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Mechanisms for Depression Risk Among those with Sexual Abuse Histories: Stress Sensitivity and Emotion Regulation Deficits

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Mechanisms for Depression Risk Among those with Sexual Abuse Histories: Stress
Sensitivity and Emotion Regulation Deficits

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MECHANISMS FOR DEPRESSION RISK AMONG THOSE WITH SEXUAL ABUSE
HISTORIES: STRESS SENSITIVITY AND EMOTION REGULATION DEFICITS

ANGELA H. BUSH

ABSTRACT

The present study examined whether childhood sexual abuse (CSA) predicts depression-related outcomes indirectly via stress sensitivity and emotion regulation (ER) deficits or whether ER difficulties mediate the relationship between CSA and stress sensitivity. Both stress sensitivity and ER deficits have been examined as mechanisms that increase depression risk for those with CSA histories, however their interplay has been largely understudied. Competing models of CSA, stress sensitivity, ER deficits and depression symptoms and affects in daily life were examined. Community dwelling and undergraduate participants (N=99) reported CSA histories via a psychosocial interview, completed measures of trait ER repertoires, perceived stress and depression symptoms, and a 7-day Ecological Momentary Assessment (EMA) protocol that measured hourly positive (PA) and negative affect (NA) and stress. Multiple mediation models and moderated mediation models were fit to examine study hypotheses. Results suggest that those with CSA histories evidence stress sensitivity for depressive affects, but not depression symptoms. Further, those with such histories tend to exhibit more maladaptive ER repertoires, but this experience was unrelated to adaptive repertoires. Maladaptive ER repertoires mediated the effects of CSA on depression symptoms as well as the effects of CSA on depressive affects via stress sensitivity. Findings emphasize the adverse effects of maladaptive ER responses within this population and suggest that these responses should be a key target of treatment for these individuals.

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CHAPTER I

INTRODUCTION

Childhood sexual abuse (CSA) is a negative life event that involves unwanted sexual contact with a minor, and is frequently perpetrated by someone considered to be in a position of responsibility or power (World Health Organization, 1999). Acts involved in CSA can include physical touching or force, such as completed or attempted sexual acts, sexual touching, and assaults with no contact such as harassment, threats, forced participation in pornography, and taking sexual images that are unwanted (Murray, Nguyen & Cohen, 2014). These experiences during childhood and adolescence are relatively common, as evidenced by their global prevalence of 18-19.7% among girls and 7.6%-7.9% among boys (Stoltenborgh et al., 2011; Pereda et al., 2009). Prevalence rates in the United States mirror the international trends, with 12%-20% of girls and 5%-8% of boys experiencing unwanted sexual contact and assaults that do not involve contact or penetration (Bolen & Scannapieco, 1999; Finkelhor, Turner, Shattuck, Sherry & Hamby, 2015; Gorey & Leslie, 1997). Prevalence rates for CSA are likely higher than those estimated, given the stigmatizing nature of CSA and the reticence for those affected to report the abuse (Katzenstein & Fontes, 2017). Sexual abuse during youths' formative

years is associated with far reaching negative functional outcomes that are burdensome to both the victim of CSA and society as a whole. For example, youths who experience sexual abuse evidence a blunted academic trajectory in the form of worse performance during primary and secondary education, and lower scores on standardized placement tests than their abuse-free peers (Boden et al., 2007). As a consequence, those with CSA histories are less likely to seek post-secondary education (Boden et al., 2007), which limits their earning potential and ability to maintain gainful employment (Gilbert, Widom, Browne, Fergusson, Webb & Janson, 2009). For society, CSA generates an economic burden in the order of \$9.3 billion worldwide. These costs arise from medical and psychological treatment expenses, lost work productivity, educational supports for the victim, suicide attempts and completion, and death related to CSA (Letourneau, Brown, Fang, Hassan & Mercy, 2018). This estimate is conservative, as it is not possible to calculate the true impact of pain, suffering and reduced quality of life experienced by those with CSA histories (Wang & Holton, 2007).

Sexual trauma during childhood and adolescence is also linked to impaired interpersonal functioning that extends into adulthood. For example, youths who experienced sexual abuse between the ages of 5 and 18 years evidence more difficulty than their peers in forming and maintaining social and romantic relationships, as well as engaging in recreational activities with their peers (Jackson, et al., 1990). Additionally, childhood trauma has been linked to decreased social competence from kindergarten to early adulthood (Raby et al., 2018). These findings mirror a corpus of work showing that survivors of CSA report less satisfaction in their intimate relationships (DiLillo & Long, 1999; Lassri, Luyten, Fonagy & Shahar, 2018), have more sexual partners (Beitchman et

al., 1992; Merrill, Guimond, Thomsen & Milner, 2003), and evidence higher rates of separation and divorce than those without CSA histories (Colman & Widom, 2004; Finkelhor et al., 1990).

Importantly, histories of CSA are associated with an increased likelihood for re-victimization. For instance, those with CSA histories are more likely to report unwanted fondling, oral-genital contact and intercourse by someone they know, and to experience sexual assault from strangers than those who are free of CSA histories (Filipas & Ullman, 2006; Messman-Moore & Long, 2000). Increased risk for re-victimization is apparent across college, clinical and community samples, and extends to risk for physical assault (Messman & Long, 1996). In particular, growing evidence suggests that CSA is related to intimate partner violence in adulthood, whereby those with CSA histories are more likely to both experience and perpetrate physical and emotional violence towards their romantic partner (Richards, Tillyer & Wright, 2017).

It is not surprising therefore that sexual abuse during the formative years is also associated with an array of enduring behavioral and emotional disturbances that follow those victimized into adulthood (Boroughs et al., 2015; Hahn, Simons & Simons, 2016; Silverman, Reinherz and Giaconia, 1996). That is, those affected by CSA engaged in more sexualized behaviors such as sexualized play with dolls, public masturbation, and age inappropriate sexual knowledge, as well as seductive behavior and requests for sexual stimulation from other children or adults (Kendall-Tackett et al., 1993).

Additionally, childhood maltreatment and CSA have been linked to increased sexual risk taking, including number of casual sex partners, intercourse without contraception and contraction of STIs (Boroughs et al., 2015; Hahn, Simons & Simons, 2016) Conversely,

CSA is also associated with a decrease in sexual functioning in the form of decreased sex drive, dissatisfaction during sexual acts, and sexual withdrawal (Beitchman et al., 1992; Lemieux & Byers, 2008; Rellini, 2014). Youths with CSA also evidence externalizing behaviors in the form of aggression and behavioral problems during their childhood years (Wang et al., 2019; Kendall-Tackett et al., 1993) that are maintained in adulthood (Trabold, Swogger, Walsh & Cerulli, 2015; Wilson, 2010) and extend to substance use and antisocial behaviors (Boroughs et al., 2015; Johnson, Sheahan & Chard, 2004; Wilson, 2010). Further, CSA is associated with internalizing problems across the developmental span, with CSA survivors evidencing more social withdrawal, higher anxiety and depression symptoms, and greater prevalence of anxiety and depressive disorders than their peers (Gilbert et al., 2009; Kendall-Tackett et al., 1993; Wilson, 2010).

1.1 Childhood Sexual Abuse and Depression

While sexual abuse has been linked to a number of adverse mental health problems (Gilbert et al., 2009; Kendall-Tackett et al., 1993; Wilson, 2010), a large literature suggests that depressive disorders are a common outcome of this experience. Depressive disorders include Major Depressive Disorder (MDD) and Persistent Depressive Disorder (PDD), and are characterized by depressed mood, reduced interest in pleasurable activities, decreased cognitive functioning and vegetative symptoms (APA, 2013; Otte et al., 2016). Depressive disorders are common, affecting 8.3% to 19.2 % of the US population (Kessler, 2014), and often feature life-long, episodic courses that are marked by periods of remission and recurrences (Fava & Kendler, 2000). When present, depressive disorders are associated with significant functional impairment that results in

the suffering of those affected and their loved ones, as well as increases in costs to society that are linked to lost productivity and treatment of depressive disorders. Indeed depressed persons report having lower quality interpersonal relationships than their healthy peers (Zlotnick et al., 2000), and increased likelihood to attempt and complete suicide (Weissman et al., 1999). Not surprisingly, depressive disorders are the leading contributor to disability across the globe (WHO, 2017). Additionally, individuals with depressive disorders are more likely to have comorbid affective, externalizing, increased substance use as well as Generalized Anxiety Disorder, Social Phobia, Specific Phobia, Panic Disorder, and personality disorders in the form of Obsessive-Compulsive and Antisocial personality disorder (Hasin et al., 2005). These co-occurring conditions further contribute to morbidity and mortality that are associated with depressive disorders.

Though occurrence of depressive disorders is multiply determined, CSA appears to be a robust risk factor for these disorders, as evidenced by a three-to-eight fold increase in prevalence of depressive disorders among those with CSA histories relative to those without CSA histories (Brown et al., 1999; Fergusson, Horwood & Lynskey, 1996). Meta-analytic research supports this increased risk as well, where findings show that those with CSA histories are twice as likely to develop depressive disorders relative to those who experienced physical abuse during their childhood (Lindert et al., 2013). Throughout adulthood, those with CSA histories also evidence more depressive episodes across their lifetime relative to those with histories of physical abuse and healthy controls (Gladstone, Parker, Mitchell, Malhi, Wilhelm & Austin, 2004). Risk stemming from CSA to experience an initial depressive episode is pronounced during the childhood years (Fergusson et al., 1996), and endures at lower levels across adolescence and young

adulthood (Meyerson, Long, Miranda & Marx, 2002; Weiss, Longhurst & Mazure, 1999). Further, reviews of the literature suggest that CSA is linked to an earlier onset, longer course, and poorer response to treatment for depressive disorder (Cook et al., 2017). For example, individuals with a history of CSA are diagnosed with affective disorders on average 10 years earlier than those without such histories (Giese, Marshall, Thomas, Dubovsky & Hilty, 1998). Additionally, after one year of treatment, those with abuse histories are less likely to reach remission from depressive disorders than their peers (Zlotnick, Ryan, Miller & Keitner, 1995).

1.2 Mechanisms for Depression Risk among those with CSA

Though the relationship between CSA and depression is well-documented, the mechanisms by which CSA confers increased risk for depressive disorders are not known. Growing evidence points to a propensity to experience stress keenly (stress sensitization) and deficits in managing distressing emotions (emotion regulation) as two candidates for elucidating the relationship between CSA and depression; however, their exact roles remain unclear. For example, each may function independently to increase the risk for depression, or they may demonstrate a more complex relationship whereby emotion regulation deficits contribute to stress sensitivity that is associated with CSA. In the sections below, I present evidence for stress sensitization and emotion regulation deficits as potential mechanisms by which CSA is related to depression, and propose a model in which emotion regulation deficits mediate the relationship between CSA and stress sensitization.

1.2a CSA, Stress Sensitivity, and Depression.

A substantial body of work illustrates the relationship between stress and risk for depression (Hammen, 2005), and there is increasing evidence that CSA is associated with a more pronounced experience of stress across physiological and subjective levels (Heim & Nemeroff, 2001). There is considerable evidence that trauma during childhood, including that associated with CSA, results in dysregulation of the body's stress response system (Heim and Nemeroff, 1999). This system includes the Hypothalamic-Pituitary-Adrenal (HPA) axis, which is the primary neuroendocrine stress response system that aides individuals in dealing with and adapting to stress (McEwen, 2004). The hypothalamus monitors free-floating stress hormones (e.g., cortisol) and up- or down-regulates the release of these hormones in response to environmental demands via the corticotropin-releasing factor (CRF) (Heim and Nemeroff, 1999). Trauma in the form of CSA during childhood has been linked to reduced hippocampal volume and insensitivity of the HPA axis to cortisol levels, thereby protracting the physiological stress response. Importantly, evidence points to increased CRF release among those with CSA histories, relative to those without, in response to comparable levels of stress (Heim et al., 2002). This increase results in greater generalized stress-sensitization among those with CSA, given that HPA axis dysregulation is progressive-- failure to downregulate cortisol results in damage to cortisol receptors, which reduces the sensitivity of the HPA axis over time (Arborelius et al., 1999). Heightened CRF release in response to stress and HPA axis dysregulation have both been shown to mediate the relationship between CSA and depression (Heim et al., 2002; 2008). Importantly, CRF is not isolated to the HPA axis, and its release has been shown to influence the subjective experience of stress as well as the physiological stress response (Heim & Nemeroff, 1999).

With respect to the subjective experience of stress, adults with CSA histories report higher levels of perceived stress (Hyman, Paliwal & Sinha, 2007; Vranceanu, Hobfoll & Johnson, 2007), and greater affective reactivity to similar stress levels relative to their peers (Glaser, van Os, Portegijs & Myin-Germeys, 2006). For example, Vranceanu and colleagues (2007) observed robust elevations in perceived stress during the prior month and depression symptoms among community-dwelling women with CSA histories relative to their peers, and moreover, perceived stress mediated the relationship between CSA history and depression symptoms (Vranceanu et al., 2007). In further support of CSA's link to stress sensitization, Glaser and colleagues (2006) noted greater emotional reactivity to minor stressors in the daily lives of adults who experienced unwanted sexual contact during childhood (Glaser, van Os, Portegijs & Myin-Germeys, 2006). Such increased reactivity to stress is a known risk factor for depression and may be linked to emotion regulation difficulties (Bylsma & Rottenberg, 2011).

1.2b CSA, Emotion Regulation, and Depression.

Emotion regulation reflects volitional and automatic processes by which individuals monitor and influence the nature, intensity, morphology, and chronicity of emotional experience and expression (Gross, 1998; Thompson & Calkins, 1996). This modulation may reflect up- or down-regulation of a given emotion, irrespective of its valence (Parrott & Schulkin, 1993). With respect to depression, ER generally pertains to unsuccessful efforts of down-regulating sadness and dysphoria (Kovacs, Sherrill, George, Pollock, Tumuluru & Ho, 2006; Gotlib, Joormann, & Kovacs, 2010). Indeed, it is increasingly understood that vulnerability for depression arises not from the experience of distress and sadness, but from responses that prolong the dysphoric emotional

experience (Joorman & Gotlib, 2010; Kovacs & Lopez-Duran, 2010; Kovacs et al., 2009). Such ER efforts reflect the insufficient or ineffective use of responses that reduce distress in the short- and long-term (adaptive ER), and the frequent use of responses that paradoxically prolong or exacerbate distress (maladaptive ER) (Kovacs, Rottenberg, & George, 2009). Meta-analytic findings support the relationship between adaptive and maladaptive ER and depression, as evidenced by robust associations between the use of maladaptive responses (e.g., rumination, avoidance, and emotional suppression) with elevated depression symptoms, and adaptive ER (e.g., problem solving and cognitive reappraisal) with reduced depression levels across clinical and non-clinical adult samples (Aldao, Nolen-Hoeksema, & Schweizer, 2010). Further, use of maladaptive responses has been shown to robustly predict new incidents of depressive episodes (Kovacs, Yaroslavsky, Rottenberg, George, Kiss, Halas et al., 2016), a protracted course of the disorder (Joormann & Stanton, 2016; Nolen-Hoeksema, Wisco, Lyubomirsky, 2008), and shorter periods of remission (Kovacs, 2016).

Emotion regulation repertoires begin developing during infancy, with normative maturation involving increased sophistication and pruning of less effective responses within adaptive repertoires (Kopp, 1989; Kovacs et al., 2017) and a reduction in maladaptive responses. Childhood maltreatment is believed to disrupt the normative development of emotion regulation repertoires (Alink et al., 2009). Indeed, a growing literature supports the relationship between CSA and the use of maladaptive ER responses. For example, adolescents and adults with CSA histories have been shown to ruminate more than their peers (Heleniak, Jenness, Stoep, McCauley & McLaughlin, 2016; Conway, Mendelson, Giannopoulos, Csank & Holm, 2004), with ruminative

responses mediating the relationships between CSA and internalizing and depression symptoms, respectively. In a similar vein, CSA has been linked to the use of avoidant coping in a sample of adults seeking treatment for cocaine use (Hyman, Paliwal & Sinha, 2007), as well as to emotional suppression among depressed persons (Liverant, Brown, Barlow & Roemer, 2008). Others have also noted a relationship between avoidant coping and CSA among depressed and symptomatic women (O'Mahen, Karl, Moberly & Fedock, 2015), as well as emotional suppression and experiential avoidance among those with such histories (Ehring & Quack, 2010)

Relatively less is known about the relationship between CSA and adaptive emotion regulation responses, and results from studies that examine this association are mixed. For example, Carvalho and colleagues found an inverse relationship between CSA histories and the use of cognitive reappraisal in a mixed-sex sample of clinical and community participants (Carvalho et al., 2014). Conversely, Esposito and Clum (2005) found no direct association between CSA and problem solving among a mixed-sex sample of incarcerated adolescents. Rather, CSA histories were found to enervate the effectiveness of problem-solving for reducing suicidal ideation (Esposito & Clum, 2005).

However, a number of studies considering constructs related to adaptive emotion regulation provide indirect support for a relationship between CSA and the use of adaptive emotion regulation responses. For instance, Kim and Cicchetti (2010) showed an inverse relationship between CSA and socially appropriate emotional displays, empathic responses, and emotional self-awareness, which in turn lead to increased internalizing symptoms over time. Others have also linked CSA with poor emotional awareness, non-acceptance of emotional experiences, as well as difficulty engaging in

goal-directed behavior and controlling impulsive behaviors during times of upset (Schierholz, Küger, Barenbrügge, and Ehring, 2016; Klanecky, Woolman & Becker, 2015), as well as a reduction in perceived efficacy in managing distress (Chang, Kaczurkin, McLean & Foa, 2017; Klanecky, Woolman & Becker, 2015; Schierholz, Küger, Barenbrügge, and Ehring, 2016). As emotional awareness is integral in facilitating the selection and use of emotion regulation strategies (Barrett & Gross, 2001) these findings suggest that CSA may undermine requisite processes to adroitly use adaptive emotion regulation strategies.

1.2c Emotion Regulation and Stress Sensitization.

A growing literature suggests that ER is related to the experience of stress across physiological and subjective levels, as stress is often associated with unpleasant emotional experiences in the form of anxiety, anger, sadness, envy, guilt and shame (Wang & Saudino, 2011). In particular, evidence shows that the habitual use of maladaptive ER responses is associated with elevated stress levels (Martin & Dahlen, 2005; Richardson, 2017). For example, college students who reported engaging in self-blame and rumination during times of distress also reported higher levels of general stress (Martin & Dahlen, 2005). This finding is consistent with those garnered from experience sampling studies. College students who reported frequently engaging in emotional suppression evidenced lower levels of positive affect when confronted with stressors in everyday life as compared to those who used emotional suppression infrequently (Richardson, 2017). Emotion regulation deficits have also been studied as a moderator for the relationship between stress and various negative outcomes (Humbel, Messerli-Bürgy, Schuck, Wyssen, Garcia-Burgos, Biedert, et al., 2018; Extremera & Rey, 2015). For

example, one study found decreased goal-directed behavior, awareness, and clarity and increase impulsivity and non-acceptance of emotion in response to a stress induction, which results in lower levels of positive affect in a sample of clinical and healthy control women (Humbel et al., 2018). Similarly, Extremera & Rey (2015) demonstrated that when Spanish speaking participants were asked to read an emotionally-charged story and identify how to best respond emotionally for one's own benefit and the benefit of an imagined other, those who evidenced higher levels of depression and lower levels of happiness demonstrated higher perceived stress and lower ability to identify effective ER (Extremera & Ray, 2015). This may suggest that ER deficits have a negative influence on well-being in the context of stress.

In addition to subjective stress, physiological stress responses have also been linked to maladaptive ER. In particular, studies have shown that suppression is related to cortisol reactivity in response to stressors (Lam, Dickerson, Zoccola, Zaldivar, 2009; Richardson, Rice & Devine, 2014). For instance, a study of undergraduate students who completed a stress induction, during which they prepared and gave a speech to a panel on why they would be a good candidate for a job, found a significant interaction between trait level suppression and cortisol reactivity (Lam et al., 2009). More specifically, individuals who scored higher on trait suppression evidenced higher peak cortisol levels after they completed the stressful task (Lam et al., 2009). Surprisingly, a study using the same stress inducing task found that individuals who evidenced higher levels of suppression and perfectionism had lower levels of cortisol in response to the task (Richardson, Rice & Devine, 2014), which may suggest that particular populations

experience chronic stress differently, where some individuals may exhibit blunted effects of stress, a finding in line with the stress sensitization theory.

Conversely, using adaptive ER strategies has been shown to reduce both the physiological and subjective experience of stress, but findings are mixed. For instance, Lam and colleagues (2009) also showed that the same undergraduate students, who implemented more reappraisal, rather than suppression, evidenced greater cortisol reactivity in response to the stress induction, suggesting that reappraisal does not ameliorate the effects of stress (Lam et al., 2009). However, a different study of community-dwelling adults who were participating in their first tandem skydive showed that individuals who scored higher on trait reappraisal evidenced lower cortisol reactivity than those who did not implement reappraisal (Carlson, Dikeciligil, Greenberg & Mujica-Parodi, 2012). Others have also found that distraction, another adaptive ER strategy, was inversely correlated with cortisol release in response to valenced faces in a mixed-sex adult sample (Kinner, Het & Wolf, 2014), which suggests that adaptive ER responses may attenuate the physiological stress response. Clarifying the role of adaptive ER on physiological stress reactivity specifically would do much to illuminate physiology's role in stress sensitivity.

Adaptive ER has also been shown to influence the subjective experience of stress. In a community sample of women who had experienced a stressful life event within the past 3 months participants were asked to use cognitive reappraisal in response to a sadness induction. Those who reported high levels of stress, as well as higher cognitive reappraisal ability, evidenced lower levels of depressive symptoms than those without these adaptive abilities (Troy, Wilhelm, Shallcross & Mauss, 2010). Additionally, a

review of the literature concluded that both attentional control and cognitive reappraisal serve as protective factors for individuals experiencing stress, whereby those who implement these adaptive strategies exhibit lower levels of depression and negative affect, even if they are experiencing high levels of stress (Troy & Mauss, 2011).

1.3 Limitations of Current Literature

Though there is compelling evidence for the mediating roles of stress sensitivity and emotion regulation deficits between CSA and depression, the current body of work on these associations has several limitations. First, despite strong evidence for a relationship between ER deficits and stress sensitivity, most studies that examine the relationship between CSA and depression risk focus on the role of either stress sensitivity or ER in depression risk. The exclusion of examining both putative mechanisms provides a limited picture on the means by which CSA perpetuates its adverse effects. Second, with few exceptions (e.g., Glaser et al., 2006; Lardinois et al., 2010) most work on CSA and depression risk has relied on cross-sectional methods which are known to be influenced by reporting biases and limited self-knowledge (Simonich, Wonderlich, Crosby, Smyth et al., 2004). Those studies that employ ecological momentary assessment (EMA) methods that examine affective experiences in the daily lives of those with CSA histories show that these individuals are more likely to engage in self-destructive behaviors, such as purging and self-harm (Wonderlich, Rosenfeldt, Crosby, Mitchell, Engel, Smyth & Mittenberger, 2007), have higher levels of perceived stress and dysfunctional attitudes (Walsh, Basu & Monk, 2015), and increased persistence of negative affect (Teicher, Ohashi, Owen, Polcari & Fitzmaurice, 2015). Given the inherent

limitation of literature as it stands, further investigation is necessary to understand the role of ER deficits in the relationship between CSA and stress sensitivity.

CHAPTER II

The Current Study

The present study tested competing models of CSA, stress sensitivity (moderation of the effects of stress on depression outcomes by CSA histories), ER deficits and depression in a sample of adults with and without histories of sexual abuse prior to the age of 18 years. Specifically, I tested whether CSA predicts depression-related outcomes independently via stress sensitivity and ER deficits (see Appendix, Figure 1), or whether ER deficits intervene in the relationship between CSA and stress-sensitivity (see Appendix, Figure 2). Based on the extant literature, I hypothesized that CSA histories would predict a reduced dispositional use of adaptive ER responses and increased use of maladaptive responses, as well as predict an increased sensitivity to stress, reflected by higher depression levels for those with CSA histories compared to those without them at comparable levels of perceived stress. Given limitations in cross-sectional methods, I also tested these models via EMA. In these models, stress is indicated by its momentary assessment at the time of the prompt, and elevated levels of negative affect and low levels of positive affect reflect depression-related outcomes. I tested whether sensitivity to daily

life stressors, defined as the moderation of momentary stress by CSA histories, and the dispositional use of adaptive and maladaptive ER responses independently mediate the effects of CSA on negative and positive affect levels in daily life, or whether the relationship between CSA and stress sensitivity may be explained, in part by ER deficits.

2.1 Hypotheses

Hypothesis 1a. CSA will predict depression via its moderation of perceived stress on depression symptoms (stress sensitivity) and ER deficits marked by the elevated use of maladaptive and limited use of adaptive ER responses (see Appendix, Figure 1).

Hypothesis 1b. Emotion regulation deficits in the form of the frequent use of maladaptive responses and the infrequent use of adaptive responses will mediate the effect of CSA on stress sensitivity (see Appendix, Figure 2).

Hypothesis 2a. A history of CSA will predict greater stress sensitivity in daily life, whereby CSA will moderate the effect of momentary stress on negative and positive affect level, and CSA effects on affective outcomes will be mediated via ER deficits described in hypothesis 1a. (see Appendix, Figure 3).

Hypothesis 2b. ER deficits will mediate the effect of CSA on stress sensitivity in daily life (see Appendix, Figure 4).

CHAPTER III

METHOD

3.1 Participants

Participants were 142 community dwelling adults and undergraduate students (71% female, $M= 26.63$ years, $SD= 10.81$) who were recruited by posting flyers in the community, Research Match, Craigslist and the Undergraduate Psychology Research Pool at Cleveland State University. Of these participants, 97 were included in analyses due to missing data on variables used within study models. Of these, 32 reported histories of sexual abuse prior to the age of 18. Participants' racial composition was made up of Caucasian (58%), African American (23%), Middle Eastern (5%), multi-racial (4%) and "other" backgrounds (2%). Community dwelling participants were paid to compensate them for their time, while undergraduate students received course credit for their participation. Eligible participants included those who completed a pre-screen survey online that assessed their adherence to directions on study measures, psychological symptomology and expressed an interest in completing the second phase of the study. Further, eligible participants provided their contact information.

3.2 Measures

3.2.1 General Measures.

Center for Epidemiologic Studies-Depression Scale (CES-D). The CES-D is a 20-item self-report depression inventory for the general population that measures current depressive symptomology with possible scores ranging from 0 to 60. Respondents are asked to report how strongly they have felt a certain way in the past week. Response options are as follows: 1=“Rarely or None of the time (less than 2 days)”, 2= “Some or a Little of the Time (1-2 days)”, 3= “Occasionally or a Moderate Amount of Time (3-4 days)”, or 4= “Most or All of the Time (5-7 days)”. Items 4, 8, 12 and 16 are phrased positively, and therefore are reverse coded (Radloff, 1977). A total score was computed and used for analyses. Reliability within this study was acceptable ($\alpha=.90$).

Perceived Stress Scale-10 (PSS). The PSS is a 10-item self-report inventory for the general population that measures the degree to which situations in one’s life are considered stressful within the past month. This scale also includes several items about current levels of experienced stress of the individual. Respondents rate how often they have felt or thought certain things within the past month (0=never; 1=almost never; 2=sometimes; 3=fairly often; 4=very often). There are four positive questions (items 4,5,7 & 8) which are reverse coded and used to calculate a total score (Cohen, 1988). This scale evidenced acceptable reliability in this study ($\alpha=.93$).

Feelings and Me-Adult version (FAM). The Feelings and Me scale is a 54-item measure of trait level adaptive and maladaptive ER responses. Adaptive responses were operationally defined as strategies that attenuate negative affect in appropriate ways and keep negative affect from getting worse. On the other hand, maladaptive responses are defined as responses likely to exacerbate negative affect in the short and long term. Items

on this scale start with: “When I feel sad or down, I...”, which is followed by statements that are rated on a scale from 0= “not true of me”, to 2= “many times true of me”.

Adaptive ER is scored by summing 30 items with a potential score ranging from 0-60 and maladaptive ER is scored by summing 24 items ranging from 0-60 (Kovacs, Rottenberg & George, 2009). Both adaptive and maladaptive indices showed strong psychometric properties in this study (adaptive $\alpha=.88$; maladaptive $\alpha=.89$).

3.2.2 Ecological Momentary Assessment Measures.

Immediate Affect. Positive and Negative affect were measured via items drawn from the Positive and Negative Affect Schedule (strong, excited, happy, sad, upset, angry, and frustrated (Watson et al., 1988). Participants were asked to rate these feelings on a 5-point Likert scale referring to how they felt at the time of the EMA prompt.

Immediate Stress. Stress was measured via a single item, that reflects the degree to which respondents experienced stress on a 5-point Likert scale (1-“not at all” to 5-“extremely”) at the time of the EMA prompt.

3.3 Procedure

Data used from this study was drawn from a larger project that examined associations between internalizing disorders, ER, and psychophysiological processes. This larger project was carried out in two phases: Phase 1 involved an online data gathering on personality dimensions, ER, and contextual factors, and Phase 2 involved data collection in the laboratory and in participants’ daily lives via EMA. Only Phase 2 data was used in this study.

When participants came into lab, they were consented and privately completed self-report surveys to assess their levels of current depressive symptoms (CES-D),

perceived stress levels (PSS-10), and dispositional adaptive and maladaptive ER implementation (FAM). Next, participants completed a psychosocial interview during which demographic and abuse histories were ascertained, as well as semi-structured clinical interviews to assess histories of psychiatric disorders, which were not used in the present study. Mandated reporting procedures were followed in instances where sexual abuse during the childhood years was reported.

3.3.1 Ecological Momentary Assessment.

EMA involves recurrent sampling of subjects' current behaviors and experiences in real time, in participants' natural setting rather than in the lab (Shiffman, Stone & Hufford, 2008). Participants received 5 prompts a day for 7 days on their cell phone to follow a link to a survey generally between 9:00 a.m. and 9:00 p.m. on SurveySignal (Hofmann & Patel, 2015). This schedule was modified occasionally due to times when the participant was not able to use his/her phone. Participants were sent a reminder prompt 15 minutes after receiving the original text message within a scheduled sampling period, and were allowed 30 minutes to answer the survey before the link expired. Participants provided contextual information about where they were, who they were with, and reported affect ratings on a 5-point likert-type scale ("very slightly/not at all", "a little", "moderately", "quite a bit" & "extremely"). Items used to create aggregate negative and positive affect were upset and sad and happy and excited, respectively. Participants also rated their current levels of stress, measured discretely by a single item, on the same 5-point scale. These reports of affectivity reflect the emotions they were feeling and the strength of these emotions at the time they received the prompt.

3.4 Statistical Analyses

Descriptive statistics and bivariate associations among study constructs were tested using SPSS v. 22. SPSS was also used to examine the assumptions necessary to conduct statistical analyses to test study hypotheses, as well as to test hypotheses 1a and b. Mediation and moderation analyses associated with hypothesis 1a and b were fit using PROCESS Macro, model 15 (Hayes, 2013) for SPSS 21. Bias-corrected 95% confidence intervals and bootstrap estimates were calculated with 5,000 samples. Hierarchical Linear Model (HLM) v. 7 (Raudenbush, Bryk, Cheong, & Congdon Jr., n.d) software was used to test hypotheses 2a and 2b that involve nested data. Participants' age and sex were covaried in all models as sexual abuse histories and depression are more common in women relative to men (Finkelhor et al., 2014; Pereda, Guilera, Forns & Gómez-Benito, 2009; Stoltenborgh et al., 2011). For hypotheses 1a. and 1b., assumptions of homoscedasticity, normality, linearity and multicollinearity were met. Assumptions of homoscedasticity for EMA data were violated and thus, heteroscedasticity-robust standard errors were used to account for this violation.

A series of regression models were employed using cross-sectional data that test the effects of 1) CSA histories' moderation effects on the relationship between perceived stress and depression symptoms (stress sensitivity), 2) the indirect effects of CSA history on depression symptoms via adaptive and maladaptive ER repertoires, and 3) sequential indirect effects of CSA on depression symptoms via maladaptive and adaptive ER repertoires and stress sensitivity. Across cross-sectional models, stress sensitivity reflects moderation effects of CSA on the relationship between perceived stress and depression symptoms which has been commonly used to model diathesis-stress relationships (e.g.,

Monroe & Harkness, 2005; Morris, Ciesla & Garber, 2010). In order to test the mediation effects of ER repertoires between CSA and stress sensitivity, a moderated-mediation model was employed. Following best practices (MacKinnon, Fairchild & Fritz, 2007), mediation analyses used bootstrapped methods to derive confidence intervals around the indirect effect (Preacher & Selig, 2012) and were conducted in a single step.

Models employed using EMA data were very similar to the models described for cross-sectional data with several important exceptions. First, all EMA models examined negative and positive affect as outcomes (level 1). Multi-level modeling was employed due to the nested nature of this data as reflected by EMA responses (level 1) within participants (level 2). Further, we examined effects of stress by its time-invariant (participant's average stress level over the measurement period, *PmStress*) and time-varying components (deviation from participant's average stress levels at a given EMA prompt, *WPStress*) (Algina & Swaminathan, 2011; Enders & Tofighi, 2007). Random effects were employed for all level 1 variables, as level 1 outcomes, predictors and covariates may differ across observations. Additionally, all continuous level-2 predictors were grand mean centered (i.e., age, ER indices & person mean stress) (Enders & Tofighi, 2007) while dichotomous variables, such as CSA history, were uncentered (Singer & Willett, 2003). Stress sensitivity within EMA models are reflected by the interaction between CSA history and participant's average stress level (*PMStress*) and the cross-level interaction between CSA and within-person deviations from average levels of stress (*WPStress*) to predict increased levels of NA and PA.

CHAPTER IV

RESULTS

4.1 Descriptive analyses

Pearson correlations were performed to examine correlations between all variables in the model (see table 1). Age was significantly correlated with CSA and maladaptive ER, while sex was significantly correlated with CSA and depression symptoms, therefore, both were entered into all models as covariates. Additionally, CSA was positively correlated with increased depression symptoms, heightened levels of perceived stress, participant's average daily life stress and negative affect, and increased implementation of maladaptive ER. Conversely, CSA was negatively correlated with participant's average positive affect in daily life.

4.2 Hypothesis Testing

4.2.1 Hypothesis 1a. Does CSA predict depression via stress sensitivity and ER deficits?

To test this hypothesis, depression symptoms were regressed on CSA histories and demographic covariates (block 1), perceived stress (block 2), and the two-way interaction between CSA and perceived stress that reflects stress sensitivity (block 3).

Independent of demographic characteristics, CSA and perceived stress each significantly predicted depression symptoms (see table 2). However, the association between perceived stress and depression symptoms did not vary as a function of abuse histories ($p=.81$).

In the second model, we examined whether CSA predicts ER deficits, in the form of an abundance of maladaptive ER responses and lack of adaptive responses, and their indirect effects on the association between CSA and depression symptoms. In order to test this, adaptive and maladaptive ER repertoires were regressed on to CSA histories and demographic characteristics (path a) to test whether ER repertoires could serve a mediating role. Then, depression symptoms were regressed on adaptive and maladaptive ER repertoires (path b) along with CSA histories and demographic covariates (path c).

CSA histories predicted ER deficits in the expected ways, whereby those with such histories evidenced increased deployment of maladaptive ER and a lack of adaptive responses. Tests of indirect effects revealed that both adaptive (indirect effect=1.63, 95% CI= .074-3.68) and maladaptive ER partially mediated the relationship between CSA and depression symptoms (indirect effect= 8.03, 95% CI= 4.54-11.98), whereby CSA histories predicted increased deployment of maladaptive ER responses and a lack of adaptive responses which in turn, predicted increased depression symptoms.

4.2.2. Hypothesis 1b. Do ER deficits mediate the effect of CSA on stress sensitivity?

Consistent with expectations, CSA predicted more abundant maladaptive ER repertoires and lack of adaptive ER repertoires, and therefore both ER indices were examined as a potential moderators of the association between perceived stress and

depression. This effect reflects stress sensitivity for those who deploy maladaptive ER responses more frequently. To test this association a path model was fit in which depression symptoms were regressed onto demographic characteristics, ER repertoires, perceived stress and the interaction between both ER indices and stress (stress sensitivity).

ER deficits did not moderate the effect of perceived stress on depression ($p_s = .25; .51$) and therefore such deficits did not contribute to stress sensitivity. Since ER deficits did not moderate these effects, these deficits were not examined as mediators for the effects of CSA on stress sensitivity.

4.2.3. Hypothesis 2a. Does CSA history predict greater stress sensitivity in daily life?

To test this hypothesis, we employed a series of multi-level regression models examining CSA history's moderation effect of time-invariant (*PMStress*) and time-varying (*WPStress*) aspects of stress on depressive affects (increased NA and decreased PA). First, to test stress sensitivity in daily life, levels of negative and positive affect were regressed onto demographic characteristics, abuse history, time-invariant (*PMStress*) and time-varying (*WPStress*) aspects of stress and the two-way interaction between CSA and both stress indices (stress sensitivity).

In partial support of hypotheses, CSA histories significantly predicted increased depressive affects in daily life, whereby those with such histories evidenced significantly increased levels of NA ($\gamma = .98, p < .01$), however CSA had no effect on PA. Both participants' average stress levels and momentary increases of stress predicted increased NA. Additionally, deviations from average stress levels also predicted decreased levels of

PA. These effects were qualified by the significant cross-level interaction between CSA and deviations from average stress to predict NA ($\gamma_{CSA \times WP_{stress}}=.44, p<.01$). Simple slopes analyses that probed this interaction revealed increased NA as a function of momentary increases in stress for those with CSA histories, $\gamma_{CSA}=.74, t(95)=8.79, p<.001$, $\gamma_{NoCSA}=.35, t(95)=3.90, p<.001$ (see Figure 5). Contrary to hypotheses, the relationship between CSA and PA did not vary as a function of average levels or deviations from average levels of stress.

In the second model, we examined whether ER deficits, as defined previously, mediate the effects of CSA on depressive affects. We did this by regressing ER responses onto CSA history (path a) and depressive affect on ER responses (path b) and CSA history (path c).

Independent of demographic characteristics, increased deployment of maladaptive responses predicted increased negative affect in daily life. Further, maladaptive ER repertoires mediated the effects of CSA on NA in daily life (indirect effect=.57, 95% CI=.16-1.09) and therefore may partially explain increased risk for depressive affects for those with CSA histories. On the other hand, adaptive ER predicted increased hedonic affect, but PA was not predicted by maladaptive repertoires. Further, adaptive ER did not mediate the relationship between CSA and negative affect, but did evidence significant indirect effects for PA (indirect effect=-.39, 95% CI= -.73--.03). More specifically, CSA predicts decreased implementation of adaptive ER, which in turn predicts lower levels of PA in daily life. These findings highlight the adverse effects of ER deficits within this population.

4.2.4. Hypothesis 2b. Do ER deficits mediate the effect of CSA on stress sensitivity in daily life?

To test this hypothesis, interaction terms between each ER index and average levels and fluctuations in stress were added to the previously defined model. This reflects a cross-level interaction whereby the slope of stress deviations was regressed onto both ER indices. As hypothesized, maladaptive ER significantly interacted with daily life stress to predict increased NA ($\gamma_{MMR \times PM_{stress}} = .04, p < .01, \gamma_{MMR \times WP_{stress}} = .02, p < .01$). Post-hoc analyses of these interactions evidenced that tendencies to deploy maladaptive ER responses exacerbate the effect of stress on NA across average levels $\gamma_{malER, +1SD} = .99, t(95) = 4.98, p < .001$ $\gamma_{malER, -1SD} = .24, t(95) = 1.32, p = .19$ (see Figure 6), and momentary increases of stress $\gamma_{malER, +1SD} = .65, t(95) = 5.05, p < .001, \gamma_{malER, -1SD} = .26, t(95) = 4.07, p < .001$ (see Figure 7). Conversely, adaptive ER significantly moderated the relationship between deviations from average stress levels and PA ($\gamma_{AMR \times WP_{stress}} = -.011, p < .05$), whereby those who highly deviate from their average stress level and deploy less adaptive ER responses exhibit lower levels of PA, $\gamma_{adER, +1SD} = -.07, t(95) = -7.16, p < .001, \gamma_{adER, -1SD} = -.48, t(95) = -5.74, p < .001$ (see Figure 8).

As hypothesized, ER deficits mediated the effects of CSA on stress sensitivity in daily life. This effect was evidenced by maladaptive ER for both average levels $\gamma_{malER, +1SD} = .38, 95\% CI = .15-.69, \gamma_{malER, -1SD} = .70, 95\% CI = .26-1.28$ and momentary increases of stress $\gamma_{malER, +1SD} = .48, 95\% CI = .11-.93, \gamma_{malER, -1SD} = .19, 95\% CI = .04-.40$, to predict NA. More specifically, those with CSA histories deploy maladaptive ER more readily which in turn, leads to greater sensitivity to stress. Adaptive ER also evidenced significant indirect effects on the relationship between CSA and stress sensitivity. This

effect was evidenced by adaptive ER and momentary deviations from average stress levels ($\gamma_{adER+1SD}=3.38$, 95% CI=.23-6.90, $\gamma_{adaER-1SD}=2.28$, 95% CI=.18-4.69) to predict PA, whereby those with CSA histories evidence decreased deployment of adaptive ER responses, which in turn decreases stress sensitivity effects by predicting increased PA.

CHAPTER V

DISCUSSION

The current study aimed to test competing mechanistic models by which CSA confers depression risk via mixed-methodology. Depression is a commonly studied outcome associated with CSA, however the mechanisms for this relationship are largely unknown (Brown et al., 1999; Fergusson et al., 1996; Lindert et al., 2013). The literature suggests that those with CSA histories evidence sensitivity to stress at comparative levels as their abuse-free peers, which is evident through physiological stress responses as well as subjective stress (Heim et al., 2000; Luthar & Zigler, 1991). Additionally, these individuals evidence difficulties in regulating distressing emotions, in the form of insufficient or ineffective use of responses that reduce distress in the short- and long-term (adaptive ER) and the frequent implementation of responses that prolong distress, i.e., maladaptive ER (Chang et al., 2017; Hebert et al., 2018; Ullman et al., 2014). While the current body of work supports both stress sensitivity and ER deficits as potential mechanisms for depression risk within this population (Conway et al., 2004; Glaser et al., 2006; Heleniak et al., 2016; Heim et al., 2002; Vranceanu et al., 2007), the nature of these associations is largely underexamined. Additionally, these investigations are largely

cross-sectional and experimental, where the limited experimental design may not reflect an accurate illustration of these relationships (Simonich et al., 2004). Two sets of hypotheses were investigated to test whether CSA predicts depression-related outcomes indirectly via stress sensitivity and ER deficits or whether ER difficulties play an intervening role in the relationship between CSA and stress sensitivity. These associations were tested across self-report and daily life settings to more thoroughly investigate the interplay of these mechanisms.

It was hypothesized that those with CSA histories would evidence elevated depression symptoms and depressive affects via stress sensitivity across self-report and daily life measures. Stress sensitivity was modeled by interaction terms between CSA and perceived as well as daily life stress indices. Main effects of CSA and stress were significant in predicting depressive outcomes across methodologies, but contrary to expectation, findings on stress sensitivity were mixed. Those with CSA histories evidenced sensitivity to momentary increases of stress in daily life, however this was not evidenced for average or perceived stress levels. More specifically, those with CSA histories whose momentary stress increased relative to their average levels reported elevated NA as compared to their abuse free peers. This may suggest that increases from average stress levels may be more detrimental for those with CSA histories which is supported by other empirical findings (Glaser et al., 2006) and emphasizes daily life stress's adverse effects for those in this population. Further, these findings imply that ongoing stress may evidence distinct effects from those that produce momentary stress responses for this population. This possibility is consistent with empirical findings that discern basal stress levels and their reactivity across physiological and subjective

measures (de Kloet, Vreugdenhil, Oitz, & Joëls, 1998; Henckens et al., 2016; Silwinski, Almedia, Smyth, Stawski, 2009). For example, effects of daily fluctuations in perceived stress levels have differentially predicted levels of dysphoria in daily life than enduring indices of such stress (Sliwinski et al., 2009).

Overall, elevations in stress levels predicted parallel increases in depression symptoms and affects, however there were exceptions to this. Such findings mirror results from several empirical studies that connect CSA to elevated stress levels (Glaser et al., 2006; Heim et al., 2008; Luthar & Zigler, 1991) and stress to depression (Hammen, 2005; Kessler, 1997; Monroe & Harkness, 2005), and emphasizes the impact of stress on those with CSA histories.

The next set of hypotheses was that ER repertoires would serve as another mechanism for the association between CSA and depression outcomes across self-report and daily life indices. Findings suggest that depression risk for those with CSA histories may be conferred, in part, due to ER deficits in the form of an abundance of maladaptive ER responses and lack of adaptive ER repertoires. Consistent with prior research (Carvalho et al., 2014; Ehring & Quack, 2010; Esposito & Clum, 2002; Heleniak et al., 2016; Hyman et al., 2007; Johnson & Lynch, 2013; Ullman et al., 2014), CSA was linked with increased deployment of maladaptive ER responses and decreased implementation of adaptive ER. In turn, adaptive and maladaptive ER responses predicted increased depression symptoms which mirrors previous findings (Aldao et al 2010; Kovacs et al., 2009; 2016). Findings on ER's indirect effects on CSA predicting depressive affect were mixed, as maladaptive ER mediated the association between CSA and NA, but adaptive ER did not. Prior research on CSA and adaptive ER evidences mixed results (Esposito &

Clum, 2005; Kim & Cicchetti, 2010) which emphasizes the importance of further examinations of this relationship.

As defined in the present study, and by others, depressive affects may be evidenced by high negative affect and low positive affect levels (Clark & Watson, 1991). Findings suggest that those with sexual abuse histories evidence greater levels of NA in daily life, however this experience did not predict decreased PA. This finding was not consistent with current work as empirical findings support decreased PA for those with CSA histories (Etter, Guathier, McDade-Montez, Cloitre & Carlson, 2013), however dysphoria and sadness are more commonly observed than anhedonia for those with depression (Baji et al., 2009; Lewinsohn, Rohde, & Seeley, 1998; Smith, Joiner, Pettit, Lewinsohn, & Schmidt, 2008) and therefore may have influenced findings within this study. Both ER indices evidenced associations with depressive affects that mirror previous findings (Brans, Koval, Verduyn, Lim, & Kuppens, 2013; Brockman, Ciarrochi, Parker, & Kashdan, 2017; Chaudhury et al., 2017; Nezlek & Kuppens, 2008); however, only maladaptive ER mediated the association between CSA and negative affect in this study.

Findings highlight the adverse effects of maladaptive ER for those with CSA histories. More specifically, engaging in maladaptive ER responses may help explain why individuals with CSA histories evidence greater depression risk. This finding is consistent with current findings that highlight the robust association between CSA and maladaptive ER deployment which in turn predicts greater depression and internalizing symptoms (Conway et al., 2004; Heleniak et al., 2016). The role of adaptive ER for depression risk in this population is less distinct. While a lack of adaptive ER responses

mediated the relationship between CSA and depression symptoms, it does not seem to clearly explain the relationship between CSA and depressive affects which has been the case in previous studies (Kovacs & Yaroslavsky 2014; Kovacs et al., 2009; 2016; Yaroslavsky et al., 2013). While adaptive ER does not mediate the effect of CSA on negative affect, it does evidence significant indirect effects for PA, whereby those with CSA histories deploy fewer adaptive responses, which in turn predicts decreased levels of PA. These associations emphasize the importance of continued examinations of the role of adaptive ER for depression risk among those with CSA.

The final set of hypotheses, that ER deficits play an intervening role in the relationship between CSA and stress sensitivity, was partially supported. While the relationship between perceived stress and depression symptoms did not vary as a function of ER, maladaptive ER interacted with both average levels and momentary increases of stress to predict increased NA. More specifically, those who experience high average levels of stress and deploy maladaptive responses more readily evidenced significantly increased NA than their peers. Further, similar effects were evidenced by those exhibiting momentary increases in stress and maladaptive ER to predict NA. Why might maladaptive ER evidence stress sensitivity for participant's average levels of daily life stress but not perceived stress? It may be feasible that the time span that stress was measured in influenced the effects of stress, as previous findings suggest that shorter time course of stress evidenced differential effects from trait measures of stress on cortisol levels (Van Eck et al., 1996). It may be feasible that long-term stress in this study evidenced similar effects. In support of the present findings, maladaptive ER has been found to interact with stress to predict greater depression symptoms in daily life

(Connolly & Alloy, 2017). Further, those who habitually deploy maladaptive ER responses tend to evidence a stronger affective response to stressors in daily life as evidenced by increased NA (Krkovic, Clamor & Lincoln, 2018).

On the other hand, adaptive ER responses moderated the effect of deviations from average stress levels on positive affectivity. Adaptive ER responses seem to attenuate the adverse effects of momentary increases in stress by increasing positive affect in daily life. Findings of the present study differ from previous findings that found no significant interaction effects for adaptive ER and stress to predict affect (Krkovic, Clamor & Lincoln, 2018), which may emphasize the importance of continued research in order to define the role of adaptive ER in stress sensitivity. The associations between ER deficits and stress sensitivity may suggest that the relationship between CSA and stress sensitivity may be more complex.

Due to significant stress sensitivity effects of both ER repertoires, indirect effects of ER responses were tested for the association between CSA and stress sensitivity. Findings from the present study suggest that habitual use of maladaptive ER responses may partially explain the association between CSA and stress sensitivity, whereby those with CSA histories may experience sensitivity to stress in daily life due to deploying maladaptive ER when distressed. Consistent with previous work, maladaptive ER repertoires evidence pernicious effects on the association between stress and depression, which highlights their salient role in depression risk (Alado et al., 2010; Extremera & Rey, 2015; Humbel et al., 2018; Kovacs et al., 2009; Richardson, 2017).

Findings also suggest that adaptive ER evidences indirect effects on the relationship between CSA and stress sensitivity to predict PA. Those with CSA histories

tend to deploy fewer adaptive ER responses, which in turn predicts less sensitivity to stress in regards to PA. More specifically, individuals within this population evidence slighter decreases in PA when faced with momentary increases in stress than those who implement more adaptive ER responses. Perhaps this can be explained by emotion regulation flexibility. The literature suggests that regulatory flexibility, rather than use of specific adaptive and maladaptive ER strategies, may best predict psychological functioning (Bonanno & Burton, 2013). For example, reappraisal has been labeled as an overtly adaptive ER response, however in the face of high-intensity emotions it may not evidence significant gains for individuals implementing it (Sheppes et al., 2011). Recent research suggests that poor fit between intensity of stress demands and ER may explain these associations (Myruski, Denefrio & Dennis-Tiwary, 2018) and may help explain the findings on adaptive ER in this study as ER repertoires were dispositional rather than situational. Present findings add to the current body of work as the interplay of these mechanisms has been largely unexamined.

5.1 Limitations

Results of the present study should be interpreted while considering several limitations. First, CSA experiences may vary in severity, frequency, source and developmental time period, all of which have been examined as variables that may influence the negative effects of experiencing CSA (Celmmmons, Walsh, DiLillo & Messman-Moore, 2007; Ruggiero, McLeer & Dixon, 2000). More specifically, these variables may influence stress sensitivity, the development of ER repertoires and depression related outcomes. While detailed accounts of CSA experiences were provided during the psychosocial interview, many participants were guarded in their disclosure

which did not allow the effects of these details to be examined in the present study. Second, individuals with CSA histories often experience other forms of abuse (Briere & Elliott, 2003; Finkelhor, Turner, Shattuck & Hamby, 2015) and these other experiences may have influenced the findings in this study. Third, as evidenced in previous research, stress responses (Kelly, Tyrka, Anderson, Price & Carpenter, 2008; Kudielka & Kirschbaum, 2005) and long-term consequences of CSA (Gray & Rarick, 2018) may evidence gender differences which were unable to be examined in this study due to a small sample of individuals with CSA histories. While we did examine sex as a covariate, these associations may vary as a function of sex and should be considered. Next, the present study examined effects of dispositional ER repertoires in all models, however ER outcomes may be contextual and state ER responses measured during EMA may have provided a clearer picture of the effect of ER among these associations. Finally, this study's measure of stress in daily life was obtained via a single-item measure which may have contributed to greater measurement error than a longer survey may have produced.

5.2 Future Directions

Future research on these associations should control for various contextual variables that have been shown to influence outcomes related to experiencing sexual abuse during childhood such as severity, frequency, age of abuse, and source of abuse. Next, future research should examine the effects of other forms of abuse such as physical and emotional abuse as well as neglect as they may evidence independent effects on outcomes measured in this study. Further, a sample containing more individuals who have experienced CSA may allow for more variability to be explained and provide a clearer picture of these associations. Third, while dispositional ER repertoires have

evidenced robust effects for the relationship between CSA and stress sensitivity, participants may be implementing different forms of ER in daily life as ER outcomes can be contextual (Aldao, Sheppes, & Gross, 2015). Lastly, subjective stress evidenced robust effects for individuals with CSA histories in the present study, but examining physiological stress responses may help verify stress sensitivity within this population and provide a more complete picture of how CSA leads to stress sensitivity as well as the role of ER repertoires.

5.3 Strengths and Clinical Implications

The present study exhibits a number of strengths. First, mixed-methodology procedures allowed us to test stress sensitivity across self-report and daily life reports. Previous research on these associations have relied heavily on cross-sectional designs, but the mixed-methodology in this study allowed for effects of experiencing stressors in daily life to be examined within this population and therefore increased the generalizability of these findings. Further, examinations of stable and time-varying daily stress allowed for a more detailed account of the relationships between CSA, stress sensitivity, ER and depression related outcomes. More specifically, momentary increases in stress appear to be more detrimental for those with CSA histories than stable stress levels. Lastly, there is a dearth of research examining the role of adaptive and maladaptive ER repertoires in stress sensitivity for those with CSA histories, and the present study may be one of the first to examine these associations in daily life. These findings are clinically salient as they highlight the exacerbating effects of maladaptive ER responses for those with CSA histories and should be a key target of treatment for this population. Further, due to the complexity of these relationships and the mixed findings

throughout the literature on stress sensitivity and ER responses for those with CSA histories, this study acts as an important precursor for future research on these associations.

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APPENDIX A: Tables

Table 1. *Descriptive statistics and correlations among demographic, CSA, depression, stress, emotion regulation and affect measures.*

Measures	M (SD)	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.
1. Age	26.61(10.73)	---	-.04	.40**	.15	-.01	-.22*	-.09	-.05	-.29**	-.12
2. Sex	.3 (.46)		---	-.18*	.18*	-.05	-.17	-.09	-.08	.28**	-.06
3. CSA	.22 (.416)			---	.54**	.42**	.23*	-.16	.28**	-.28**	.14
4. CES-D	18.30(13.23)				---	.84**	.61**	-.39**	.51**	-.50**	.40**
5. PSS	28.68 (8.89)					---	.62***	-.05	.52**	-.36**	.46**
6. MMR	42.08 (8.89)						---	-.03	.56**	-.21*	.62**
7. AMR	54.29(10.85)							---	-.26**	.50**	-.24*
8. pNA	2.93 (1.22)								---	-.29**	.68**
9. pPA	4.49 (1.68)									---	-.18
10. pStress	2.01 (.81)										---

Note. CSA = History of Childhood Sexual Abuse, CES-D= Center for Epidemiologic Studies-Depression Scale, PSS = Perceived Stress Scale, MMR = Feelings and Me-Maladaptive Emotion Regulation Subscale, AMR= Feelings and Me-Adaptive Emotion Regulation Subscale, pNA = within-participant average of negative affect, pPA = within-participant average positive affect, pStress= within-participant average stress levels.
 ***p ≤ .001, **p ≤ .05

Table 2. Child Sexual Abuse History effect on Self-Reported Depression Symptoms via Stress Sensitivity and Emotion Regulation.

Variables	DV: Depression Symptoms			
	<i>B</i>	<i>SE</i>	<i>B</i>	<i>SE</i>
Age	-.003	.08	.01	.08
Sex	-1.63	1.36	-1.48	1.36
Stress	1.01**	.08	.98***	.08
CSA	9.45**	2.20	6.40*	3.05
CSA*Stress	---	---	.43	.29
	Med.1 Adaptive ER		Med.2 Maladaptive ER	
	<i>B</i>	<i>SE</i>	<i>B</i>	<i>SE</i>
Age	.05	.09	-.29***	.07
Sex	-2.72	2.13	-3.13	1.61
CSA	-5.25*	2.54	7.26***	1.93
	DV: Depression Symptoms			
	<i>B</i>	<i>SE</i>	<i>B</i>	<i>SE</i>
Age	.07	.08	.06	.08
Sex	-1.60	1.29	-1.41	1.30
Stress	.83***	.09	.85***	.09
CSA	6.25*	2.84	6.99*	2.89
CSA*Stress	.29	.28	-.08	.33
aER	-.19**	.06	-.18**	.06
mER	.19*	.09	.21*	.09
aER*Stress	---	---	.00	.01
mER*Stress	---	---	.01	.01

Note. Sex (0 = female, 1 = male), Stress = Perceived Stress Scale, CSA = Child Sexual Abuse (0 = absent, 1 = present), aER = Feeling and Me Scale, Adaptive subscale, mER = Feeling and Me Scale, Maladaptive subscale, Depression Symptoms = Center for Epidemiologic Studies Depression Scale.

*** $p \leq .001$, ** $p \leq .01$, * $p \leq .05$

Table 3. Child Sexual Abuse History effect on Negative and Positive Affects via Stress Sensitivity and Emotion Regulation in daily life.

Variables	DV: NA				DV: PA			
	γ	<i>SE</i>	γ	<i>SE</i>	γ	<i>SE</i>	γ	<i>SE</i>
Age	-.01	.01	-.01	.01	-.04**	.01	-.04**	.01
Sex	-.07	.19	-.05	.21	.65	.36	.88*	.38
wSt	.55***	.05	.43***	.07	-.61***	.06	-.61***	.06
PmSt	.69***	.16	.80***	.11	-.29	.19	-.33	.22
CSA	.23	.21	.74**	.24	-.49	.28	-.51	.33
CSA*wSt	---	---	.44***	.15	---	---	-.02	.14
CSA*PmSt	---	---	-.32	.19	---	---	.11	.31

	Med.1: aER		Med.2: mER	
	<i>B</i>	<i>SE</i>	<i>B</i>	<i>SE</i>
Age	.05	.09	-.25**	.07
Sex	-1.88	2.10	-3.33*	1.54
CSA	-4.78*	2.25	6.67***	1.96

	DV: NA				DV: PA			
	γ	<i>SE</i>	γ	<i>SE</i>	γ	<i>SE</i>	γ	<i>SE</i>
Age	.00	.01	.00	.01	-.04**	.01	-.04**	.01
Sex	.05	.15	-.01	.15	.82**	.29	.99**	.30
wpSt	.41***	.06	.45***	.07	-.62***	.07	-.59***	.06
PmSt	.85***	.18	.60***	.14	-.20	.21	-.20	.21
CSA	1.09*	.64	.34	.22	-.19	.30	-.09	.30
CSA*wSt	.41***	.12	.29*	.14	-.04	.13	-.10	.14
CSA*PmSt	-.42	.30	-.48*	.22	.13	.26	.13	.30
aER	-.01	.02	-.01	.01	.07***	.01	.08***	.01
mER	.04	.04	-.05***	.01	-.01	.02	.00	.02
aER*wSt	---	---	.00	.01	---	---	-.01*	.01
aER*PmSt	---	---	.02	.01	---	---	.00	.01
mER*wSt	---	---	.02**	.01	---	---	.00	.01
mER*PmSt	---	---	.04**	.01	---	---	.00	.02

Note. NA = Negative Affect, PA = Positive Affect, Sex (0 = female, 1 = male), wSt = deviation in average stress level at EMA prompt, PmSt = average stress level across EMA period, CSA = Childhood Sexual Abuse (0 = absent, 1 = present), aER = Feeling and Me Scale, Adaptive subscale, mER = Feeling and Me Scale, Maladaptive subscale.

*** $p \leq .001$, ** $p \leq .01$, * $p \leq .05$

APPENDIX B: Figures

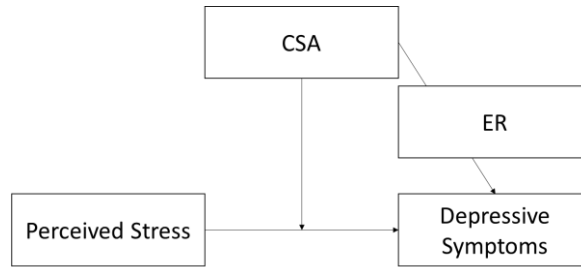


Figure 1. CSA= 0,1 for abuse free or history of abuse; Perceived Stress= PSS total score; Depressive Symptoms= CES-D total score; ER= Total scores on the adaptive and maladaptive subscales of the FAM.

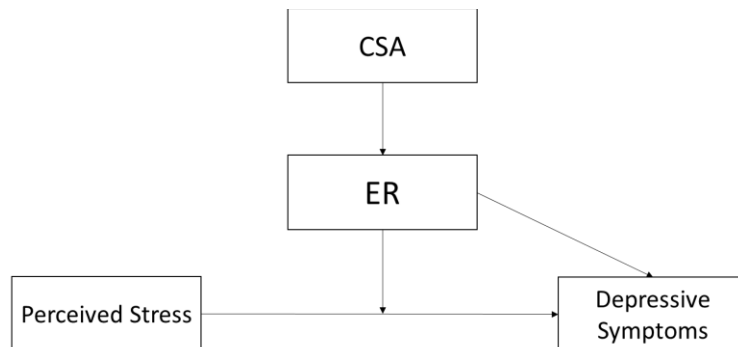


Figure 2. CSA= 0,1 for abuse free or history of abuse; Perceived Stress= PSS total score; Depressive Symptoms= CES-D total score; ER= Total scores on the adaptive and maladaptive subscales of the FAM.

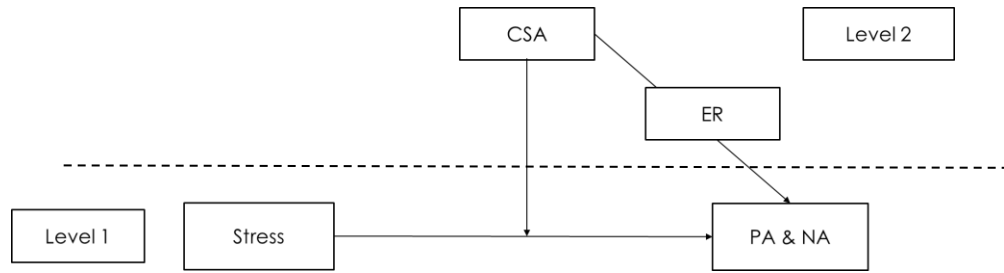


Figure 3. CSA= 0,1 for abuse free or history of abuse; PA & NA= Total positive and negative affect scores at the time of the prompt (i.e., sad, upset, angry, frustrated; happy, strong, excited); Stress= Average stress rating at the time the participant received the prompt and momentary stress (fluctuations from their average level of stress).

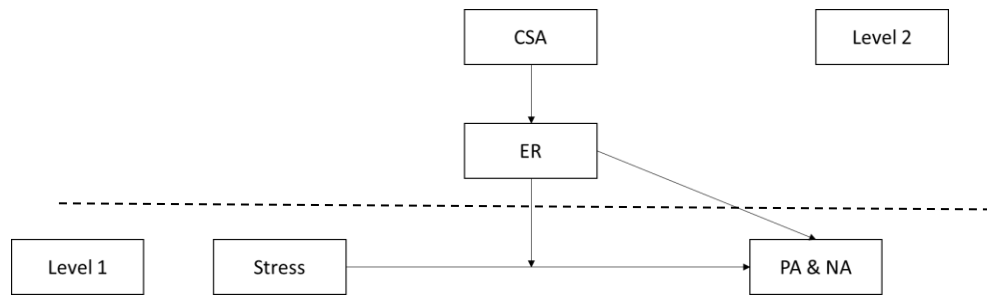


Figure 4. CSA= 0,1 for abuse free or history of abuse; ER= Total scores on the adaptive and maladaptive subscales of the FAM; PA & NA= Total positive and negative affect scores at the time of the prompt (i.e., sad, upset, angry, frustrated; happy, strong, excited); Stress= Average stress rating at the time the participant received the prompt and momentary stress (fluctuations from their average level of stress).

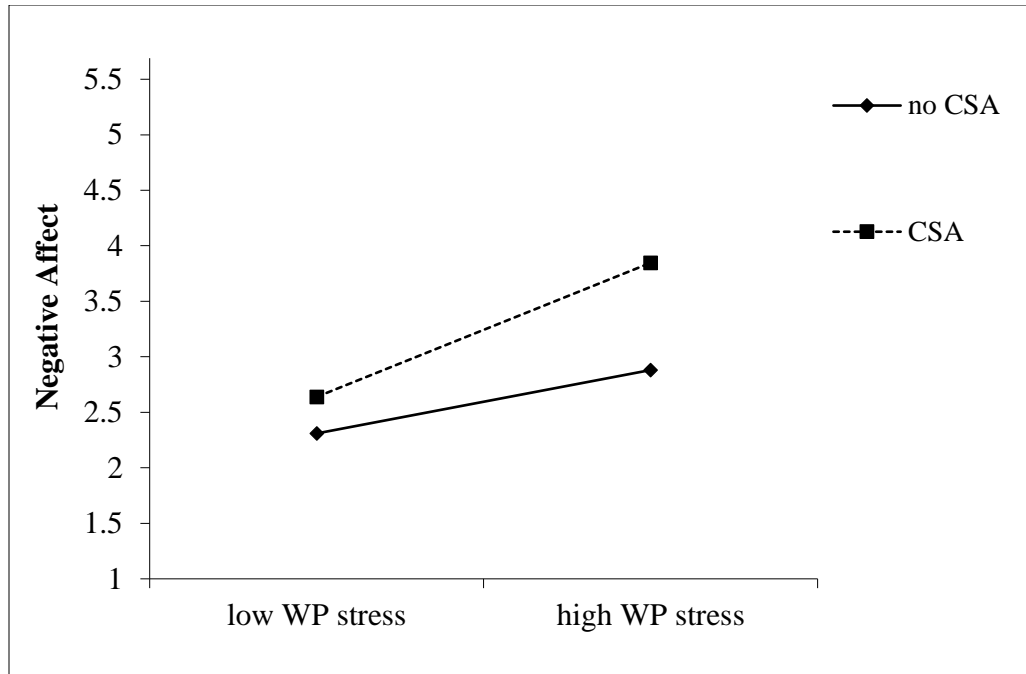


Figure 5. Childhood sexual abuse (level 2) moderation of within-subject stress fluctuations (level 1) on levels of negative affect (level 1). WP stress= deviations from participant's average stress levels.

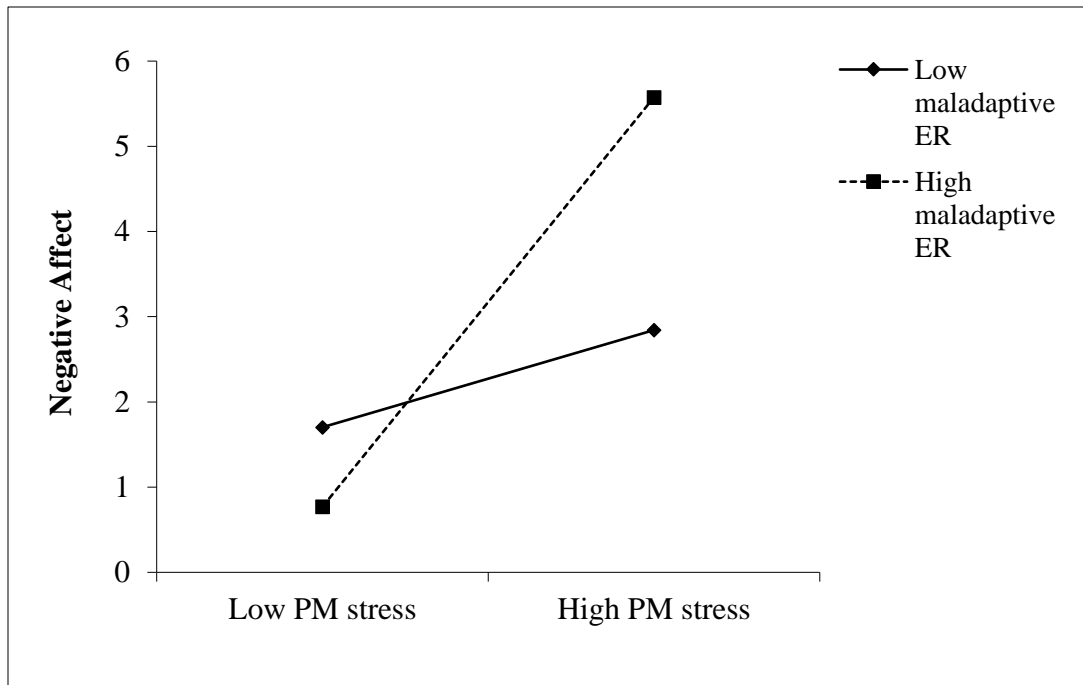


Figure 6. Maladaptive ER (level 2) moderation of average stress levels (level 2) on concurrent negative affect (level 1). MMR= maladaptive ER; PMSTRESC= participant's mean stress level; CNA= current negative affect level.

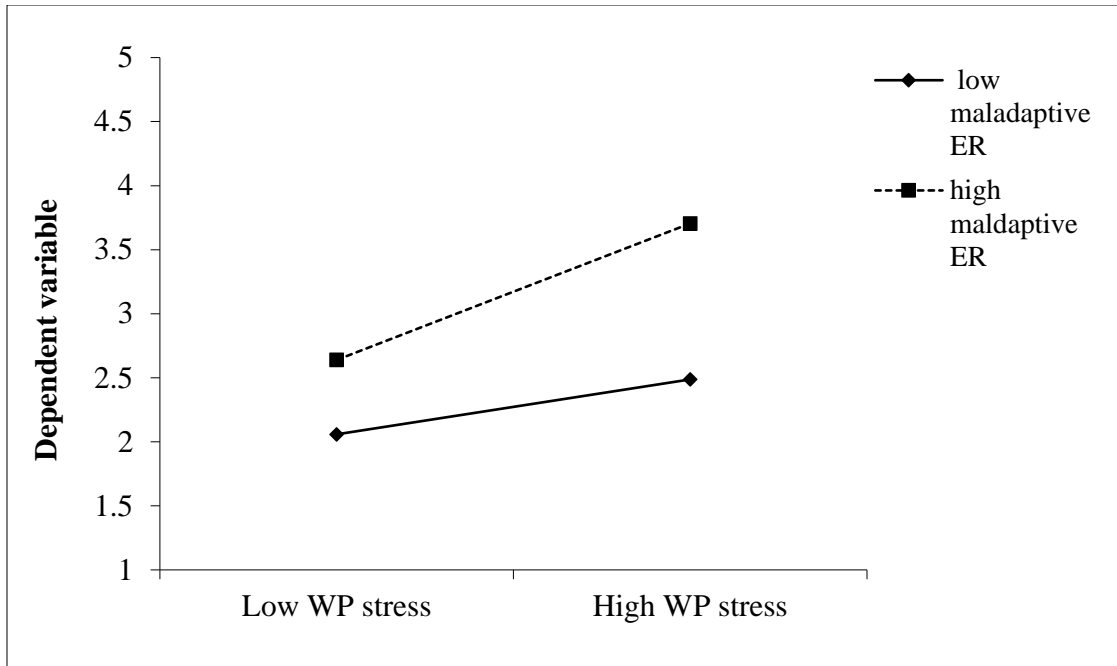


Figure 7. Maladaptive ER (level 2) moderation of within-subject stress fluctuations (level 1) on concurrent negative affect (level 1). MMR= maladaptive ER; WPSTRS= within-subject stress deviation; CNA= current negative affect level.

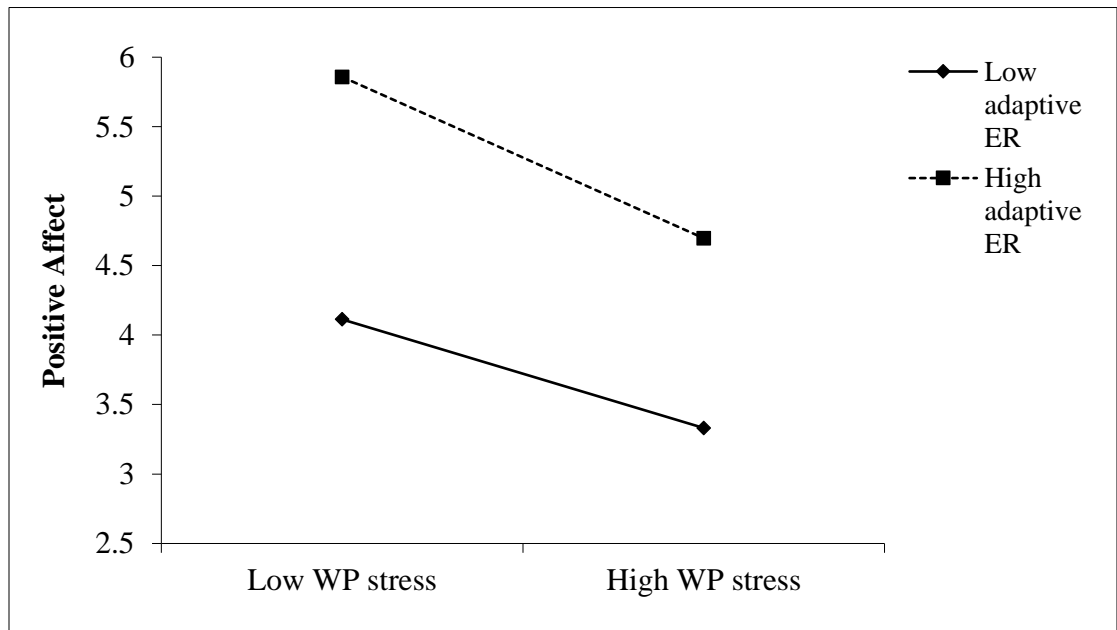


Figure 8. Adaptive ER (level 2) moderation of within-subject stress fluctuations (level 1) on concurrent positive affect (level 1). AMR= adaptive ER; WPSTRS= within-subject stress deviation; CPA= current positive affect level.