2019

THE ROLE OF CHILDHOOD TRAUMA IN THE ASSOCIATIONS AMONG POSTTRAUMATIC STRESS DISORDER, EMOTIONAL REACTIVITY, AND EMOTION DYSREGULATION: A MODERATED MEDIATION MODEL

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A THESIS SUBMITTED IN PARTIAL FULFILLMENT OF THE

REQUIREMENTS FOR THE DEGREE OF MASTER OF ARTS

IN PSYCHOLOGY

UNIVERSITY OF RHODE ISLAND

2019

MASTER OF ARTS

OF

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2019

ABSTRACT

A substantial body of research over the past decade underscores the role of emotion dysregulation in posttraumatic stress disorder (PTSD). Notably, however, this research has been limited in its lack of attention to factors that may influence (e.g., childhood trauma) or explain (e.g., emotional reactivity) the association of emotion dysregulation to PTSD and inform intervention efforts. The current study addresses these limitations by using a moderated mediation approach to examine the role of childhood trauma (i.e., moderator) in the associations among PTSD (i.e., independent variable), objective and subjective emotional reactivity (i.e., mediating variable), and emotion dysregulation (i.e., dependent variable). In the mediation models, emotional reactivity did not significantly explain the relationship between PTSD symptoms and emotion dysregulation. In the moderation models, level of childhood trauma influenced the strength of the associations among PTSD symptom severity, subjective emotional reactivity, and emotion dysregulation. Specifically, subjective emotional reactivity was a significant mediator of the relation between PTSD symptom severity and emotion dysregulation at mean (but not high or low) levels of childhood trauma. Findings highlight the importance of assessing for levels of childhood trauma among women who experience IPV to identify those at risk for developing emotion dysregulation.

AKNOWLEGMENTS

I would like to thank my major professor, Dr. Nicole Weiss for her patience, guidance, support, and encouragement throughout the writing of this thesis. I would also like to thank Drs. Molly Greaney and Robert Laforge, the other members of my thesis committee, for their time, support, and contributions to this project, and Dr. Steven Cohen, my thesis defense chair for his time.

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Chapter 1.

INTRODUCTION

Posttraumatic stress disorder (PTSD) may develop following direct or indirect exposure to a traumatic event and is characterized by intrusions (e.g., recurrent, involuntary, and intrusive distressing memories of the traumatic event), avoidance of trauma-related internal and external cues (e.g., avoidance of distressing memories, thoughts, or feelings associated with the traumatic event), negative alterations in mood and cognition (e.g., markedly diminished interest or participation in significant activities), and alterations in arousal and reactivity (e.g., heightened startle; hypervigilance; American Psychiatric Association, 2013). While the majority of people in the general population report experiencing a traumatic event in their lifetime (89.7%), only 6.8% will go on to meet criteria for a PTSD diagnosis (Kilpatrick et al., 2013). Further, even among trauma-exposed individuals who do not meet full PTSD criteria, the presence of PTSD symptoms is clinically significant and common (Kilpatrick et al., 2013). Overall, symptoms of PTSD, even among individuals who do not meet full PTSD diagnostic criteria, are highly associated with a wide range of negative outcomes such as physical (e.g., chronic pain; Pacella, Hruska, & Delahanty, 2013), psychological (e.g., depression; Rytwinski, Scur, Feeny, & Youngstrom, 2013), and behavioral (e.g., risky behaviors; Tull, Weiss, & McDermott, 2015) health concerns. Notably, these negative outcomes have been found to be driven by emotion dysregulation (Weiss, Tull, Anestis, & Gratz, 2013; Weiss, Tull, Sullivan, Dixon-Gordon, & Gratz, 2015; Weiss, Walsh, DiLillo, Messman-Moore, & Gratz, in press), which is heightened among individuals with PTSD

(Tull, Barrett, McMillan, & Roemer, 2007; Weiss, Tull, Anestis, et al., 2013). The above findings underscore the need for additional research examining mechanisms underlying the relation between PTSD and emotion dysregulation; to this end, the current study will examine mediators and moderators of this association.

Emotion dysregulation is one factor that has been found to be strongly associated with PTSD. Emotion dysregulation is a multi-faceted construct involving maladaptive ways of responding to emotions, regardless of their intensity or reactivity, including: (a) a lack of awareness, understanding, and acceptance of emotions; (b) the inability to control behaviors when experiencing emotional distress; (c) lack of access to situationally appropriate strategies for modulating the duration and/or intensity of emotional responses in order to meet individual goals and situational demands; and (d) an unwillingness to experience emotional distress as part of pursuing meaningful activities in life (Gratz & Roemer, 2004; Gratz & Tull, 2010). PTSD (diagnosis and symptom severity) has been shown to be positively associated with overall emotion dysregulation as well as the specific dimensions of lack of emotional clarity and awareness, emotional nonacceptance, difficulties engaging in goal-directed behaviors and controlling impulsive behaviors when upset, and limited access to effective emotion regulation strategies (Ehring & Quack, 2010; Tull et al., 2007; Weiss, et al., 2013). Speaking to the generalizability of these findings, an association of PTSD to emotion dysregulation has been demonstrated across a wide range of populations, including (but not limited to) college students (Tull et al., 2007), community individuals (Ehring & Quack, 2010), Black emerging adults (Weiss et al., 2012; 2013), inpatients with a substance use disorder (McDermott, Tull, Gratz, Daughters, & Lejuez, 2009; Weiss, Tull, Dixon-Gordon, et al., 2013), and women

who experience domestic violence (Weiss, Dixon-Gordon, Peasant, & Sullivan, 2018). Furthermore, emotion regulation skills are considered a key target in empirically-supported treatments for PTSD, including dialectical behavior therapy prolonged exposure (DBT PE; Harned, Korslund, & Foa, 2012) and skills training in affective and interpersonal regulation (STAIR; Cloitre, Koenen, Cohen, & Han, 2002). The aforementioned results provide robust evidence of a relation of PTSD to emotion dysregulation.

Notably, although theoretical frameworks suggest emotion dysregulation as a potentially unifying factor that underlies many forms of psychopathology, including PTSD, the mechanisms that underlie the development and maintenance of emotion dysregulation following traumatic exposure has not been sufficiently researched in adults. One of the most extensively researched theories of emotion dysregulation is Linehan's biosocial theory (1993). This theory posits that emotion dysregulation stems from emotional vulnerabilities within an invalidating environmental context. Emotional vulnerabilities include (a) heightened emotional sensitivity (i.e., the tendency to pick up emotional cues and react quickly), (b) intense emotional responses (i.e., extreme reactions to emotional stimuli), and (c) slow return to emotional baseline (i.e., long lasting emotional reactions). An invalidating environment is characterized by intolerance toward the expression of private emotional experiences, which inhibits the understanding, labeling, regulating, or tolerance of emotional responses. Consequently, these environments do not provide the opportunity for individuals to practice modulating emotional arousal or coping with distress (Crowell, Beauchaine, & Linehan, 2009; Linehan, 1993). The combination of emotional vulnerabilities with an invalidating

environment drives emotion dysregulation, as the individual is experience intense, reactive, and prolonged emotions that they are unable to effectively modulate (Kuo & Linehan, 2009; Linehan, 1993).

Given this theoretical framework, emotional reactivity may be a key factor underlying the association between PTSD and emotion dysregulation. Indeed, in addition to demonstrating relations to emotion dysregulation (Fitzgerald et al. 2018; Kaczmarek & Zawadzki; 2012; Spiller et al., 2019; Strelau and Zawadzki, 2005; Wisco et al, 2018), emotional reactivity has been shown be a predominant feature of PTSD (American Psychiatric Association, 2013). A plethora of research provides support for heightened emotional reactivity among individuals with PTSD. For instance, Strelau and Zawadzki (2005) found that emotional reactivity predicted PTSD symptom severity three months, 15 months, and three years' post-traumatic exposure. Moreover, in a recent study, Spiller et al. (2019) found that individuals with PTSD exhibited greater emotion dysregulation and emotional reactivity compared to individuals without PTSD, and that higher levels of fear reactivity in particular were strongly associated with low emotion regulation capacity. It is also important to note that trauma and PTSD may elicit invalidation. For instance, individuals may experience invalidation from others at the time of the trauma (e.g., being told to put the experience behind them) or as a result of their PTSD symptoms (e.g., being told there is nothing to be fearful of). In addition, self-invalidation (e.g., believing that you should not feel a certain way) may lead to dysfunctional beliefs and heightened distress. This combination of emotional vulnerability (e.g., emotional reactivity) and invalidation post-trauma may heighten risk for emotion dysregulation.

Notably, one index of emotional reactivity is the limbic system, which controls many of the complex emotional behaviors. Indeed, stress related disorders, such as PTSD, are marked by alterations in glucocorticoid secretion, suggesting that dysfunction of the hypothalamo-pituitary-adrenocortical (HPA) axis may be involved in the adverse effects of stress on one's overall emotional reactivity (Herman at al., 2011). Exposure to a traumatic experience triggers a stress reaction, which in turn results in the production and release of the glucocorticoid hormone - cortisol. Overall, cortisol facilitates the stress response and promotes the system's homeostasis (Schulkin, McEwen, & Gold, 1994). However, prolonged activation of the HPA response can have adverse physiological and psychological consequences (McEwen, 2003). Cortisol abnormalities in PTSD have been largely manifested in hypoactivation of the stress response system, which is attributed to an enhanced HPA feedback function (Yehuda, 2002) leading to a progressive sensitization of the HPA-axis (Kendall-Tackett, 2000). Indeed, research suggests that individuals with PTSD following a single traumatic event tend to have lower basal cortisol levels than healthy or trauma-exposed individuals without PTSD (Meewisse et al., 2007). Yet, a dearth of research has examined objective indicators of emotional reactivity – such as cortisol response – in relation to PTSD and emotion dysregulation.

Another key limitation of the existing research is the lack of attention to moderators, or factors that may influence the strength and/or direction of the associations among PTSD, emotional reactivity, and emotion dysregulation. Linehan's theoretical framework highlights both biological and social factors that contribute to emotion dysregulation in PTSD. This model posits that early onset traumatic experiences alter biological processes (e.g., limbic system) responsible for emotional

reactivity, and result in vulnerability for emotion dysregulation (Linehan, 1993; Kou, Linehan, 2009). Indeed, early onset traumatic experiences are associated with changes to brain structure and function that have been found to individually contribute to deficits in emotional reactivity (Benedetti et al., 2012; Glaser, Van Os, Portegijs, & Myin-Germeys, 2006). Specifically, early onset traumatic experiences, particularly those that are recurrent and prolonged, initiate critical changes in the biological stress systems, most notably activation of the amygdala, a central component of the brain's fear detection and anxiety circuits. As a result, cortisol levels become elevated through transmission of fear signals to neurons in the prefrontal cortex, hypothalamus, and hippocampus, and activity increases in the locus coeruleus and sympathetic nervous system (for a review, see Bellis & Zisk, 2014). Indeed, research over the past decade has repeatedly demonstrated that childhood trauma as well as other childhood adversities (e.g., neglect) result in impairment in developmental processes related to emotion dysregulation through alterations to the biological process that underlie emotional reactivity (Banyard & Williams, 1996; Bellis & Zisk, 2014; Browne & Finkelhor, 1986; Cloitre, 1998; Leahy et al., 2004; Shields & Cicchetti, 1998; Shipman, Edwards, Brown, Swisher, & Jennings, 2005; Shipman, Zeman, Penza, & Champion, 2000). Furthermore, research has demonstrated that childhood trauma is highly associated with numerous negative outcomes, especially higher PTSD symptom severity (Banyard & Williams, 1996; Browne & Finkelhor, 1986; Childhelp, 2005; Finkelhor & Dziuba-Leatherman, 1994; Kessler, 2000; Kilpatrick et al., 2013; Leahy et al., 2004; Lev-Wiesel et al., 2005), and that these negative outcomes stem from early

onset emotion dysregulation (Cloitre, 1998; Tull, Gratz, Salters, & Roemer, 2004; Tull et al., 2007).

Given these gaps in literature, the purpose of the present study is to explore childhood trauma (i.e., moderator) and the role of emotional reactivity (i.e., mediator) in the relation between PTSD (i.e., independent variable) and emotion dysregulation (i.e., dependent variable) using a moderated mediation analysis. We expected that emotional reactivity would mediate the relation of PTSD symptoms to emotion dysregulation, and that these models would be moderated by childhood trauma. Further, consistent with past research, we hypothesized that the paths between PTSD and emotion dysregulation (Weiss et al., 2013), PTSD and emotional reactivity (Tull et al., 2007), and emotional reactivity and emotion dysregulation (Bellis and Zisk, 2014) would be stronger for those who experienced childhood trauma compared to those who did not. We also expected that childhood trauma severity will moderate the associations among PTSD, emotional reactivity, and emotion dysregulation.

Chapter 2.

METHODOLOGY

Procedures.

All procedures were reviewed and approved by the University of Rhode Island Institutional Review Board. Data were collected as part of a larger ongoing study examining the relations among PTSD, emotion dysregulation, substance use, and HIV/sexual risk. Participants are recruited from Providence County in Rhode Island using posters, brochures, and flyers posted in community establishments and internet forums. Eligibility is determined through a phone screen. Inclusion criteria includes: (a) female gender, (b) age 18 or older; (c) English speaking; (d) involvement in a heterosexual intimate relationship with the presence of physical and/or sexual victimization; and (e) and the use of any amount of drugs/alcohol. Exclusion criteria includes: (a) current mania/psychosis; (b) self-reported pregnancy; (c) colorblindness; (d) cardiovascular disease; and (e) residence in a shelter/group home. In addition, women are required to abstain from alcohol and illicit drugs for four hours prior to the experimental session. Women who test positive for illicit drugs or whose blood alcohol level > 0.01 are rescheduled. Women who test positive and report marijuana use in the past 30 days, but not past 24 hours are allowed to participate. Those who meet inclusion criteria are provided with information about study procedures and associated risks, following which written informed consent is obtained.

The study is completed in four parts: (a) an initial session, (b) an experimental session, (c) 30 days of thrice daily experience sampling methodology, and (d) a follow-up session. Data from the initial and experimental sessions were used in the current study.

In the initial session, a computerized version of the SCID-5 and self-report measures, including those assessing PTSD, emotional reactivity, emotion dysregulation, and childhood trauma, are administered. In addition, a protocol for developing emotion induction scripts is implemented to induce physiological and behavioral responses during the experimental session. Specifically, participants are randomly assigned to one of three emotion induction conditions: negative, positive, and neutral. Data collection for each condition is concurrent and will continue until 50 women per condition have completed the protocol. A semi-structured interview is used to elicit a personal narrative about the most recent or vivid memory (not involving trauma or substance use) during which participants became "very angry," "very excited" or "mostly neutral, and had neither unpleasant nor pleasant feelings" (negative, positive, neutral conditions, respectively). They describe in detail the situation that occurred and their emotional, physical, and cognitive responses. This interview is recorded and used to create 1-minute scripts in the second person and present tense, which are recorded and played to participants during the experimental session. This well-established method reliably induces physiological and behavioral responses (Lang & Cuthbert, 1984; Lang, Kozan, Miller, Levin, & McLean, 1980).

In the experimental session (occurring approximately five days after the initial session), participants are administered a vanilla baseline procedure (displaying colors on a computer screen for five minutes) to induce neutral mood. This procedure produces a more neutral mood compared to the absence of activities (Jennings, Kamarck, Stewart, Eddy, & Johnson, 1992). To determine baseline emotions, participants complete a self-report measure of emotional responding and provide saliva for cortisol analysis.

Following this, they listen to their personalized audiotape (one minute), and then close their eyes and vividly imagine the event for one minute (i.e., emotion induction). To determine emotional responses, participants complete a self-report measure of emotional responding; saliva for cortisol analysis is collected 20-min post-induction (because cortisol peaks 20 minutes post-stressor; Lemon, Verhoek-Oftedahl, & Donnelly, 2002).

Participants are remunerated up to $215, which included payments for initial, experimental, and follow-up sessions and graduated payments with bonus payments for compliance with daily surveys. Participants are provided with a list of community resources relevant to domestic violence, mental health and substance use, social services, employment, and economic stability.

Measures.

Initial session.

Demographic information. Information regarding age, gender, ethnicity, race, income, educational level, employment status, ethnicity, and relationship status is obtained.

Traumatic Exposure. The Life Events Checklist for DSM-5 (LEC; Blake et al., 1990) is a 17-item, self-report measure designed to screen for potentially traumatic events (PTEs) in a respondent's lifetime. The LEC assesses exposure to 16 PTEs and includes one item assessing any other extraordinarily stressful event not captured in the first 16 items. For each item, the respondent is asked to indicate if (a) the event happened to them personally, (b) they witnessed the event, (c) they learned about the event, or (d) they did not experience the event in any way. To calculate the total number of PTEs experienced, items endorsed as having been experienced (either directly or indirectly) by the

respondent received a score of 1 and were summed. The LEC has demonstrated convergent validity with measures assessing varying levels of exposure to PTEs and psychopathology known to relate to traumatic exposure (Gray, Litz, Hsu, & Lombardo, 2004).

PTSD. The PTSD Checklist for *DSM-5* (PCL-5; Blevins, Weathers, Davis, Witte, & Domino, 2015) is a 20 item self-report measure assessing PTSD symptoms over the past 30 days. Participants completed the PCL-5 in response to their most distressing traumatic event endorsed on the LEC-5. Response options range from 0 (*not at all*) to 4 (*extremely*). Higher scores indicate greater PTSD symptom severity, and a score of 33 or higher indicates probable PTSD diagnosis (Blevins et al. 2015). The PCL-5 has excellent psychometric properties (Bovin et al., 2016; Wortmann et al., 2016). Cronbach's α was .95 in the current study.

Emotional Reactivity. The Affect Intensity Measure (AIM; Larsen & Diener, 1987) is a 40-item self report measure of the trait intensity and reactivity of emotional responses, independent from the frequency and hedonic level of emotional responses. Response options range from 1 (*never)* to 6 (*always*). Higher scores indicate higher levels of emotional reactivity. The AIM has high internal consistency and good test-retest reliability over a period of two years (Larsen & Diener, 1987; Larsen et al., 1986). In this study, only reactivity scores will be evaluated. Cronbach's α for the emotional reactivity subscale was .82 in the current study.

Difficulties Regulating Negative Emotions. The Difficulties in Emotion Regulation Scale (DERS; Gratz & Roemer, 2004) is a 36-item self-report measure that assesses individuals' typical levels of emotion dysregulation across six domains:

nonacceptance of negative emotions (DERS Nonacceptance), difficulties engaging in goal-directed behaviors when experiencing negative emotions (DERS Goals), difficulties controlling impulsive behaviors when experiencing negative emotions (DERS Impulse), limited access to emotion regulation strategies perceived as effective (DERS Strategies), lack of emotional awareness (DERS Aware), and lack of emotional clarity (DERS Clarity). Participants rate each item using a 5-point Likert-type scale (1 = *almost never*, 5 = *almost always*). Higher scores indicate greater difficulties regulating negative emotions. The DERS demonstrates adequate psychometric properties (Gratz & Roemer, 2004). Cronbach's α was .97 in the current study.

Childhood Trauma. The Childhood Trauma Questionnaire-Short Form (CTQ; Bernstein & Fink, 1998) is a 25-item self-report measure that assesses childhood maltreatment experiences (i.e., "when I was growing up") using a 5-point Likert-type scale (*1 = never true*, *5 = very often true*). The CTQ has five subscales measuring childhood experiences of sexual abuse, physical abuse, emotional abuse, emotional neglect, and physical neglect. Scores for each subscale range from 5 to 25. The CTQ demonstrates good psychometric properties (Bernstein & Fink, 1998). Cronbach's α was .82 in the current study.

Experimental session.

Subjective Emotional Reactivity. The Positive and Negative Affect Scale (PANAS; Watson, Clark, & Tellegen, 1988) consists of two 10-item mood scales and was developed to provide brief measures of positive affect (PA) and negative affect (NA). Respondents were asked to rate the extent to which they had experienced each emotion in that moment on a 5-point Likert-type scale anchored from 1 (very slightly or

not at all) to 5 (very much). The PANAS demonstrates good psychometric properties (Watson, Clark, & Tellegen, 1988). Cronbach's α was .87 in the current study.

Physiological Emotional Reactivity. Recommended guidelines for assessing cortisol response to acute stressors are followed (Nicolson, 2008). Saliva is collected after 1pm to limit the influence of diurnal fluctuations. Participants are instructed to not eat, drink caffeine, or smoke < 60 minutes prior to the study session because of the influence of caffeine, nicotine, and food intake on cortisol. Saliva samples are obtained at two time points during the study: (a) at baseline and (b) 20 minutes-post emotion induction. Emotional reactivity is calculated by assessing the change in cortisol level from baseline to 20-minutes post-emotion induction. Saliva samples are collected by having participant's pool saliva in their mouth and then transfer the saliva into a centrifuge tube with a Salivette. Approximately 0.5 ml of saliva is collected and then sealed and stored in a freezer. All samples are assayed in duplicate for salivary cortisol off-site.

Data Analysis.

Analyses were conducted using IBM SPSS Statistics Version 24.0 (IBM Corporation, 2016). First, assumptions of independence, normality, linearity, and homogeneity of variance were evaluated. Second, descriptive data for the primary study variables was calculated. Third, Pearson product-moment correlations were conducted to evaluate the relations among, PTSD symptom severity (PCL-5), emotion dysregulation (DERS), emotional reactivity (AIM and change in cortisol levels), and childhood traumatic severity (CTQ). Fourth, in order to determine whether aggregating across emotion induction conditions (which would increase power) was possible for further analyses, one-way ANOVAs were calculated to explore between-group differences in

emotion induction conditions on the PANAS and cortisol reactivity scores. Significant ANOVAs were followed up with post hoc comparisons using Tukey HSD tests.

Lastly, moderated mediation analysis were conducted using the PROCESS SPSS macro (Hayes, 2012). First, emotional reactivity was examined as a mediator of the association between PTSD symptom severity and emotion dysregulation. The bootstrap method was used for estimating the standard errors of parameter estimates and the bias-corrected confidence intervals of the indirect effects (MacKinnon, Lockwood, Hoffman, West, & Sheets, 2002; Preacher & Hayes, 2004). The bias-corrected confidence interval is based on a non-parametric re-sampling procedure that has been recommended when estimating confidence intervals of the mediated effect due to the adjustment it applies over a large number of bootstrapped samples (Efron, 1987). The mediated effect is significant if the 95% confidence interval does not contain zero (Preacher & Hayes, 2004). In this study, 5,000 bootstrap samples will be used to derive estimates of the indirect effect.

Following this, a moderated mediation model was tested, whereby the influence of severity of childhood traumatic events was examined in the associations among PTSD symptom severity (independent variable), emotional reactivity (mediating variable), and emotion dysregulation (dependent variable). Moderated mediation occurs when either path *a* (from PTSD to emotional reactivity) or path *b* (from emotion reactivity to emotion regulation), or both are moderated (Edwards & Lambert, 2007). This model examined whether childhood trauma moderates the paths in the mediation model (*a*, *b*, and *c* [from PTSD to emotion regulation]) as well as the indirect effect (*a* x *b*).

Chapter 3.

FINDINGS

Preliminary Analyses.

Study variables were assessed for assumptions of normality using recommendations set by Tabachnick and Fidell (2007). All variables met acceptable standards for skewness and kurtosis, except salivary cortisol reactivity (skewness = 9.65, kurtosis = 98.60). The SPSS PROCESS macro, which employs the bootstrapping technique, does not require any assumptions be made with regard to the shape of the sampling distribution (Preacher, Rucker, & Hayes, 2007). Thus, the raw salivary cortisol reactivity variable was used in the study analyses.

In this sample, 46.3% (n = 62) of women reported symptoms consisted with a diagnosis of PTSD, with PTSD symptom scores ranging from 0 to 80 (M = 33.84, SD = 21.61). Almost half (49.0%; n = 77) of women reported any experience of childhood trauma, with childhood trauma scores ranging from 29 to 113 (M = 58.07, SD = 20.49). Emotion dysregulation scores ranged from 10 to 116 (M = 68.38, SD = 19.45). The average PANAS Total score was 23.63 (SD = 13.07) at baseline and 24.95 (SD = 13.91) at post-induction. The average PANAS Positive score was 16.81 (SD = 9.38) at baseline and 17.16 (SD = 10.95) at post-induction. The average PANAS Negative score was 6.82 (SD = 8.10) at baseline and 7.79 (SD = 9.81) at post-induction. The average cortisol level was 0.23 (SD = 0.47) at baseline and 0.21 (SD = 0.56) post-induction. Descriptive statistics regarding variables of interest presented in Table 3.

Pearson correlations were calculated to examine zero-order associations among the primary study variables (see Table 4). Results indicated that there were significant

positive associations between PTSD symptoms and both emotion dysregulation (r = .35, p < .001) and childhood trauma (r = .38, p < .001). Significant positive associations were also found between the PANAS Total reactivity score and both the PANAS Positive reactivity score (r = .63, p < .001) and the PANAS Negative reactivity score (r = .65, p < .001). Significant negative associations were found between the PANAS Positive reactivity score and both the PANAS Negative reactivity score (r = -.18, p < .05) and childhood trauma (r = -.20, p < .05). Finally, a significant positive association was found between emotion dysregulation and childhood trauma (r = .28, p < .001).

Next, one-way ANOVAs were calculated to explore between-group differences in emotion induction conditions on the PANAS reactivity scores and cortisol reactivity (see Table 5). Results showed significant differences on the PANAS reactivity scores as a function of emotion induction condition: PANAS Total reactivity score, F(2,112) = 11.05, p < .001, PANAS Positive reactivity score, F(2,112) = 23.33, p < .001, and PANAS Negative reactivity score, F(2,112) = 9.26, p < .001. Significant differences in cortisol as a function of emotion induction condition were not detected, F(2,99) = 1.75, p = .18.

Significant ANOVAs were followed up with post hoc comparisons using Tukey HSD tests (see Table 6). Post hoc comparison of the PANAS Total score indicated that the mean reactivity scores for the positive (M = 6.77, SD = 7.75) and negative (M = 2.09, SD = 12.67) emotion induction conditions were significantly higher than the mean reactivity score for the neutral emotion induction condition (M = -3.58, SD = 6.54). Post hoc comparisons for the PANAS Positive score indicated that the mean reactivity score for the positive emotion induction condition (M = 7.48, SD = 7.64) was significantly

higher than the mean reactivity scores for the negative (*M* = -2.82, *SD* = 7.37) and neutral (*M* = -1.69, *SD* = 5.52) emotion induction conditions. Post hoc comparisons for the PANAS Negative score indicated that the mean reactivity score for the negative emotion induction condition (*M* = 4.92, *SD* = 11.59) was significantly higher than the mean reactivity scores for the positive (*M* = -.70, *SD* = 3.24) and neutral (*M* = -1.88, *SD* = 4.43) emotion induction conditions. These findings indicate a similar pattern of findings for the entire sample compared to the subsamples that were assigned to the negative and positive emotion induction conditions, suggesting that analyses using the entire sample would be appropriate to test the current study aims.

Primary Analyses.

Mediation and moderated mediation models were conducted to further explore the relations among PTSD symptoms, emotional reactivity, emotion dysregulation, and childhood traumatic severity (see Table 7).

In a mediation model, as seen in Figure 1, the PANAS Total reactivity score did not significantly mediate the relationship between PTSD symptoms and emotion dysregulation, *B* = -.003, *SE* = .02, 95% CI (-.05, .02). In a moderated mediation model, as seen in Figure 2, childhood trauma did not moderate the relations between (a) PTSD symptoms and emotion dysregulation, path a_i; *B* = -.00, *SE* = .00 , 95% CI (-.00, .00), (b) PANAS Total reactivity and emotion dysregulation, path b_i *B* = -.30 , *SE* = .60 , 95% CI (-1.50, .90), or (c) PTSD symptoms and emotion dysregulation, path c_i *B* = .00, *SE* = .01, 95% CI (-.01, .03). The index of moderated mediation (difference between conditional indirect effects) was non-significant at the one standard deviation below, *B* = .01, *SE* = .01, 95% CI (-.14, .14), and above, *B* = .00, *SE* = .02, 95% CI (-.00, .07), mean levels of

childhood trauma, but was significant at the mean level of childhood trauma, *B* = -.01, *SE* = .08, 95% CI (.12, .20). These results indicate that subjective emotional reactivity explained the relation between PTSD symptoms and emotion dysregulation, but only at the mean level of childhood trauma.

Similarly, as seen in Figure 3, cortisol reactivity did not significantly mediate the relation between PTSD symptoms and emotion dysregulation, *B* = .004, *SE* = .01, 95% CI (-.001, .02). In a moderated mediation model, as seen in Figure 4, severity of childhood trauma did not moderate the relations between PTSD symptom severity and emotion dysregulation, path a_i; *B* = -.00, *SE* = .00, 95% CI (-.00, .00), or cortisol reactivity and emotion dysregulation, path b_i; *B* = -.13, *SE* = .19, 95% CI (-.52, .25). Childhood trauma did moderate the relation between PTSD symptom severity and emotion dysregulation, path c_i; *B* = -.01, *SE* = .00, 95% CI (-.02, -.00), such that the strength of the relation between PTSD symptom severity and emotion dysregulation was weaker at higher (vs. lower) levels of childhood trauma severity. The index of moderated mediation (difference between conditional indirect effects) was non-significant at the one standard deviation above, *B* = -.00, *SE* = .04, 95% CI (-.04, .04), below, *B* = .04, *SE* = .06, 95% CI (-.03, .21), and at mean levels of, *B* = -.01, *SE* = .07, 95% CI (-.08, .09), childhood trauma. These results indicate that mediation and moderated mediation analyses for objective emotional reactivity were not significant.

Chapter 4.

DISCUSSION

The goal of this study was to explore the roles of childhood trauma and emotional reactivity in the relation between PTSD symptom severity and emotion dysregulation. This study extends research in two critical ways. First, it is the first empirical investigation of the mediating role of both objective and subjective measures of emotional reactivity in the relation between PTSD symptom severity and emotion dysregulation. Second, despite theoretical and empirical evidence to suggest that trauma exposure affects the associations between (a) PTSD and emotion dysregulation (Weiss et al., 2013), (b) PTSD and emotional reactivity (Tull et al., 2007), and (c) emotional reactivity and emotion dysregulation (Bellis & Zisk, 2014), no investigations to date have examined the role of childhood trauma in the associations among PTSD, emotion dysregulation, and emotional reactivity. Consistent with past research, significant zero-order positive associations were found between PTSD symptom severity, emotion dysregulation, and childhood trauma. However, emotional reactivity (neither objective nor subjective) was not found to be significantly associated with the other primary study variables at zero-order, with the exception of a significant negative association between positive subjective emotional reactivity and childhood trauma. Inconsistent with study hypothesis, emotional reactivity did not significantly mediate the relationship between PTSD symptoms and emotion dysregulation. Partially consistent with study hypothesis, childhood trauma moderated the associations among PTSD, emotional reactivity, and emotion dysregulation, such that emotional reactivity mediated the relationship between

PTSD symptom severity and emotion dysregulation at mean (but not high or low) levels of childhood trauma. These findings have important implications for future research and practice in this area, described below.

In the current sample of women who experienced IPV, 41.4% of women reported symptoms consistent with PTSD and 49.0% reported experiencing childhood trauma. Overall, high rates of both childhood trauma and adult PTSD appear to be consistent for women who experience IPV in similar samples (Becker, Stuewig, & McCloskey, 2010; Brown, Burnette, & Cerulli, 2015). Not surprisingly, research shows strong associations among childhood trauma, IPV, and PTSD in adulthood, emphasizing the importance of further evaluating the associations among these variables. For instance, Wuest et al. (2009) found that both trauma and IPV significantly and independently contributed to PTSD symptoms in adulthood, while others (e.g., Becker, et al., 2010) have found that IPV mediates the relationship between child physical abuse and adult PTSD symptom severity. Research shows that emotion dysregulation appears to be an underlying mechanism that links childhood trauma and PTSD in women who experience IPV (Lily, London, & Bridgett, 2014). Indeed, theoretical frameworks suggest that childhood trauma disrupts the development of appropriate emotion regulation skills, which can subsequently influence PTSD symptom severity and revictimization (e.g., IPV; Cicchetti & White, 1990; Shields & Cicchetti, 1998). In the current sample, rates of emotion dysregulation (as assessed by DERS), averaged 68.38. In similar samples, levels of emotion dysregulation are somewhat higher (M = 81.10, Grigorian et al., 2019). Considering the theoretical frameworks that support the role of emotion dysregulation in PTSD, emotional reactivity, and childhood trauma, future research is needed to better

understand the factors that contributed to lower than average levels of emotion dysregulation in this sample of women who experience IPV. For instance, existing research on women who experience IPV has generally focused on clinical samples. It is possible that there is greater variability in emotion dysregulation among women who experience IPV in the community.

Mediation results for this study showed that emotional reactivity did not significantly account for the relation between PTSD symptom severity and emotion dysregulation. Theoretical and empirical research highlights the role of emotional reactivity in the associations between PTSD and emotion dysregulation (Fitzgerald et al., 2018; Kaczmarek & Zawadzki; 2012; Spiller et al., 2019; Strelau & Zawadzki, 2005; Wisco et al., 2018). Although studies show increased levels emotional reactivity immediately following a traumatic event, emotional reactivity seems to decrease overtime. These changes are often attributed to desensitization of the biological processes that underlie emotional reactivity (i.e., HPA axis function) following repeated exposure to stressful or traumatic events (Pitman, et al., 2012). Thus, childhood traumatic events, followed by revictimization in adulthood (e.g., IPV) accompanied by PTSD symptoms, could result in progressive desensitization of the HPA axis, resulting in low subjective and objective reports of emotional reactivity. Alternatively, the relationship between PTSD symptom severity and emotion dysregulation could be mediated through other important mechanisms. Indeed, in addition to emotional reactivity (most closely linked to the Arousal and Reactivity PTSD symptom cluster), other PTSD symptom clusters could be significantly associated with emotion dysregulation. A recent study found that the Negative Alterations in Cognition and Mood PTSD symptom cluster showed the

strongest association to overall emotion dysregulation (Seligowski, Rogers, & Orcutt, 2016). As such, it is possible that negative thoughts (e.g., overly negative thoughts and assumptions about oneself or the world) may have played a more significant role in the associations within the current study. Furthermore, as previously discussed, levels of emotion dysregulation were relatively low in comparison to similar samples. As such, lower levels of emotion dysregulation could have affected the strength of the associations. Future investigations are needed to test the above-mentioned hypotheses.

Notably, results of this study show that childhood trauma moderated the associations among PTSD symptom severity, subjective emotional reactivity, and emotion dysregulation. Specifically, emotional reactivity was found to mediate the relation between PTSD symptom severity and emotion dysregulation at mean (but not high or low) levels of childhood trauma. Theoretical framework suggests that emotion dysregulation stems from emotional vulnerabilities within an invalidating environmental context (Linehan, 1993). The current study assessed one form of an emotional vulnerability (i.e., emotional reactivity) and one form of an invalidating environment (i.e., childhood trauma). Findings are counter to theory, which suggest that emotional vulnerability (assessed as emotional reactivity here) would mediate the link between PTSD symptom severity and emotion dysregulation at high levels of environmental invalidation (assessed as childhood trauma here). One explanation for these findings is that this study narrowly assessed emotional vulnerability and invalidating environmental context, and other facets of these constructs may play a more critical role in these associations. Two forms of emotional vulnerability worth studying in this regard are emotional intensity and emotional duration. For instance, one study found that trauma-

exposed individuals with more severe PTSD symptoms reported elevated levels of negative affect intensity (Vujanovic et al., 2013). Likewise, Spiller (2019) found that trauma-exposed individuals experience slow return to emotional baseline following an emotional event in the context of high levels of PTSD symptoms and emotion dysregulation. Emotional intensity and emotional duration have also been linked to emotion dysregulation (Crowell, et al., 2009; Linehan, 1993). Alternatively, it is also possible that other forms of environmental invalidation (e.g., criticism, minimization, or rejection) may have influenced these associations. Indeed, Linehan's biosocial theory (1993) posits that emotion dysregulation may stem from a wide range of invalidating environmental contexts that do not provide the opportunity for individuals to practice modulating emotional arousal or coping with distress (Crowell, et al., 2009; Eisenberg, Cumberland, & Spinard, 1998; Linehan, 1993; Shipman, Zeman, Nesin, & Fitzgerald, 2003). In sum, these findings underscore the need for comprehensively examining emotional vulnerabilities (e.g., emotional reactivity *and* intensity and duration) and invalidating environments (e.g., abuse *and* criticism, minimization, and rejection).

There are additional explanations as to why findings for the moderated mediation model diverged from expectations. For instance, it is possible that specific forms of childhood trauma play a more influential role in the associations in question. For instance, two recent studies found that childhood emotional abuse was significantly positively correlated to emotion dysregulation, while other types of childhood traumatic events (e.g., physical or sexual abuse) were not (Lilly, et al., 2014; Weiss, et al., 2013). Future research would benefit from evaluating whether the specific forms of childhood trauma (e.g., physical/sexual/emotional abuse and physical/emotional neglect)

differentially attenuate the relations among PTSD symptom severity, emotional reactivity, and emotion dysregulation. Alternatively, although theoretical frameworks and subsequent research supports the notion that childhood trauma may result in impairment in developmental processes related to emotion dysregulation through alterations to the biological process that underlie emotional reactivity (Banyard & Williams, 1996; Bellis & Zisk, 2014; Browne & Finkelhor, 1986; Cloitre, 1998; Leahy et al., 2004; Shields & Cicchetti, 1998; Shipman, et al., 2005; Shipman, et al., 2000), the current sample may not be representative of the samples investigated in the studies that support this theory. Indeed, the current sample is characterized by high levels of revictimization, as half of the participants reported childhood trauma and all participants reported current adult victimization in an intimate relationship. Replication of the current study findings in populations characterized by high rates of revictimization is needed in order to further investigate the associations in question.

Finally, it warrants mention that subjective and objective measures of emotional reactivity were not significantly associated with each other. Although both subjective and objective measures have been empirically shown to index emotional reactivity in the laboratory, several factors might influence the current findings. The validity of salivary cortisol in the assessment of emotional reactivity may be influenced by the nature of this sample. As previously discussed, findings with regard to salivary cortisol in trauma-exposed populations are generally inconsistent in literature (Elzinga et al., 2003). Relatedly, rather than stress, other factors, such as the circadian patter of cortisol secretion (Kalman & Grahn, 2004), may have affected the levels of salivary cortisol, regardless of the presence or absence of trauma and/or other forms of psychopathology.

Specifically, the typical circadian pattern of cortisol secretion shows an increase in the early morning hours that peaks at or slightly before the time of waking. However, depending on the strength of the stimulus (e.g., stressor), cortisol levels in the afternoon and evening can be elevated above those of the circadian peak (Kalman & Grahn, 2004). Relatedly, cortisol levels in trauma-exposed individuals may also vary as function of childhood trauma type and severity (Meewisse et al., 2007; Schalinski, Steudte-Schmiedgen, & Kirschbaum, 2015), as well as in relation to PTSD in adulthood (Burri, Maercker, Krammer, & Simmen-Janevska, 2013). For instance, a study that evaluated the relation between different types of childhood trauma and cortisol levels showed that sexual abuse was more strongly associated to high cortisol levels when compared to emotional abuse (Yehuda, Halligan, & Grossman, 2001). Further, Yehuda et al. (2001) found that the presence of childhood sexual abuse and absence of PTSD was associated with high levels of cortisol, while the presence of both PTSD and childhood sexual abuse were associated with low cortisol levels. These findings suggest that subjective measures may more accurately capture emotional reactivity than objective measures within trauma-exposed populations, particularly those with high rates of PTSD. Moreover, they highlight the complexity of salivary cortisol as an objective measure of emotional reactivity.

Although the present study adds to the growing body of literature on PTSD, emotional reactivity, emotion dysregulation, and childhood trauma, several limitations must be considered. First, the cross-sectional and correlational nature of the data precludes determination of the nature and precise direction of the relations examined. Although theoretical frameworks and an extensive body of literature support the

directionality in the associations in the current study (Larsen & Diener, 1987; Vujanovic et al., 2013), it is indeed possible that the associations are reciprocal. Future research is needed to further investigate the nature and direction of these relations through prospective, longitudinal investigations. Second, the relations among PTSD, emotional reactivity, emotion dysregulation, and childhood traumatic events were evaluated in a community sample of substance using women who experience IPV. As such, these findings cannot be assumed to generalize to other IPV (e.g., men, women in same-sex relationships) or trauma-exposed (e.g., clinical) populations. Future research is needed to replicate such findings in other populations. Third, the relatively small sample size may have not been powered to detect a significant effect. A larger sample size is needed in order to better evaluate the associations in question. Lastly, although preliminary data supported an aggregating across emotion induction conditions, doing so may have accounted for the resulted patterns of findings. Examination of these relations in positive and negative emotion induction conditions in necessary.

Despite these limitations, results address important gaps in the literature with regard to the role of childhood trauma in the relations among PTSD symptom severity, emotional reactivity, and emotion dysregulation. The findings of this study have important implications for clinical practice. First, they suggest that assessing for levels of childhood trauma among women who experience IPV may identify those with or at risk for developing emotion dysregulation. Among women who experience IPV with moderate levels of childhood trauma and high levels of PTSD symptoms, emotional reactivity may be an important treatment target for reducing emotion dysregulation. For instance, among these women, findings may inform prevention efforts aimed at reducing

disruptions in the processes that underlie the development of emotional reactivity and ultimately emotion dysregulation. Future studies are needed to better understand the utility of targeting emotional reactivity in treatments aimed at reducing emotion dysregulation in this population, and the consequences of these potential reductions on other clinically relevant outcomes associated with emotion dysregulation (Pacella, et al., 2013, Rytwinski, et al., 2013; Tull, et al., 2015; Weiss, et al., 2013; Weiss, et al., 2015).

Table 1. Sample characteristics

	M (SD)	*n (%)*
Age	40.98 (11.69)	
Gender		
Female		124 (92.5%)
Female to Male Transgender		1 (0.7%)
Male to Female Transgender		2 (1.5%)
Gender Queer		1 (0.7%)
Ethnicity		
Hispanic/Latinx		25 (18.7%)
Not Hispanic/Latinx		94 (70.1%)
Race		
White		51 (40.5%)
African American/Black		37 (29.4%)
American Indian/Alaskan Native		10 (7.9%)
Hispanic/Latinx		15 (11.9%)
Other		13 (10.3%)
Employment Status		
Full-time		8 (6.0%)
Part-time		13 (9.7%)
Unemployed		98 (73.1%)
Annual Household Income		
Less than $15,000		122 (91.0%)
$15,000 - $24,999		1 (0.7%)
$25,000 - $34,999		1 (0.7%)
Relationship Status		
Married		11 (8.2%)
Unmarried (in a relationship)		95 (70.9%)
Separated or Divorced		12 (9.0%)
Relationship Length (Months)	65.17 (71.64)	
Contact with Partner (Mean Days Per Week)	5.82 (3.27)	
Probable PTSD Diagnosis		62 (46.3%)

Note: PTSD = posttraumatic stress disorder.

Table 2. Index of traumatic events

Potentially Traumatic Events	*n* %
Physical assault (e.g. being attacked, hit, slapped, kicked, beaten up)	88 (56.1%)
Transportation accident (e.g. car accident, boat accident, train wreck, plane crash)	71 (45.2%)
Sexual assault (e.g. rape, attempted rape, made to perform any type of sexual act through force or threat of harm)	71 (45.2%)
Other unwanted or uncomfortable sexual experience	65 (41.4%)
Assault with a weapon (e.g. being shot, stabbed, threatened with a knife, gun, bomb)	61 (38.9%)
Sudden accidental death	52 (33.1%)
Any other very stressful event or experience	52 (33.1%)
Life-threatening illness or injury	51 (32.5%)
Fire or explosion	49 (31.2%)
Sudden violent death (e.g. homicide, suicide)	49 (29.9%)
Serious accident at work, home, or during recreational activity	45 (28.7%)
Natural disaster (e.g. flood, hurricane, tornado, earthquake)	42 (26.8%)
Severe human suffering	42 (26.8%)
Captivity (e.g. being kidnapped, abducted, held hostage, prisoner of war)	37 (23.6%)
Serious injury, harm, or death you caused to someone else	30 (19.1%)
Combat or exposure to a war-zone (i.e. in the military or as a civilian)	21 (13.4%)
Exposure to toxic substance (e.g. dangerous chemicals, radiation)	18 (11.5%)

Table 3. Descriptive statistics regarding variables of interest

	M	*SD*	Median	Range	Skew	Kurtosis
PCL-5	33.84	21.62	35	0 – 80	0.16	-0.71
Δ PANAS Total	1.39	10.36	1	-42 – 29	-0.31	2.32
Δ PANAS – Positive Affect	0.42	8.03	-0.50	-29 – 19	-0.11	0.86
Δ PANAS – Negative Affect	0.97	8.21	0	-30 – 36	0.55	4.94
Δ Cortisol	0.03	1.19	-0.32	-1.70 – 12.09	9.65	98.60
DERS	68.38	19.45	69	10 – 116	-0.07	-0.06
CTQ	58.07	20.49	56	29 – 113	0.54	-0.25

Note: PCL-5 = PTSD Symptom Checklist for DSM-V; PANAS = Positive and Negative Affect Schedule; DERS = Difficulties in Emotion Regulation Scale; CTQ = Childhood Trauma Questionnaire.

Table 4. Bivariate correlations regarding variables of interest (overall sample)

	1	2	3	4	5	6	7
1. PCL-5	-	-.30	-.00	-.05	-.11	.35**	.38**
2. Δ PANAS Total		-	.63**	.65**	.07	.08	-.10
3. Δ PANAS – Positive Affect			-	-.18*	.13	.10	-.20*
4. Δ PANAS – Negative Affect				-	-.04	-.00	.06
5. Δ Cortisol					-	-.08	.13
6. DERS						-	.28**
7. CTQ							-

Note: PCL-5 = PTSD Symptom Checklist for DSM-V; PANAS = Positive and Negative Affect Schedule; DERS = Difficulties in Emotion Regulation Scale; CTQ = Childhood Trauma Questionnaire.

Table 5. Summary of ANOVA findings regarding differences between emotion induction conditions on variables of interest

	df	*F*	*p*
Δ PANAS Total	112(2)	11.05	< .001
Δ PANAS –Positive Affect	112(2)	23.33	< .001
Δ PANAS –Negative Affect	112(2)	9.26	< .001
Δ Cortisol	99(2)	1.75	.178

Note: PANAS = Positive and Negative Affect Schedule.

Table 6. Summary of Tukey HSD post hoc tests comparing emotion induction conditions on variables of interest

	Emotion Condition		Mean Difference	Std. Error	*p*	95% *CI*
Δ PANAS Total	Positive	Negative	4.67	2.25	.09	-.67, 10.02
	Positive	Neutral	10.35*	2.22	< .001	5.05, 15.65
	Negative	Neutral	5.67*	2.06	.01	.77, 10.58
Δ PANAS – Positive	Positive	Negative	10.31*	1.62	< .001	6.45, 14.16
	Positive	Neutral	9.18*	1.60	< .001	5.36, 12.99
	Negative	Neutral	-1.13	1.48	.72	-4.66, 2.40
Δ PANAS – Negative	Positive	Negative	-5.63*	1.81	.007	-9.94, -1.32
	Positive	Neutral	1.17	1.79	.791	-3.09, 5.44
	Negative	Neutral	6.81*	1.66	< .001	2.85, 10.76
Δ Cortisol	Positive	Negative	.43	.30	.33	-.29, 1.17
	Positive	Neutral	.56	.30	.16	-.17, 1.30
	Negative	Neutral	.12	.28	.89	-.54, .79

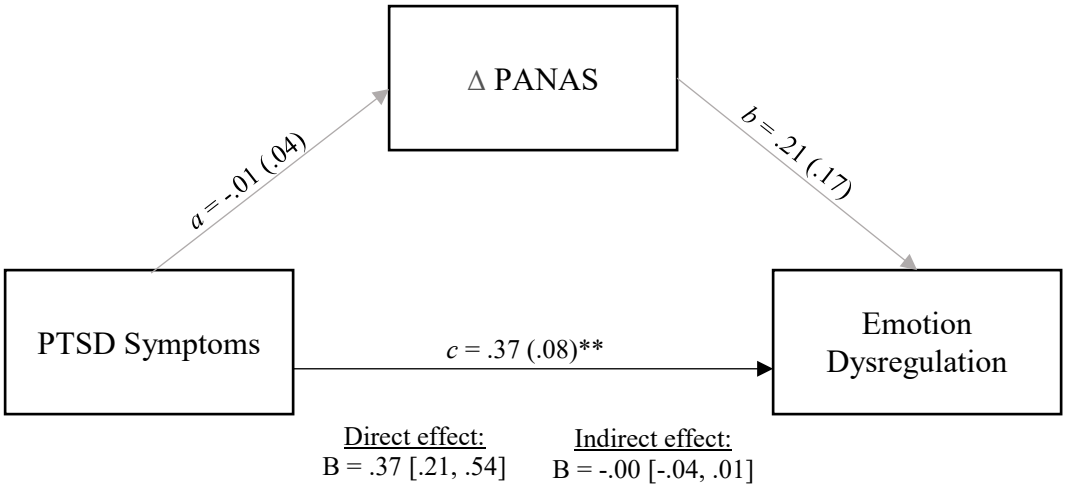
Note: *$p < .05$. PANAS = Positive and Negative Affect Schedule

Table 7. Summary of moderation and mediation analysis

Mediation and Moderation Paths	*B*	*SE*	*t*	*p*	*CI*
Model 1 - PANAS					
PTSD → Δ PANAS Total (*a*)	-.00	.00	-.68	.49	-.01, .00
PTSD x CTQ → Δ PANAS Total (a_i)	-.00	.00	-1.42	.15	-.00, .00
Δ PANAS Total → ED (*b*)	2.37	6.03	.39	.69	-9.60, 14.36
Δ PANAS Total x CTQ → ED (b_i)	-.30	.60	-.49	.62	-1.50, .90
PTSD → ED (*c*)	.38	.09	3.92	.00	.19, .58
PTSD x CTQ → ED (c_i)	.00	.01	.66	.50	-.01, .03
Model 2 – Cortisol					
PTSD → Δ Cortisol (*a*)	.01	.02	.83	.40	-.02, .06
PTSD x CTQ → Δ Cortisol (a_i)	-.00	.00	-1.51	.13	-.00, .00
Δ Cortisol → ED (*b*)	8.79	14.93	.58	.55	-20.90, 38.50
Δ Cortisol x CTQ → ED (b_i)	-.13	.19	-.68	.49	-.52, .25
PTSD → ED (*c*)	1.02	.30	3.33	.00	.41, 1.63
PTSD x CTQ → ED (c_i)	-.01	.00	-2.22	.02	-.02, -.00
Indirect Effects					
Model 1 - Δ PANAS Total					
CTQ < 1 SD	.01	.10			-.14, .14
CTQ Mean	-.01	.08			.12, .20
CTQ > 1 SD	.00	.02			-.00, .07
Model 2 – Δ Cortisol					
CTQ < 1 SD	-.00	.04			-.04, .04
CTQ Mean	-.01	.07			-.08, .09
CTQ > 1 SD	.04	.06			-.03, .21

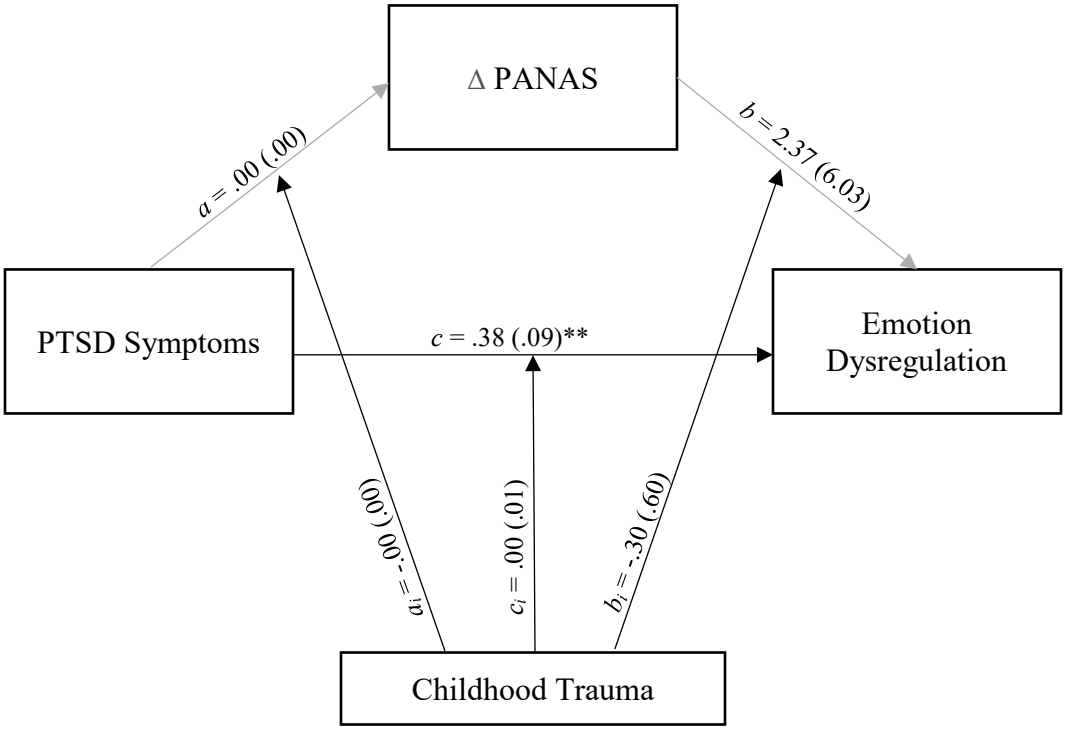
Note: PTSD = posttraumatic stress disorder; ED = emotion dysregulation; PANAS = Positive and Negative Affect Schedule; CTQ = Childhood Trauma Questionnaire.

Figure 1. Summary of mediation analysis explicating the mediating role of subjective emotional reactivity on the association between PTSD symptom severity and emotion dysregulation



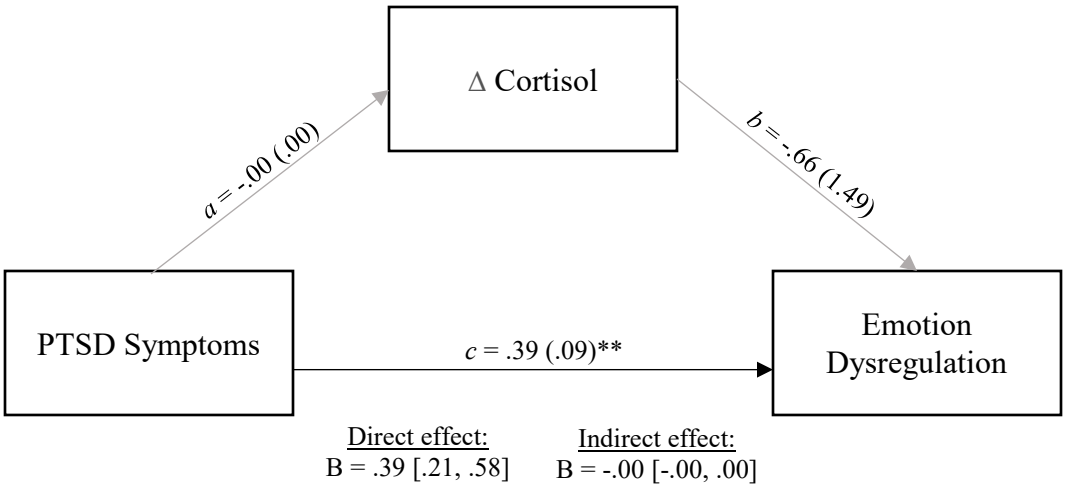
Note: **$p < .001$. PTSD = posttraumatic stress disorder; PANAS = Positive and Negative Affect Schedule.

Figure 2. Summary of moderated mediation analysis explicating the mediating role of subjective emotional reactivity and the moderating role of childhood traumatic experiences on the association between PTSD symptom severity and emotion dysregulation



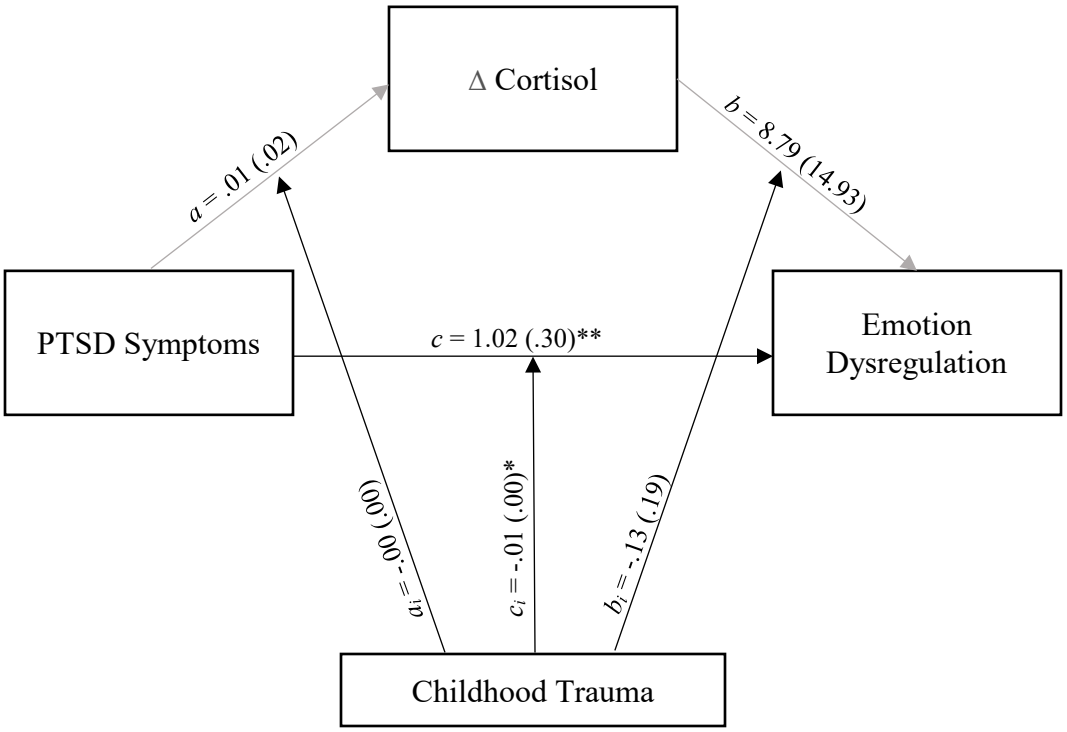
Note: **$p < .001$. PTSD = posttraumatic stress disorder; PANAS = Positive and Negative Affect Schedule.

Figure 3. Summary of mediation analysis explicating the mediating role of objective emotional reactivity on the association between PTSD symptom severity and emotion dysregulation



Note: **$p < .001$. PTSD = Posttraumatic Stress Disorder.

Figure 4. Summary of moderated mediation analysis explicating the mediating role of objective emotional reactivity and the moderating role of childhood traumatic experiences on the association between PTSD symptom severity and emotion dysregulation



Note: * $p < .05$. ** $p < .001$. PTSD = Posttraumatic Stress Disorder.

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