and PTSD symptoms often were specified as the outcome measure (e.g., Banducci et al., 2017; Levy et al., 2018), which limits understanding of these changes. Although not a treatment study, Simons et al. (2021) used experience sampling methodology to examine changes in PTSD symptoms and distress tolerance and their associations over 1.5 years, and they found that distress tolerance had indirect effects on negative affectivity through baseline PTSD symptoms such that distress tolerance was associated with subsequent PTSD symptoms. However, this was not consistent with the current study, as distress tolerance did not predict subsequent PTSD symptom severity in the RI-CLPM. Thus, the findings suggest that distress tolerance can be predicted from an individual's prior deviation from their expected PCL-5 score while controlling for previous deviations in

TABLE 3 Random-intercept cross-lagged panel modeling path model estimates

Path	Standardized estimate	Unstandardized estimate	*SE*	R^{2a}
PCL-5 total score and DTS				
PCL-5 T1 → PCL-5 T2	.464*	0.553	0.139	PCL-5 T2 = .247*
PCL-5 T2 → PCL-5 T3	.541*	0.553	0.139	PCL-5 T3 = .354*
DTS T1 → DTS T2	.028	0.036	0.175	DTS T2 = .094
DTS T2 → DTS T3	.041	0.036	0.175	DTS T3 = .173
PCL-5 T1 → DTS T2	−.296*	0.036	0.175	
PCL-5 T2 → DTS T3	−.395*	0.036	0.175	
DTS T1 → PCL-5 T2	−.077	−0.145	0.159	
DTS T2 → PCL-5 T3	−.099	−0.145	0.159	
INT and DTS				
INT T1 → INT T2	.382*	0.464	0.132	INT T2 = .186
INT T2 → INT T3	.470*	0.464	0.132	INT T3 = .299*
DTS T1 → DTS T2	.026	0.033	0.202	DTS T2 = .052
DTS T2 → DTS T3	.036	0.033	0.202	DTS T3 = .095
INT T1 → DTS T2	−.219	−0.585	0.277	
INT T2 → DTS T3	−.292*	−0.585	0.277	
DTS T1 → INT T2	−.121	−0.069	0.050	
DTS T2 → INT T3	−.155	−0.069	0.050	
AV and DTS				
AV T1 → AV T2	.406*	0.402	0.156	AV T2 = .192
AV T2 → AV T3	.404*	0.402	0.156	AV T3 = .198
DTS T1 → DTS T2	.060	0.079	0.173	DTS T2 = .090
DTS T2 → DTS T3	.095	0.079	0.172	DTS T3 = .137
AV T1 → DTS T2	−.271*	−1.223	0.476	
AV T2 → DTS T3	−.325*	−1.223	0.476	
DTS T1 → AV T2	−.069	−0.020	0.026	
DTS T2 → AV T3	−.092	−0.020	0.026	
NACM and DTS				
NACM T1 → NACM T2	.256*	0.286	0.146	NACM T2 = .081
NACM T2 → NACM T3	.301	0.286	0.146	NACM T3 = .137
DTS T1 → DTS T2	.022	0.029	0.175	DTS T2 = .035
DTS T2 → DTS T3	.033	0.029	0.175	DTS T3 = .060
NACM T1 → DTS T2	−.183	−0.381	0.225	
NACM T2 → DTS T3	−.229	−0.381	0.225	
DTS T1 → NACM T2	−.088	−0.063	0.065	
DTS T2 → NACM T3	−.123	−0.063	0.065	
AAR and DTS				
AAR T1 → AAR T2	.332*	0.339	0.201	AAR T2 = .112
AAR T2 → AAR T3	.353	0.339	0.201	AAR T3 = .130
DTS T1 → DTS T2	.056	0.079	0.166	DTS T2 = .057
DTS T2 → DTS T3	.095	0.079	0.166	DTS T2 = .100
AAR T1 → DTS T2	−.219*	−0.556	0.272	
AAR T2 → DTS T3	−.269*	−0.556	0.272	
DTS T1 → AAR T2	−.014	−0.008	0.056	
DTS T2 → AAR T3	−.021	−0.008	0.056	

(Continues)

TABLE 3 (Continued)

Note. Time (T) 1 represents intake, T2 represents 6-week follow-up, and T3 represents 12-week follow-up. PCL-5 = PTSD Checklist for *DSM-5*; DTS = Distress Tolerance Scale; INT = intrusion symptoms; AV = avoidance symptoms; NACM = negative alterations in cognitions and mood symptoms; AAR = alterations in arousal and reactivity symptoms.

[a]R^2 of the endogenous variables.

*$p < .05$.

distress tolerance. The reciprocal of this was not supported; thus, the results suggest that PTSD symptom severity cannot be predicted from a person's previous deviations in expected DTS scores when considering prior deviations in PTSD symptom severity.

Additional analyses were also conducted to explore the associations between the four PTSD symptom clusters and distress tolerance both prospectively and concurrently. All four symptom clusters had moderate-to-large negative correlations with DTS scores at the bivariate level, which is consistent with Akbari and colleagues (2022). The results for each of the symptom clusters differed. Both avoidance symptoms and AAR symptoms were associated with subsequent DTS scores at both T2 and T3; however, NACM symptoms did not predict subsequent DTS scores. Intrusion symptoms only had one cross-lagged path from T2 intrusion symptoms predicting T3 DTS scores. In general, these results conflict somewhat with previous research because, like the results for PCL-5 total scores, prior research suggested that distress tolerance predicts subsequent PTSD symptom cluster scores, although these previous studies did not examine bidirectional effects (Banducci et al., 2017).

The prospective association between PTSD avoidance symptoms and distress tolerance was notable given the theoretical assumption that individuals with lower emotional distress tolerance may be prone to more avoidance (Zvolensky et al., 2010). Vujanovic and Zegel's (2020) review also posits that avoidance may be important in the development of PTSD after trauma exposure when an individual has low distress tolerance. For example, Hancock and Bryant (2018) found that individuals in a symptomatic PTSD group responded with more avoidance of distress compared to those in a nonsymptomatic group. This finding was also consistent with previous research exploring potential associations between AAR (i.e., hyperarousal) symptoms of PTSD and subsequent distress tolerance. Vujanovic et al. (2013) found the strongest association between distress tolerance and hyperarousal symptoms. Similarly, hyperarousal has been found to be an important maintenance factor for PTSD symptom severity over time (Marshall et al., 2006). It was surprising that no cross-lagged paths between NACM symptoms and emotional distress tolerance reached statistical significance. Because of the high saturation of negative affect in these NACM symptoms (Byllesby & Palmieri, 2023), it would be reasonable that these symptoms would be negatively associated with the tolerance of negative emotional states. However, other studies have found that distress tolerance and NACM symptoms are not related. Fetzner and colleagues (2014) found that DTS scores were not significantly associated with emotional numbing or NACM symptoms. Examining these more nuanced associations between PTSD symptom clusters and distress tolerance may be an important avenue for future studies.

Emotional distress tolerance is a fundamental assumption of most trauma-focused treatments, as clients are expected to experience and engage with trauma-related emotions rather than rigidly persisting in the negative reinforcement of cognitive, behavioral, and affective avoidance. As this study is more characteristic of an effectiveness study, it cannot inform the understanding of this association with more specific mechanistic or process conclusions, as there was no control group or active manipulation. Higher emotional distress tolerance over time may be characterized by decreased patterns of avoidance behaviors (e.g., Vujanovic & Zegel, 2020) or decreased distress through habituation (Foa & Kozak, 1986). Aspects of therapy may also provide disconfirming evidence or inhibitory learning regarding expectations of in-the-moment tolerance of emotional states (Craske et al., 2014). Because distress tolerance was not associated with subsequent PTSD symptom change in the present study, it may not support the role of emotional distress tolerance as a maintenance factor for PTSD symptoms, though that may be related to the conceptualization of distress tolerance in the DTS, which assesses perceived—not actual—tolerance of negative emotions, as well as the absorption of the distress and emotional appraisal. However, the current model supports that PTSD symptoms can influence perceived emotional distress tolerance, whereas previous models, with fewer assessment points and without considering the bidirectional effects, have found that distress tolerance influences PTSD symptoms. It is possible that this association is more nuanced than previously considered.

The effects might be especially important to understanding change over time because symptom improvement begets improved perceived distress tolerance. Given that only significant cross-lagged paths emerged for PTSD symptoms predicting subsequent distress tolerance, it may be that PTSD symptom remission should be emphasized to improve the tolerance of emotional states. It could also be that decreased PTSD symptom severity leads to less emotional distress and, in turn, a perception of improved distress tolerance. Although causal interpretations cannot be made, skills to improve emotional distress tolerance may not be needed to reduce PTSD symptom severity, and providers may wish to prioritize trauma-focused interventions. Thus, the influences of distress tolerance and the changes in symptom severity may be more complex than previously considered.

Although not all individuals were engaged in interventions specifically targeting distress tolerance skills (e.g., dialectical behavior therapy), there were still changes in perceived distress tolerance observed during treatment. This supports that distress tolerance can be modified, and changes in this construct might be an important mechanism or process for symptom amelioration during therapy (Sripada et al., 2016). It should be noted that there were only differences reported between T1 and T2, and distress tolerance appeared to plateau by T3, though there was a difference between T1 and T3 distress tolerance scores. Reese et al. (2019) found that during and following substance use disorder treatment, perceived distress tolerance demonstrated nonlinear changes over time, which also may be the case in the current study. Maintaining motivation for therapy and self-efficacy for emotional engagement in therapy may be important to maximize therapeutic gains. It may be valuable for future studies to examine whether there is a dose–response effect or minimum level of emotional tolerance or engagement that predicts clinically significant change.

There are some limitations to the present study. The participants were assessed as part of routine clinical care, but data regarding the specifics of their treatment services are not available (e.g., specific intervention, number of sessions attended, comorbid diagnoses, treatment duration, level of engagement). Further, most participants were still engaged in treatment at T3 (i.e., 12 weeks into treatment), so the data may speak to progress rather than posttreatment outcomes; additional changes in PTSD symptom severity or distress tolerance could have developed further along in treatment but were not captured here. It is also possible that the current sample is skewed in that they persisted in treatment for up to 12 weeks, as individuals with very low distress tolerance may be inclined to terminate therapy services earlier (Daughters et al., 2005). Although the sample was diverse regarding trauma history, most of the participants identified as White, which may limit the generalizability of the results. These limitations should be considered within the context of some notable strengths. The present sample was a relatively large treatment-seeking sample of individuals who endorsed a substantial history of trauma exposure, and effectiveness data were collected at three assessment points during treatment. Having three assessment points is an improvement on previous findings that used only two assessment time points (McCormick et al., 2023), as two assessment points cannot adequately model linear effects and may misidentify patterns of change. The results extend the current understanding of the interaction between distress tolerance and PTSD symptoms. Further research should extend follow-up periods to examine if these associations are consistent at longer treatment intervals and posttreatment.

OPEN PRACTICES STATEMENT

The study reported in this article was not formally preregistered. Neither the data nor the materials have been made available on a permanent third-party archive; requests for the data or materials can be sent via email to the corresponding author at palmierp@summahealth.org.

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