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Exposure to potentially traumatic experiences during development, such as physical and psychological abuse or neglect, is one of the most reliable predictors of psychological symptoms, including post-traumatic stress disorder (PTSD; Sicorello et al., 2020). Characterized by avoidance, hyperarousal, and negative alterations in cognitions and mood following exposure to a stressor (American Psychiatric Association, 2013), PTSD is associated with impaired role functioning and excess health-related costs (Von der Warth et al., 2020). Extensive research has documented the associations between childhood trauma exposure and PTSD. Literature reviews from a biobehavioral perspective suggest a link via heightened cortisol levels (e.g., Li & Seng, 2018) and meta-analytic findings point to neuropsychological deficits (e.g., Malarbi et al., 2017). Additionally, although PTSD diagnosis can follow a single-traumatic event, research suggests a dose-response relationship, with cumulative exposure to trauma raising vulnerability for the disorder (Copeland et al., 2007). However, not all trauma exposure results in the development of PTSD symptoms (Copeland et al., 2007; Paolucci et al., 2001), inspiring a wealth of research investigations to identify potential mediators (e.g., Murphy et al., 2014).

Longitudinal studies have noted that psychosocial difficulties like poor social support and lack of perceived belongingness result in elevated posttraumatic symptoms among adults with a history of childhood trauma (e.g., Steine et al., 2017). Cross-cultural studies have documented the mediating role of mindfulness deficits and dissociation (e.g., Kratzer et al., 2018). One's ability to be aware (i.e., mindful) and present with (i.e., not dissociated from) one's emotions is related to emotion regulation and distress tolerance (DT), two constructs that have each been described as transdiagnostic risk factors for psychopathology (Leyro et al., 2010; Mennin &

Fresco, 2010). Specifically, individuals with a history of childhood trauma (CT) have been found to experience greater emotion regulation difficulties (ED; Banducci et al., 2014), as well as lower DT (Arens et al., 2014; Koball et al., 2016). To better understand how such mechanisms influence the etiology, maintenance, and treatment of PTSD symptoms, however, more research is needed to elucidate the distinct roles of ED versus DT.

Emotion Regulation

Emotion regulation is the process of modifying, experiencing, and expressing one's emotional responses to internal and external stimuli (Gross, 1998). Attempts to regulate emotions may result in more adaptive strategies, such as cognitive reappraisal (Andreotti et al., 2013), as well as strategies that may reflect ineffective and potentially harmful attempts to regulate emotions, such as emotional suppression (Dunkley et al., 2009). To integrate multiple theories of emotion regulation and create a more comprehensive measurement, Gratz and Roemer (2004) identified six dimensions of ED: (a) emotional nonacceptance, (b) difficulty engaging in goal-directed behaviors, (c) difficulty controlling impulses, (d) a lack of emotional awareness, (e) limited access to emotion regulation strategies, and (f) a lack of emotional clarity. Dimensions such as a lack of emotional awareness and poor impulse control are strongly associated with a variety of mental health symptoms. As such, healthy and adaptive emotion regulation capacities may support optimal well-being, whereas emotion regulation difficulties (ED), may impose transdiagnostic risk for numerous mental health problems, including PTSD (e.g., Finlay-Jones et al., 2015; Gross & Muñoz, 1995; Tull et al., 2007).

ED are common among trauma survivors (e.g., Cloitre et al., 2005) and can include a lack of clarity about one’s emotions, as well as nonacceptance of emotions, and perceived lack of strategies to regulate/modulate one’s emotional responses (Gratz & Roemer, 2004). Among

trauma survivors, ED are strongly related to increased PTSD symptoms (Cloitre et al., 2005, Tull et al., 2007; Weiss et al., 2013), and may play a role in the development and maintenance of a PTSD diagnosis (e.g., Badour & Feldner, 2013). Reliance on maladaptive emotion regulation strategies, or a lack of access to adaptive ones, is associated with increased symptoms of PTSD (Shepherd & Wild, 2014). Maladaptive emotion regulation strategies, or ED, contribute to numerous additional adverse outcomes, including impairments in both work and social domains (e.g., Cloitre et al., 2005). Further, research models that include ED demonstrate their direct effects on PTSD symptoms (Mazloom et al., 2016; Sundermann & DePrince, 2015), as well as their distinctive associations with a range of associated mental health problems (Tull et al., 2007). Regarding the clinical relevance of ED, psychological interventions that promote adaptive emotional regulation strategies (e.g., cognitive reappraisal), and aim to replace maladaptive ones (e.g., emotional suppression), are linked to significant reductions in PTSD symptoms and related difficulties (Badour & Feldner, 2013, Tull et al., 2007).

ED have been shown to result from exposure to CT, and to prefigure the development and maintenance of PTSD (Burns et al., 2010; Powers et al., 2015, Tull et al., 2007). In addition to their association with increased PTSD symptoms among adult and adolescent survivors of childhood abuse, ED also contribute to depression and anxiety (Sundermann & DePrince, 2015), and to symptom complexity paired with social withdrawal and destructive behavior (Choi et al., 2014). CT from caregiver-directed aggression in particular may predispose children to ED through caregivers' failure to model healthy emotion regulation strategies, as well as through impairment of attachment relationships that could enhance emotion regulation skills (Alink et al., 2009). Further, exposure to invalidating environments may discourage appropriate emotional expression and instead encourage emotional suppression (Linehan, 1993). As such, abusive

home environments may influence both the development of ED and their relation to PTSD symptoms. Overall, research largely supports ED as a transdiagnostic risk factor that can emerge early in life, and as an optimal treatment target for the complex emotional sequelae of CT exposure (Goldsmith et al., 2013).

Distress Tolerance

DT is defined as one's actual or perceived capacity to experience and withstand negative psychological states (Simons & Gaher, 2005). Studies examining traumatic stress response in both community and clinical samples indicate that lower DT is associated with more PTSD symptoms (Marshall-Berenz et al., 2010; Vujanovic et al., 2016), with studies measuring perceived DT yielding significantly greater effect sizes than those using behavioral measures (see Akbari, 2021 for a review). Low DT has been theorized to play a causal role in experiential avoidance, an often-cited mechanism in both etiological and maintenance models of PTSD (Vujanovic et al., 2015). Perceived inability to withstand distress may lead to avoidance of distress-related cues, which precludes learning to cope, especially the earlier and longer the process endures. As such, research that seeks to understand the influence of DT on psychopathology should measure the extent of childhood exposure.

Childhood appears to represent a particularly sensitive period for experiencing the adverse effects of trauma, given that such early exposure has been shown to impair stress reactivity and emotion regulation in self-report (Banducci et al., 2014), clinical laboratory (Hagan et al., 2014), and imaging studies (Birn et al., 2014). Theory of stress sensitization, for example, suggests that exposure to stressful life events is more likely to lead to mental health problems among individuals who experienced trauma in childhood as compared to those who did not (Heim et al., 2002). According to the stress sensitization hypothesis, early-life trauma alters

the regulation of stress sensitive systems (e.g., HPA axis), thereby increasing the impact of subsequent exposures. The manner in which such functional deficits may relate to one's ability to tolerate distress remains an empirical question. To date, few studies of CT and DT have been conducted. Across these studies, greater CT severity was associated with lower perceived DT in college student, community, and treatment-seeking samples (Arens et al., 2014; Banducci et al., 2014; Koball et al., 2015). These studies provide preliminary evidence that CT may relate to a decreased perceived ability to tolerate distress.

Overlapping Constructs

Overall, research has demonstrated that both emotion regulation and DT are important transdiagnostic risk factors for psychopathology (Lass & Winer, 2020; Rosencrans et al 2017). Although many studies treat emotion regulation as a unitary variable, it has also been conceptualized as a multifactor construct (Gross, 1999; Lee et al., 2016), broadly defined as the extrinsic and intrinsic processes that serve to modify, monitor, and evaluate emotional reactions (Gross, 1999; Thompson, 1994). Emotion regulation has been conceptualized as a broader and higher-order order construct than DT (see Leyro et al., 2010). Additionally, there is evidence to suggest that low DT may lead to maladaptive emotion regulation strategies. Low DT was found to predict greater use of suppression, avoidance, and rumination as the primary emotion regulation strategies, whereas there was no significant association with the use of reappraisal, (Jeffries et al., 2016). As such, although these constructs are theorized to be related, they are posited to be conceptually distinct (Zvolensky et al., 2010). Only a few studies to date, however, have demonstrated that DT and emotion regulation are distinct constructs (Bernstein & Brantz, 2013; McHugh et al., 2013). Bernstein and Brantz (2013) differentiated these constructs by conceptualizing DT as the ability to tolerate negative emotions and emotion regulation as

attempts to change the emotional experience. However, there is limited research examining the potentially differential mediating roles of both constructs in various relations, such as CT exposure and psychologically relevant outcomes (e.g., PTSD), which would support their distinction.

One reason it is important to differentiate emotion regulation from DT is that these constructs may represent a variety of potentially modifiable coping strategies. Studies have shown that certain strategies, such as antecedent-focused strategies (e.g., cognitive reappraisal), rather than response-focused strategies (e.g., emotional suppression), may be more effective in regulating emotions (Mennin & Fresco, 2010). Research also suggests that perceived control is pertinent to coping with stressful life events (Frazier et al., 2011). Emotion regulation coping strategies can be problem-centered, as they represent efforts to address the underlying problem by actively altering one's emotional experience, while DT coping strategies can be emotion-centered, as they reflect the ability to endure negative emotions associated with distress (Compas et al., 1993). If a stressful situation is perceived as controllable, research suggests that problem-centered coping strategies may be most useful in reducing negative emotions and associated outcomes, whereas emotion-focused coping strategies may be most useful in situations perceived as uncontrollable (Zeidner, 2005). Therefore, emotion regulation coping strategies may be more effective in situations that are controllable, while strategies that emphasize the tolerance of negative emotions may be more effective in situations that are uncontrollable, such as potentially traumatic events.

The Current Study

Given that research demonstrates an association between childhood trauma (CT) exposure, ED and low DT, this study aimed to investigate the mediating role of both constructs

and explore which accounts for more of the variance. It was hypothesized that (1) ED and DT would demonstrate an inverse relationship; (2) greater cumulative CT exposure would predict higher current PTSD symptoms; (3) via mediation by ED and DT individually; and finally, (4) based on theoretical models of experiential avoidance, DT was anticipated to have a greater effect on the relation between cumulative CT exposure and current PTSD symptoms.

Method

Participants

Assuming a medium effect size with $p < .05$ and three independent variables (i.e., the largest interaction had 3 independent variables, e.g., CT, ED, and PTSD), G*Power (Faul et al., 2007) indicated a sample size of 119 was required. The data used in this study originate from a larger online survey on lifetime trauma exposure and outcomes among undergraduate students at a southern university. Inclusion criteria for the current study consisted of participant age (18+ years) and endorsement of at least one potentially traumatic event in childhood (prior to age 18), resulting in a sample of 385. Participants, of whom 71.9% self-identified as female, ranged in age from 18- to 48-years ($M = 20.02$, $SD = 3.47$). Additionally, the sample predominantly identified as White (69.9%), and remaining participants reported Black (26.0%), Asian/Asian-American (4.4%), American Indian/Alaska Native (1.8%), Native Hawaiian/Pacific Islander (0.5%), and other (0.5%) ethnicities.

Measures

Traumatic Events Questionnaire (TEQ). Participants reported cumulative childhood exposure to potentially traumatic events using a modified version of the TEQ (Vrana & Lauterback, 1994). The original TEQ assesses 11-types of traumatic experiences, including interpersonal trauma and natural disaster trauma. Each experience is rated for frequency, severity

of injury, extent of endangerment, and age at occurrence. For this study, only items endorsed prior to the age of 18 *(*e.g., *How old were you at the time(s)?)* were used to assess CT. These items include experiences of childhood physical, sexual, and psychological abuse, as well as household dysfunction and bullying, among other potentially traumatic experiences. Items beginning with *"As an adult (18 years or older) …"* were not included. The TEQ has demonstrated high test-retest validity (Norris & Hamblen, 2004). Additionally, although the TEQ is a multi-dimensional construct, the Cronbach's alpha for child-related items in this current study is α = .53. This is similar to what has been reported in past research assessing retrospective CT exposure (Webermann & Murphy, 2019), and it has been suggested that such low internal consistency is due to the heterogeneity of trauma subtypes (Prino et al., 2018).

Difficulties in Emotion Regulation Scale (DERS). The DERS (Gratz & Roemer, 2004) was used as an indicator of ED. The DERS is a 36-item questionnaire with six subscales measuring: awareness of emotions (e.g., *when I'm upset, I become irritated with myself for feeling that way*) acceptance of emotions, ability to engage in goal-directed behavior, ability to control impulses, and access to effective ER strategies. Items are rated on a scale from 1 (*almost never*) to 5 (*almost always*). Higher DERS scores represent greater ED, and the global score demonstrates excellent internal consistency (α = .93; Gratz & Roemer, 2004). This is consistent with the internal consistency demonstrated in this current study **(**α = .90).

Distress Tolerance Scale (DTS). The DTS (Simons & Gaher, 2005) is a 15-item measure comprising of 4 subscales which assess tolerance, absorption, appraisal, and regulation. The total score represents participants' perceived capacity to tolerate distress, or negative emotional states. Participants rated each statement on a 5-point scale from 1 (*strongly agree*) to 5 (*strongly disagree*). Higher total scores on the DTS scale indicate greater DT. The DTS

demonstrates good internal consistency (α = .89; Simons & Gaher, 2005), and total scores have been shown to correlate with various psychological outcomes, including PTSD symptoms (Ameral et al., 2014). The Cronbach's alpha for the sum score in this current study is α = .90, suggesting excellent internal consistency.

PTSD Checklist for DSM-5 (PCL-5). The PCL-5 (Weathers et al., 2013), a 20-item measure designed to assess PTSD symptoms, was constructed based on DSM-5 criteria. More specifically, items are intended to broadly capture symptoms related to avoidance, intrusion, hypervigilance/arousal, and negative mood and cognitions. On a scale ranging from 0 (*not at all*) to 4 (*extremely*), participants rated level of distress associated with each symptom over the past month. Total symptom severity scores, which were used for this current study, range from 0 to 80. Research suggests that a PCL-5 cutoff score of 33 or greater is indicative of probable PTSD across samples (Weathers et al., 2013). Past research indicates that the measure has strong internal consistency (α = .94; Blevins et al., 2015), which is consistent with the excellent internal consistency demonstrated in this current study (α = .96).

Procedure

Following approval by the university institutional review board, participants were recruited from the psychology research pool at a university in the southern United States. Participants accessed the survey using a link to Qualtrics. Participants first gave informed consent online in Qualtrics. Then, participants provided demographic data, including age, ethnicity, and sex assigned at birth. Next, participants completed a series of measures, a subset of which are analyzed in the current study. Measures were presented in a randomized order. After study completion, participants were provided with information about on-campus psychological services. Participants received course credit as compensation for participation in the study.

Statistical Analyses

Data analyses were performed using AMOS 27.0. As discussed, to examine the effects of CT exposure on PTSD symptoms, we included only participants with endorsement of at least one CT. Missing data occurred at less than 5% and were handled with listwise deletion per recommendations from Schafer (1999) that such missingness is unlikely to bias data. Data were checked for normality and multicollinearity and were found to be within normal limits per recommendations from Kline (2016). Correlation analyses were conducted to test the relation between ED and DT. Additionally, a multiple mediation analysis was used to examine the indirect and direct effects of CT exposure on total current PTSD symptoms via ED and DT. Indirect effects were estimated with bootstrapping using 5,000 iterations as suggested by Hayes (2009) and MacKinnon (2008). This allowed us to bypass assumptions of normality typically used in mediation analyses and conduct a more powerful analysis (Hayes & Preacher, 2014).

Our data was cross-sectional, thus ruling out direct causal inferences including mediation. Nonetheless, using a mediation model allowed us to examine the degree to which DT and ED account for the relationship between the CT exposure and current total symptoms of PTSD. Although all data were collected over a single time point, we assumed for the purposes of these analyses that traumatic childhood experiences preceded ED and/or deficits in DT that ostensibly impact PTSD symptoms in adulthood. This assumption is fallible; see below for further discussion. To assess whether there might exist a mediational effect of components from the ED or the DT between CT and current total PTSD symptoms, we conducted both single and multiple mediation analyses. The single mediation analysis bypassed possible suppression effects that could have obscured the indirect effects, while the multiple mediation analysis allowed

accounting for other mediators. A significant cross-sectional mediation model would suggest that the mediators should be examined longitudinally to facilitate causal inference.

Results

The ranges, means, and standard deviations of the measures of ED, PTSD, and DT, as well as CT exposure frequencies, are reported in Table 1. Overall, participants reported mild PTSD symptoms (> 33) on the PCL-5, although there was significant variability (M = 22.5, SD = 17.91). Approximately twenty-nine percent of this sample exceeded the measure's clinical cutoff for PTSD (≥ 33). The mean score in DT for our overall sample is similar to the mean scores reported for nonclinical college student samples (e.g., Simons & Gaher, 2005). Additionally, participants reported moderate levels of ED, although there was significant variability (M = 85.63, SD = 25.33), which is similar to average reported levels of ED for trauma-exposed college student samples (e.g., Pan & Yang, 2021). Regarding CT exposure, on average, participants reported exposure to three potentially traumatic childhood events, although over 30% endorsed four or more trauma types, which may pose greater risk (Dong et al., 2004; Felitti et al., 1998). As shown in Table 1, the most frequently endorsed trauma types in this sample include childhood exposure(s) to natural disasters and parental separation, followed by bullying, psychological abuse, and mental illness within the household. The average age of exposure was approximately 9.5 years old.

Correlational analyses support hypothesis 1, such that ED and DT demonstrated an inverse relationship (r = -.63, p < .001). Additionally, all variables correlated as expected based on past literature. Specifically, CT exposure demonstrated significant positive relations with ED, PTSD, and a significant inverse relation with DT. PTSD had a significant positive relation with ED, and significant inverse relation with DT. See Table 2.

Mediation Analyses

As outlined above, we employed a 5,000-sample bootstrapping method in order to compute an estimate of the indirect effects of the mediators. When examined as individual mediators and in a multiple mediator model, both ED and DT significantly mediated the effect of CT exposure on current total PTSD symptoms, although ED accounted for greater variance. Specifically, as shown in Figures 1 and 2, CT exposure associated negatively with DT, and DT associated negatively with total current PTSD symptoms. Additionally, supporting hypothesis 2, greater cumulative CT exposure predicted higher current PTSD symptoms in all mediational models. Subsequent linear regression models indicate a stronger relationship between CT exposure and PTSD in adulthood for participants who endorsed exposure to four of more trauma subtypes, ($F(1, 114) = 11.66$, $p = .001$), compared to participants who endorsed exposure to less than four trauma subtypes ($F(1, 267) = 4.12$, $p = .04$). Further, unstandardized results, as recommended by Hayes for mediation frameworks (Hayes, 2017), indicate that every additional exposure to a CT subtype results in a 3.22 increase in symptoms of PTSD. Supporting hypothesis 3, CT exposure had a significant indirect effect on total current PTSD symptoms via DT in both individual ($B = .50$, 95% BCa [.15, .90], $p < .05$) and multiple ($B = .19$, 95% BCa [.05, .45], $p < .05$) mediator models. As shown in Figures 1 and 3, CT exposure associated positively with ED, and ED associated positively with total current PTSD symptoms. Contrary to hypothesis 4, CT exposure had a larger significant indirect effect on total current PTSD symptoms via ED in both individual ($B = .93$, 95% BCa [.48, 1.47], $p < .001$) and multiple ($B = .76$, 95% BCa [.38,1.25], $p < .001$) mediator models.

To further examine the larger indirect effect of ED, post-hoc mediation analyses assessing all six subscales of the DERS were conducted. The CLARITY, AWARENESS,

IMPULSIVITY, AND GOALS subscales all demonstrated significant mediating effects ($p < .05$), although the STRATEGIES and NONACCEPTANCE subscales were most significant ($p < .001$). Specifically, CT exposure had a significant indirect effect on total current PTSD symptoms via a lack of access to emotion regulation strategies ($B = .90$, 95% BCa [.47, 1.42], $p < .001$) and an even larger significant indirect effect via nonacceptance of emotional responses ($B = .98$, 95% BCa [.58, 1.50], $p < .001$). See Table 3.

Discussion

To our knowledge, this is the first study using multiple mediation models to examine whether DT and ED mediate the relationship between CT exposure and PTSD symptoms in adulthood. As anticipated, CT exposure explained current PTSD symptoms, with ED and DT each accounting for a significant portion of the variance in PTSD symptoms, although ED more strongly explained this relation as indicated by a larger effect size. This finding is consistent with past literature conveying significant relationships between various forms of CT and emotion processing and regulation (e.g., Gould et al., 2012; Young & Widom, 2014). The difference in indirect effect size for DT in the single mediation model compared to the multiple mediation model, however, should be noted. Given the overlap between DT and ED, it is possible that a proportion of variance accounted for by DT is diluted in the multiple mediation model, highlighting the important yet entangled roles of both mechanisms.

Our findings that various facets of ED may best explain its large mediating role in the relation between CT exposure and PTSD in adulthood are interesting as they may hint at potential etiological pathways. Specifically, participants with greater exposure to CT and higher current PTSD symptoms were more likely to report lacking access to efficient ER strategies, as well as report nonacceptance of their negative emotions: follow-up mediation analyses

confirmed that both the STRATEGIES and NONACCEPTANCE subscales of the DERS significantly mediated the relationship between CT exposure and PTSD symptoms in adulthood, more so than the other four subscales.

Research suggests that cognitive-affective defense strategies, such as nonacceptance of emotional experiences (NONACCEPTANCE; Rauch & Foa, 2006), may reinforce PTSD symptoms. The development of the DERS utilized an early measure of experiential avoidance, the Acceptance and Action Questionnaire (AAQ-I; Hayes et al., 2004) to assess individual willingness to experience and endure emotional experiences (Gratz & Roemer, 2004). The NONACCEPTANCE dimension of the DERS refers to such willingness to remain in contact with negative emotional experiences without subsequent negative reactions to those experiences. Given that frequent and intense negative emotions are common among individuals with a history of CT (Marusak et al. 2015), emotional nonacceptance may represent a particularly harmful regulatory strategy which may heighten PTSD symptoms (Raudales et al., 2020; Viana et al., 2017). Such finding supports post-traumatic growth theories maintaining that processing, rather than avoiding trauma-related thoughts and feelings until they become integrated into a coherent model of the self is a required component of recovery (Foa & Kozak, 1986). Processing to integrate and recover would require exposure to negative emotions related to the traumatic experiences (Batten et al., 2005). Consistent with Gratz and Roemer's (2004) initial conceptualization of emotion regulation, higher acceptance of negative emotions may help individuals stay engaged during negative emotions and may matter more for recovery, relative to the perceived ability to tolerate distress.

In considering difficulties in accessing emotion regulation strategies perceived as effective (STRATEGIES), there is evidence supporting the association between such difficulties

and PTSD (e.g., O'Bryan et al., 2015). As previously discussed, emotion regulation strategies are often learned in childhood through modeling and healthy attachment relationships, although this learning process may be disrupted by exposure to CT. There is evidence that PTSD is associated with a greater reliance on putatively maladaptive emotion regulation strategies (e.g., emotional avoidance, rumination; Seligowski et al., 2015; Short et al., 2018), and such deficits in emotion modulation may be related to CT experiences.

One potential pathway from CT exposure to PTSD in adulthood may be conceptualized using a wave metaphor. Our data suggest that when individuals with a history of CT are hit by a wave of negatively perceived emotion, they dive underwater (i.e., nonacceptance of negative emotions) and lack the swimming skills to resurface (i.e., lack of access to efficient ER strategies). In other words, emotional nonacceptance may interfere with emotional processing of the trauma and trauma related sequelae (Foa and Kozak, 1991). Specifically, deficits in this facet of emotion regulation may contribute to a greater reliance on emotion regulation strategies that are geared towards the avoidance or escape of distressing stimuli without concern for the potentially long-term negative consequences of engaging in such strategies. Although these strategies may be effective in reducing distress in the short-term, they may prevent functional exposure to trauma-related stimuli in the long-term, interfering with emotional processing and contributing to the exacerbation or maintenance of PTSD symptoms.

Overall, these findings expand on the literature by highlighting associations of CT exposure and maladaptive behaviors and emotional patterns in adulthood (e.g., Banducci, 2015; Vujanovic & Zegel, 2020). Further, results provide preliminary evidence that ED may explain—or account for—the association between CT exposure and PTSD-related symptoms, above and beyond DT among adults in a college student population. Integrating skills of ER, along with DT

training, however, may prove most useful in the prevention of PTSD for individuals with a history of CT. Longitudinal studies are necessary to define the temporal order of associations among these variables. Findings support theoretical conceptualizations of CT exposure, such that CT may lead to lower acceptance of negatively perceived emotional states and a lack of adaptive strategies to modulate such emotional states, which may confer increased risk for symptoms of PTSD in adulthood (e.g., Vujanovic & Zegel, 2020). The sequencing of such associations, however, remains an empirical question. Future research might explore the specific types of childhood TE that may lead to higher ED, as well as the various facets of DT—particularly as they relate to experiential avoidance, a commonly cited mechanism in etiological and maintenance models of PTSD (Vujanovic et al., 2015).

Limitations

Several limitations of the present study should be noted. First, trauma exposure was assessed retrospectively through a cross-sectional study design. Retrospective reports of traumatic experiences, particularly in childhood, may be less reliable due to an individual's current psychological functioning, inaccurate memory recall, and potential reluctancy to report sensitive personal information (Hardt & Rutter, 2004). More recent research, however, has found similar effect estimates for psychologically relevant outcomes, including PTSD, with both retrospective and prospective measures of CT (Scott et al., 2012). Research also notes that adults tend to minimize their degree of trauma exposure when providing retrospective reports, suggesting there is a greater likelihood of false negative reports (Brewin et al., 1993; Shaffer et al., 2008). Given such findings, potential inaccuracies in reported CT exposure may exist within our sample. Future prospective research would be ideal to verify our cross-sectional findings. Second, the characteristics of CT assessed in this study were limited to cumulative exposure (i.e.,

count of potentially traumatic events). Other aspects of trauma, such as its severity or duration may influence psychological symptoms and functioning. Lastly, our study relied solely on a self-report measure of DT, rather than behavioral measures. Current research indicates a lack of significant correspondence among self-report and behavioral measures of DT (Marshall-Berenz et al., 2010; McHugh et al., 2011). Given that various measures of DT do not appear to capture a single DT construct, multimodal studies are needed to better define the nature and correlates of DT as it relates to CT exposure and internalizing symptoms.

Despite these limitations, the current study provides preliminary evidence in support of ED and DT as distinct mechanisms in the development of post-traumatic stress. As transdiagnostic models and interventions become more popular, it is important to clearly operationalize transdiagnostic constructs such as ED and DT. Unclear distinction between potentially overlapping meta-emotional constructs hampers our abilities to draw valid inferences. Thus, continued evaluation of the similarities and differences among measures of ED and DT, and the patterns of association with CT exposure and psychiatric outcomes, will be useful for understanding the relationships among such constructs, as well as guide measure selection in future studies. Further, results may inform the development and implementation of evidence-based intervention methods designed to target and rebuild skills for accepting and modulating negative emotional states. Such intervention methods may mitigate the emergence and/or maintenance of PTSD symptoms among individuals who have experienced potentially traumatic events in childhood.

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Table 1

***Characteristics of Potentially Traumatic Events Among Participants* (*N* = 385)**

Trauma Type	*N*	%	Age range	Age mean	Age *SD*
Serious accident	86	22.34	3 – 17	12.22	4.37
Natural disaster	172	44.68	1 – 17	8.45	4.19
Parental separation	158	41.03	0 – 17	7.59	4.98
Physical abuse	38	9.87	2 – 16	9.68	4.63
Sexual abuse	33	8.57	2 – 16	9.42	4.17
Psychological abuse	99	25.71	0 – 17	9.42	4.19
Neglect	14	5.00	2 – 17	10.64	5.14
Household incarceration	33	3.63	2 – 16	9.42	4.17
Household mental illness	98	25.45	0 – 17	9.42	4.19
Household drug/alcohol abuse	81	21.04	0 – 17	6.63	5.05
Bullying	153	39.74	5 – 17	10.48	3.16
Witnessing violence	16	4.16	2 – 16	10.19	4.23
Near-death experience	36	9.35	2 – 17	12.83	4.51
News of violent death	69	17.92	4 – 17	12.91	3.81
Participation in atrocities	1	0.26	9 – 9	9 .00	0.00
Other	30	7.79	3 – 17	12.43	4.32
Overall				9.48	4.07

Event Count	*N*	%
1 Potentially Traumatic Event	94	24.41
2	102	26.49
3	73	18.96
4	41	10.65
5	31	8.06
6	23	5.97
7 or more PTEs	21	5.45

Table 2

Descriptive Statistics and Bivariate Correlations

Variable	1.	2.	3.	4.	*M*	*SD*	*Range*
1. Childhood Trauma Exposure	1	__	__	__	3.00	2.02	12
2. Emotion Regulation Difficulties	.20**	1	__	__	85.63	25.22	127
3. Distress Tolerance	-.13**	-.63**	1	__	3.15	.84	4
4. PTSD Symptoms	.36**	.58**	-.46**	1	22.25	17.91	74

* $p < .05$. ** $p < .001$.

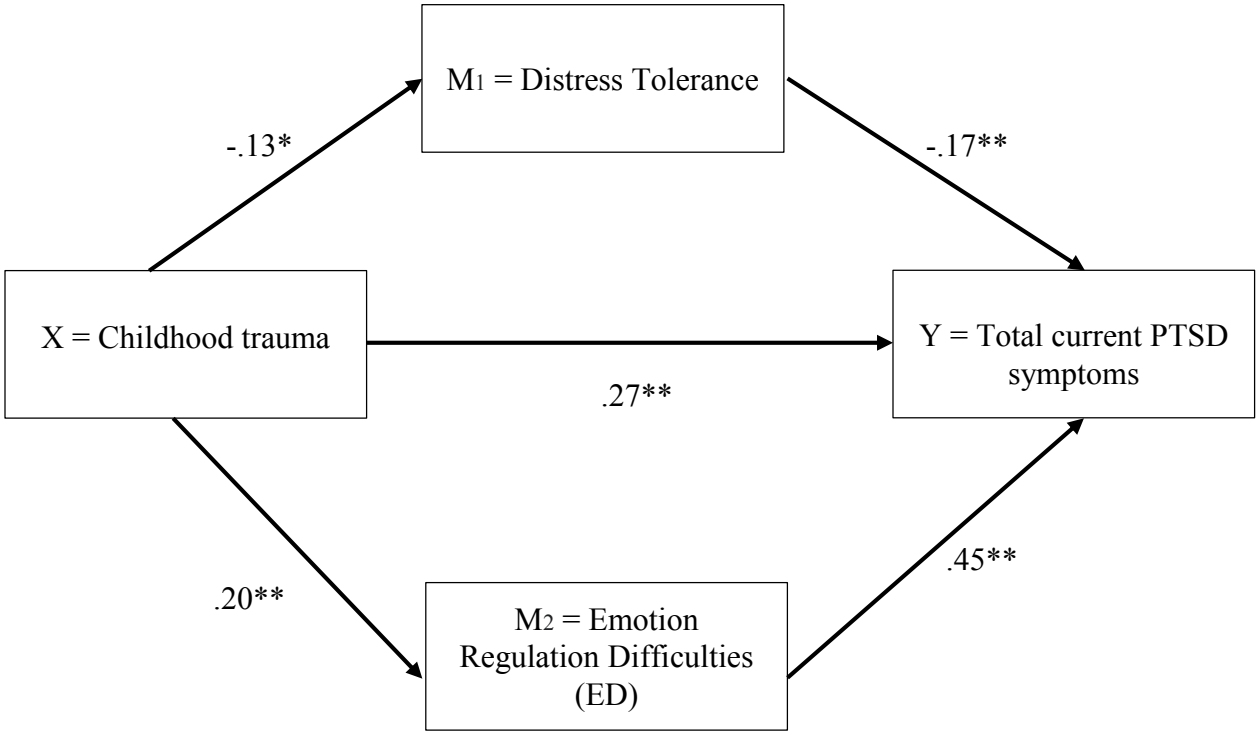
Table 3

Direct, Indirect, and Total Effects

Mediator	Effect of CT on mediator (*a*)	Unique effect of mediator (*b*)	Indirect effect (*ab*)	95% CI BCa	
				Lower	Upper
Distress Tolerance	.13*	-.42**	.50*	0.15	0.90
Emotion Regulation Difficulties (ED)	20**	.53**	.93**	0.48	1.47
ED Strategies	21**	.49**	.90**	0.47	1.42
ED Nonacceptance	25**	.45**	.98**	0.58	1.50

* $p < .05$. ** $p < .001$. CT = childhood trauma

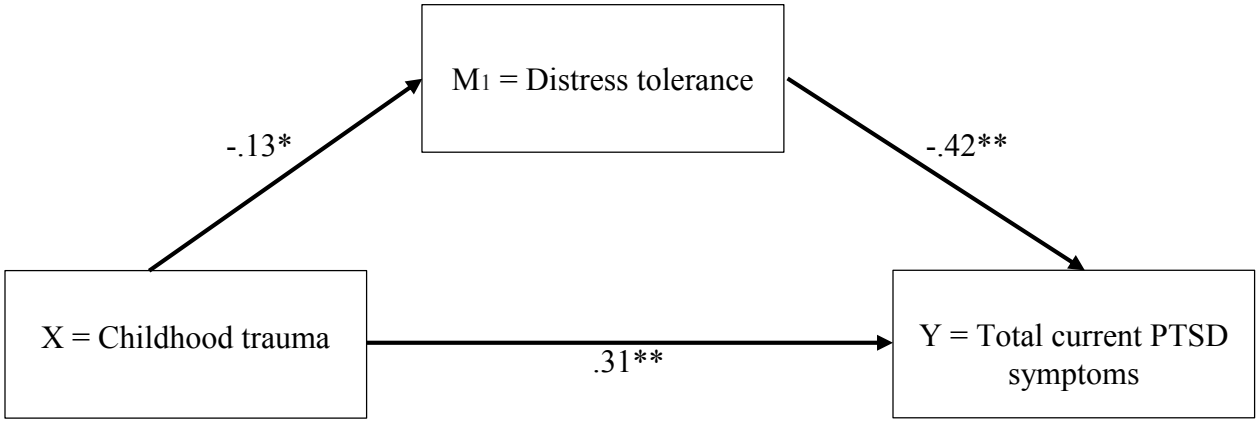
Figure 1



Indirect effect via Distress Tolerance on PTSD symptoms = .19, 95% BCa [.05, .45], $p < .05$
Indirect effect via ED on PTSD symptoms = .76, 95% BCa [.38,1.25], $p < .001$

Figure 1. Illustration of multiple mediation model. Coefficients indicate standardized regression weights. * indicates $p < .05$. ** indicates $p < .001$. Error terms are omitted for clarity.

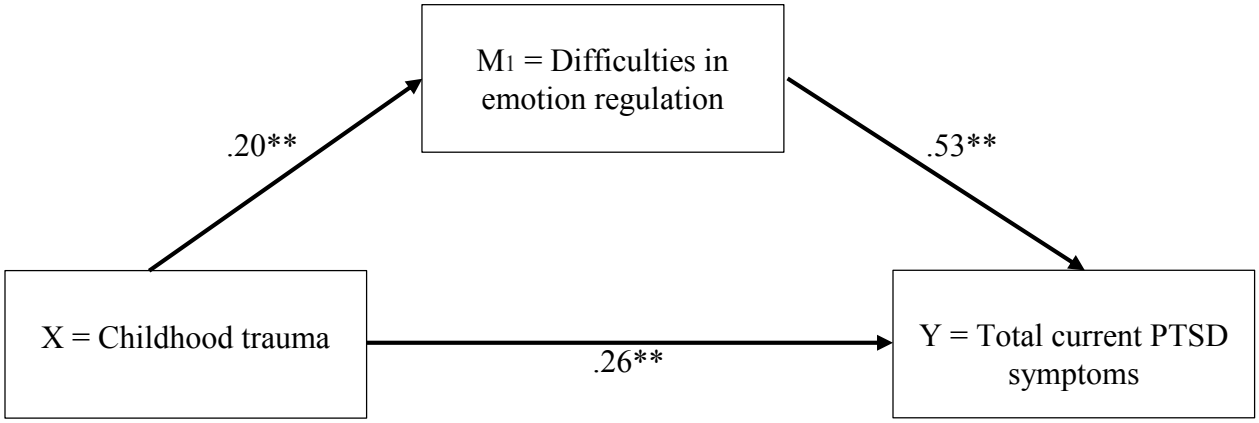
Figure 2



Indirect effect on DT on PTSD symptoms = .50, 95% BCa [.15, .90], $p < .05$

Figure 2. Illustration of single mediation model. Coefficients indicate standardized regression weights. * indicates $p < .05$. ** indicates $p < .001$. Error terms for endogenous variables omitted for clarity.

Figure 3



Indirect effect on DERS on PTSD symptoms = .93, 95% BCa [.48, 1.47], $p < .001$

Figure 3. Illustration of single mediation model. Coefficients indicate standardized regression weights. * indicates $p < .05$. ** indicates $p < .001$. Error terms for endogenous variables omitted for clarity.