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Regular Article

Emotion regulation deficits mediate childhood sexual abuse effects on stress sensitization and depression outcomes

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Abstract

Child sexual abuse (CSA) is a notable risk factor for depressive disorders. Though multiply determined, increased sensitivity to stress (stress sensitization) and difficulty managing distress (emotion regulation) may reflect two pathways by which CSA confers depression risk. However, it remains unclear whether stress sensitization and emotion regulation deficits contribute to depression risk independently or in a sequential manner. That is, the frequent use of maladaptive emotion regulation responses and insufficient use of those that attenuate distress (adaptive emotion regulation) may lead to stress sensitization. We tested competing models of CSA, stress sensitization, and emotion regulation to predict depression symptoms and depressive affects in daily life among adults with and without histories of CSA. Results supported a sequential mediation: CSA predicted greater maladaptive repertoires that, in turn, exacerbated the effects of stress on depression symptoms. Maladaptive responses also exacerbated the effects of daily life stress on contemporaneous negative affect (NA) levels and their increase over time. Independent of stress sensitization, emotion regulation deficits also mediated CSA effects on both depressive outcomes, though the effect of maladaptive strategies was specific to NA, and adaptive responses to positive affect. Our findings suggest that emotion regulation deficits and stress sensitization play key intervening roles between CSA and risk for depression.

Keywords: childhood sexual abuse, depression, ecological momentary assessment, emotion regulation, stress

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Sexual abuse in the form of unwanted sexual contact or related sexual experiences (e.g., child pornography) during childhood and adolescence is common (Murray, Nguyen, & Cohen, 2014; World Health Organization, 1999), and globally affects between 18–19.7% of girls and 7.6–7.9% of boys (Stoltenborgh, Van Ijzendoorn, Euser, & Bakermans-Kranenburg, 2011). These rates mirror those observed in the US (Merrick, Ford, Ports, & Guinn, 2018) and likely underestimate the prevalence of child sexual abuse (CSA), given its stigmatizing nature and the reticence of those affected to report its occurrence (Katzenstein & Fontes, 2017). Importantly, the adverse effects of CSA are far reaching and are associated with academic underachievement (Boden, Horwood, & Fergusson, 2007), underemployment (Gilbert et al., 2009), and with difficulty forming and maintaining fulfilling social and romantic relationships (DiLillo & Long, 1999; Finkelhor, Hotaling, Lewis, & Smith, 1990; Richards, Tillyer, & Wright, 2017). Further, histories of CSA are linked to many adverse mental health outcomes across development (Gilbert et al., 2009; Turner, Taillieu, Cheung, & Afifi, 2017), of which depressive disorders are one common endpoint (Lindert et al., 2014). Indeed, CSA confers a three-to-eight fold increase in risk for developing depression before adulthood (Brown, Cohen, Johnson, & Smailes, 1999; Fergusson, Horwood, & Lynskey, 1996), and predicts more cumulative lifetime depressive episodes than histories of physical abuse (Gladstone et al., 2004; Lindert et al., 2014). As functional impairment increases with the accumulation of depressive episodes (Lewinsohn, Rohde, & Seeley, 1998), clarifying mechanisms that confer risk for depression among those with CSA may offset the far-reaching consequences of this recurrent disorder.

Mechanisms for depression risk among those with CSA

Though the relationship between CSA and depression is multiply determined, an increased sensitivity to stress (stress sensitization) (Heim et al., 2000; Luthar & Zigler, 1991) and difficulty managing distressing emotions (emotion regulation) (Chang, Kaczkurkin, McLean, & Foa, 2018; Ullman, Peter-Hagene, & Relyea, 2014) may reflect two pathways by which depression risk arises for those with histories of CSA. Further, as emotion regulation deficits exacerbate adverse effects of stress (Extremera & Rey, 2015; Humbel et al., 2018; Richardson, 2017), it is also feasible that the stress-sensitizing effects of CSA may in part be mediated by emotion regulation (ER) deficits. In the following sections, we examine the evidence for stress sensitization and ER deficits as key mechanisms for depression-related outcomes among individuals reporting CSA, and potential for the relationship between CSA and stress sensitization to be in part mediated by ER problems.

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Stress sensitization

A diathesis for the adverse effects of stress is a component of many depression models (Hammen, 2005; Hankin, 2008), and has strong support in the empirical literature (Hammen, 2005; Kessler, 1997; Monroe & Harkness, 2005). CSA may serve as one diathesis that sensitizes those with such histories to experience stress more keenly than their peers (Andersen & Teicher, 2008; Lupien, McEwen, Gunnar, & Heim, 2009), with stress sensitization evident across physiological (Heim et al., 2000) and subjective levels (Glaser, Van Os, Portegijs, & Myin-Germeys, 2006). For instance, studies link trauma during childhood, including CSA, with dysregulation in the hypothalamic–pituitary–adrenal (HPA) axis, a system in the neuroendocrine stress response (Chida & Hamer, 2008; Ulrich-Lai & Herman, 2009) that serves as a diathesis for stress in predicting suicidal ideation and self-harm behaviors (Eisenlohr-Moul et al., 2018) and is associated with depression risk (Goodyer, Herbert, Tamplin, & Altham, 2000). In a similar vein, adults with histories of CSA demonstrate greater affective reactivity, worse health outcomes, and risk for depressive disorders than their peers at comparable stress levels (Glaser et al., 2006; Thakkar & McCanne, 2000). For example, adults who experienced unwanted sexual contact during childhood endorsed higher levels of negative affect (NA) in their daily lives when exposed to minor stressors than those without histories of CSA (Glaser et al., 2006) or when they perceived the day as stressful (Weltz, Armeli, Ford, & Tennen, 2016). In parallel, Thakkar and McCanne (2000) observed a sensitivity to stress concerning self-reported symptoms of physical illness among women with histories of CSA whose daily hassles were monitored during a 1-month period. Though those with and without CSA reported similar levels of daily life hassles, somatic consequences were more severe for participants reporting CSA (Thakkar & McCanne, 2000). Importantly, findings also show that adults reporting CSA are at an increased risk for major depression than their peers when exposed to negative life events (Bandoli et al., 2017). Taken together, these results, along with those from physiological studies, suggest that CSA may serve as a diathesis for stress, although the mechanisms by which CSA stress sensitization occurs are not entirely clear.

Emotion regulation

Considerable evidence suggests that unsuccessful efforts to downregulate distress are mechanisms for depression risk (Joormann & Gotlib, 2010). Such failures may arise from the insufficient or ineffective use of strategies that reduce distress in the short- and long-term (adaptive ER) and the abundant use of those that paradoxically exacerbate it (maladaptive ER) (Kovacs & Lopez-Duran, 2010; Kovacs, Rottenberg, & George, 2009). Abuse disrupts ER development that is believed to follow a maturational course (Heleniak, Jenness, Vander Stoep, McCauley, & McLaughlin, 2016; Kim-Spoon, Cicchetti, & Rogosch, 2013; Kovacs et al., 2019; Thompson, 2011). In support, CSA is associated with less effective ER repertoires across development (Kim-Spoon et al., 2013; Séguin-Lemire, Hébert, Cossette, & Langevin, 2017; Ullman et al., 2014), including the frequent use of such maladaptive ER responses as rumination (Heleniak et al., 2016), avoidance (Hyman, Paliwal, & Sinha, 2007; O'Mahen, Karl, Moberly, & Fedock, 2015), and substance use (Ullman et al., 2014). A small section of available literature also links CSA with the reduced use of adaptive ER responses (e.g., cognitive reappraisal, Carvalho Fernando et al., 2014; but see Esposito & Clum, 2002) and deficits in processes that support adaptive ER (Kim & Cicchetti, 2010; Klanecky, Woolman, & Becker, 2015). In summary, a corpus of work associates CSA with ER deficits that may serve an intervening role between CSA and depression risk.

Stress sensitization via emotion regulation deficits

Though not examined in the literature, conceptual and empirical works support the possibility for ER deficits to mediate effects of CSA on stress sensitization. Like CSA, ER deficits have been put forward as a diathesis (Deater-Deckard, Li, & Bell, 2016), with maladaptive ER responses exacerbating the adverse effects of stress (Extremera & Rey, 2015; Humbel et al., 2018; Richardson, 2017) and adaptive responses reducing them (Troy, Wilhelm, Shallcross, & Mauss, 2010). For example, those who frequently engage in emotional suppression report lower levels of positive affect (PA) when confronted with stressors in everyday life as compared to those who infrequently suppress their emotions (Richardson, 2017). Others found similar negative associations between state PA and emotional nonacceptance, low emotional clarity, and poor emotional awareness (Humbel et al., 2018). Conversely, those who frequently engage in cognitive reappraisal have been shown to report less stress-related NA (Troy & Mauss, 2011) and to be protected against a reduction in PA (Deater-Deckard et al., 2016). Such buffering effects were also noted for depression symptoms (Deater-Deckard et al., 2016; Troy et al., 2010). Therefore, as CSA is tied to ER deficits that, in turn, exacerbate the effects of stress, it is feasible that ER deficits and stress sensitization relate CSA to depression risk in a sequential manner. However, most studies that examine the relationship between CSA and depression risk focus on either stress sensitization or ER deficits, employ cross-sectional designs, and rely on self-report methods that are susceptible to reporting biases and limited self-knowledge (Simonich et al., 2004).

Current study

The present study examined two conceptual models by which stress sensitization and ER deficits intervene between histories of CSA and two depression-related outcomes, contemporaneous depression symptoms and depressive affects marked by low PA and high NA (Clark & Watson, 1991) across a week-long ecological momentary assessment (EMA). For each outcome, we investigated whether stress sensitization and ER deficits serve as independent, multiple mediators of CSA effects. Then, we tested a sequential mediation between CSA, ER deficits, stress sensitization, and depression-related outcomes.[1] Based on the extant literature, we hypothesized that histories of CSA would: (a) exacerbate the effects of stress on depression symptoms and depressive affects (i.e., stress sensitization), and (b) predict the reduced dispositional use of adaptive ER responses and the frequent use of maladaptive responses that, in turn, would mediate the relationship between CSA and depression-related outcomes. We also hypothesized that the effects of CSA on depression-related outcomes would be sequentially mediated via ER deficits and stress sensitization.

[1]We treat stress sensitization as a single construct when describing its conceptual relationships with CSA, ER, and depression-related outcomes. Statistically, however, stress sensitization is measured as the moderation of stress by CSA. Therefore, though we refer to stress sensitization and ER deficits as intervening variables in mediation models, stress sensitization indexes the conditional effects of stress for those with histories of CSA in multiple mediation models, and the conditional indirect effects of CSA via ER deficits in the sequential mediation models.

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Method

Participants

Participants were 142 community-dwelling adults and undergraduate students (71% female, M = 26.63 years, SD = 10.81) who were recruited through online advertisements, referrals from outpatient treatment facilities, and from an undergraduate psychology student subject pool; 32 (23%) reported histories of sexual abuse before the age of 18 years. Participants' racial composition was predominantly Caucasian (58%) and African American (23%), with some endorsing Middle Eastern (5%), multi-racial (4%), and "other" backgrounds (2%), and the remaining participants self-identifying as South Asian, South-East Asian, Native American, and Hawaiian. Of these participants, 109 (29 with histories of CSA) took part in the EMA protocol that is described later in this section; those who did not participate did not have access to cellular phones or had phones that were incompatible with our EMA software.

Seventy-one participants (50%) reported lifetime histories of depressive disorders (45% major depressive disorder, 4% dysthymic disorder, 1% depressive disorder nos), of whom 26 were in a major depressive episode at the time of the study. Participants also evidenced lifetime histories of generalized anxiety disorder (23%), social anxiety (14%), panic disorder (12%), specific phobia (12%) and posttraumatic stress disorder (PTSD) (6%). Twenty-three participants' (16%) histories were free of lifetime psychiatric disorders.

Psychosocial and clinical evaluation

Current and lifetime histories of psychiatric disorders were assessed using the Structured Clinical Interview for DSM-IV (fourth edition of *Diagnostic and Statistical Manual of Mental Disorders*) Disorders (SCID-I) (First, Spitzer, Gibbon, & Williams, 1994) by advanced graduate students and one of the authors (IY). The SCID-I is a well-validated measure of psychiatric disorders that evidenced good inter-rater reliability in this study (SCID-I, Fleiss' κ = .91). Histories of "sexual abuse or rape" were ascertained during the clinical interview, as were the age and circumstance of their experience. A positive history of CSA reflects unwanted sexual contact from an adult that occurred before the participant reached the age of majority. All mandated reporting procedures were followed when instances of CSA were disclosed.

Measures

Depression symptoms

The Center for Epidemiological Depression Scale (CES-D; Radloff, 1977) was used to measure participants' depression levels during the prior week. The CES-D is a well-validated 20-item scale that demonstrated good psychometric properties in this study (α = .90).

Perceived stress

The Perceived Stress Scale (PSS; Cohen, Kamarck, & Mermelstein, 1983) is a well-validated 10-item survey that measures the degree to which respondents appraise events during the prior month as unpredictable, uncontrollable, and stressful. The PSS had good psychometric properties in this study (α = .93).

Emotion regulation

The Feelings and Me (FAM; Kovacs et al., 2009) questionnaire is a 54-item survey of adaptive and maladaptive cognitive, behavioral, and interpersonal responses that are dispositionally deployed in order to attenuate sadness and upset feelings. These responses measure mood repair, a component of ER dealing with downregulation of distress and dysphoria (Kovacs et al., 2009). Response repertoires are aggregated into adaptive and maladaptive ER indices that have shown excellent clinical validity in predicting incidents of depressive episodes and their recurrence (Kovacs et al., 2009, 2016). Both adaptive and maladaptive ER indices showed strong internal consistency in this study (α = .88–.89).

Depressive affects

EMA indices of PA and NA were measured using items drawn in part from the Positive Affect and Negative Affect Schedule (Watson, Clark, & Tellegen, 1988). Participants responded via a 5-point Likert scale (0 = *very slightly/not at all* and 4 = *extremely*) to adjectives indexing PA (happy & excited) and NA (sad & upset) concerning how they "feel at this moment." Item pairs were aggregated to form PA and NA indices that evidenced good internal consistency at each EMA assessment across the measurement period (PA average α = .83; NA average α = .78).

Daily life stress

Stress in daily life reflected the degree to which respondents perceived stress at the time of the EMA prompt. Participants responded via a 5-point Likert scale (0 = *very slightly/not at all* and 4 = *extremely*) concerning how "stressed" they "feel at this moment."

Overall procedures[2]

Study procedures were approved by the Institutional Review Board and executed in two parts: a laboratory visit and an EMA protocol. During the laboratory visit, participants completed psychosocial and clinical evaluations, survey measures, and experimental procedures, the data from which are not included in this study. Participants were then oriented to the EMA protocol, familiarized with the EMA questions, and signed up to receive text messaged links for the EMA survey on their cell phones using the SurveySignal software (Hofmann & Patel, 2015) or software akin to SurveySignal that was developed for one of the authors (IY). Participants were compensated for their time following their lab visit and EMA completion.

EMA sampling generally occurred five times between 9 a.m. and 9 p.m. during the 7–8 contiguous days following the laboratory visit via fixed prompts that sampled participants' affective states evenly across the morning, afternoon, and evening hours. Following best practices (Mehl & Conner, 2012), participants were sent a reminder prompt 15 min after receiving the initial text message within a given sampling period and were allowed a 30-min window to complete a given EMA survey before its deactivation.

Statistical analyses

Data analyses were conducted using SAS version 9.4 software. Missing values comprised less than 9% of cross-sectional survey

[2]Eleven participants were drawn from a study that involved an 8-day EMA measurement period and did not include the PSS. Data from the PSS were missing by design for these participants and was therefore not included in the Little's χ^2 calculation (Enders, 2010).

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data, with the exception of the PSS for which data were missing by design for 8% of respondents, and was partially completed by 8% of those who received the measure. Participants responded on average to 78% of EMA prompts (3,016 of the possible 3,870), which reflects a high level of compliance (Courvoisier, Eid, & Lischetzke, 2012), and less than 2% of data were missing from incomplete or skipped responses. Missing value analyses revealed that cross-sectional and EMA data were missing completely at random, Little's χ^2 (16–18) = 12.95–21.22, p = .17–.28. Following best practices (Enders, 2010), we employed multiple imputation to recover missing values in order to reduce the analytic bias that is associated with their presence. Specifically, we imputed 200 data sets via the estimation–maximization (EM) algorithm that were then used in hypothesis testing: statistical models were fit to each data set, and the resulting parameter estimates were pooled across imputations (Enders, 2010).

Depression symptoms models

We employed moderation, mediation, and moderated-mediation models using cross-sectional data from the full sample (N = 142) to test (a) stress sensitization effect of sexual abuse histories on depression symptoms, (b) the mediation of sexual abuse history effect on depression symptoms by adaptive and maladaptive emotion regulation repertoires, and (c) the sequential mediation of sexual abuse effect on depression symptoms via emotion regulation deficits and stress sensitization. In these models, stress sensitization reflects the CSA moderation of perceived stress effect on depression symptoms, a common methodological approach to modeling diathesis–stress relationships (e.g., Monroe & Harkness, 2005; Morris, Ciesla, & Garber, 2010).

First, we tested the effects of CSA on stress sensitization and the two ER indices to establish relationships between our predictor of interest and intervening variables. Then, akin to multiple mediation, we regressed depression symptoms on CSA, stress sensitization, and ER, and, where appropriate, calculated simple slopes and indirect effects of CSA onto our outcome of interest. Finally, akin to sequential mediation, we tested the indirect effects of CSA on stress sensitization and subsequent depression symptoms via adaptive and maladaptive ER indices. This model is mathematically equivalent to the moderated-mediation model, wherein the indirect effects of sexual abuse via ER are conditional (Preacher, Rucker, & Hayes, 2007; K. Preacher, personal communication, October 26, 2018).

Following best practices, asymmetric confidence intervals (CIs) around indirect effects were estimated using the PRODCLIN method (Tofighi & Thoemmes, 2014) in the RMediation package (Tofighi & MacKinnon, 2011), as were the CIs for the conditional indirect effects that were modeled at the uncentered values of CSA, and at one standard deviation above and below the means of the ER indices.

Depressive affects models

Our general approach to testing our hypotheses with EMA data from the reduced sample (n = 109) mirrored that of the depression symptom models; we first tested the effects of CSA on stress sensitization and ER indices, as well as the multiple and sequential intervening effects of stress sensitization and ER between CSA and depressive affects. However, these models were conducted in a multilevel framework to accommodate the nesting of EMA responses (Level 1) within observation days (Level 2) and participants (Level 3), as well as the notable within-person stability of affective states (Level 3 ICC_{NA} = .50 and ICC_{PA} = .46, ps < .001) and the influence of observation days on both affects (Level 2 ICC_{NA} = .09 and ICC_{PA} = .21, ps < .001). Also, we tested our hypothesized associations between sexual abuse, ER and stress sensitization with respect to the contemporaneous effects of stress on NA and PA at the time of the EMA prompt (contemporaneous models), as well as their prospective effects on both affective outcomes (prospective models); separate models were fit for each affective outcome.

Following best practices, EMA-based stress measurements were decomposed into their time-invariant (participant's average stress level over the measurement period) and time-varying components (deviation from participant's average stress levels at a given EMA observation) (Algina & Swaminathan, 2011; Enders & Tofighi, 2007). Continuous Level 3 predictors were grand mean centered (i.e., age and ER indices) (Enders & Tofighi, 2007) while dichotomous categorical variables (i.e., gender and CSA) retained their original metric (Singer & Willett, 2003).

In contemporaneous models, stress sensitization effects reflect the Level 3 interaction between CSA and participants' average stress levels and the cross-level interaction between CSA and within-person deviation from their average stress level at the time of the EMA prompt. In prospective models, stress sensitization is indicated by the previously described Level 3 interaction and cross-level interaction between CSA and within-person deviation from a given participant's average stress level at the prior EMA prompt. In these models, the effects of affective state at the prior EMA prompt, time since the prior EMA prompt, and their interaction were covaried to accommodate the unequal temporal distance between affect measurements. Random intercept and slopes were estimated for Level 1 variables as warranted (see Supplemental material for model equations).

Results

Sample characteristics

Demographic, psychiatric, and psychosocial characteristics of those with and without histories of CSA are presented in Table 1. Those who experienced CSA were approximately 11 years older and more likely to be women. They also reported higher levels of perceived stress and depression symptoms and were overrepresented among those with depression histories and those in the midst of a depressive episode. Those with CSA did not significantly differ from their peers in their racial distributions, nor histories of anxiety disorders. To account for differences in demographic characteristics, the effects of age and gender were statistically covaried in models.

Do emotion regulation deficits and stress sensitization mediate effects of CSA on depression symptoms?

Stress sensitization

As hypothesized, our results showed that, independent of demographic characteristics, histories of CSA and high levels of perceived stress were associated with concurrent elevation in depression symptoms, B_{CSA} = 7.37, t (137) = 2.70, p = .008, B_{PSS} = 10.73, t (137) = 3.56, p < .001. However, the association between perceived stress and depression symptoms did not vary as a function of abuse histories, as indicated by the nonsignificant second-order effect of the two variables (see Table 2).

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Table 1. Demographic characteristics, psychiatric parameters, and perceived stress levels of those with and without childhood sexual abuse (CSA) histories

Variable	CSA ($n = 32$), M (SD)/%	No CSA ($n = 110$), M (SD)/%	Test statistic
Age	35.03 (10.92)	24.18 (9.52)	t (140) = 5.49, $p < .001$
Sex (% Female)	87%	66%	χ^2 (1) = 5.39, $p = .02$
Race[a]			χ^2 (2) = 4.72, $p = .09$
African American	37%	19%	
Caucasian	47%	62%	
"Other"	16%	19%	
Current Dep. D/O	63%	17%	χ^2 (1) = 25.45, $p < .001$
Past Dep. D/O Hx	66%	41%	χ^2 (1) = 6.09, $p = .01$
Anx. D/O Hx	47%	38%	χ^2 (1) = .78, $p = .38$
Depression Sx	31.29 (13.49)	14.37 (10.74)	t (139) = 7.31, $p < .001$
Perceived Stress	36.65 (10.52)	27.42 (8.38)	t (140) = 4.58, $p < .001$

Note. Dep. D/O = depressive disorder (major depression, dysthymic disorder, or depressive disorder NOS), Anx. D/O = anxiety disorder (generalized anxiety disorder, social anxiety disorder, panic disorder, specific phobia, and posttraumatic stress disorder), Depression Sx = Center for Epidemiologic Studies Depression Scale, Perceived Stress = Perceived Stress Scale.
[a]"Other" reflects aggregated racial categories to accommodate 0 frequency cells.

Emotion regulation

Next, we tested whether ER deficits could serve as mediators of CSA by regressing dispositional adaptive and maladaptive ER repertoires on abuse group membership, and then adding both emotion regulation indices to the stress sensitization model described above (see Table 2). In support of their potential mediation effects, those with histories of sexual abuse reported deploying maladaptive ER responses more readily, $B_{CSA} = 6.97$, t (138) = 3.56, $p < .001$, and engaging in adaptive ER responses less frequently during times of distress than their abuse-free peers, $B_{CSA} = -4.78$, t (138) = 2.13, $p = .04$. In turn, the frequent use of maladaptive ER responses predicted elevation in depression symptoms, $B_{mER} = .36$, t (134) = 8.37, $p < .001$, while adaptive ER reduced depression symptoms, $B_{aER} = -.18$, t (134) = 2.95, $p = .004$. Both ER indices mediated the direct effects of CSA on depression symptoms, $B_{indmER} = 2.54$, 95% CI .70–5.01, $B_{indaER} = .84$ 95% CI .04–1.99.

Do emotion regulation deficits and stress sensitization sequentially mediate effects of CSA on depression symptoms?

To test this possibility, second-order effects between each emotion regulation index and perceived stress were added to the previously described "multiple mediation" models in order to test the conditional indirect effects of abuse on depression symptoms via ER deficits and stress sensitization (see Table 2). Because stress sensitization reflects a moderation effect, indirect effects were examined via simple slopes at one standard deviation above and below the mean of a given ER index (Cohen, Cohen, West, & Aiken, 2003).

As hypothesized, maladaptive ER repertoires significantly moderated the effect of perceived stress on depression, $B_{mERxPSS} = .02$, t (132) = 1.99, $p = .05$ (see Figure 1). Simple slopes analyses that probed the interaction revealed greater depression severity as a function of stress for those with elevated maladaptive ER levels, $B_{mER,+1SD} = .97$, t (134) = 7.57, $p < .001$, relative to those with low maladaptive ER repertoires, $B_{mER,-1SD} = .61$, t (134) = 4.84, $p < .001$. Tests of conditional indirect effects revealed significant mediation of CSA on depression via the moderation of perceived stress by maladaptive ER, $B_{indmER,+1SD} = 6.79$, 95% CI 2.90–11.22, $B_{indmER,-1SD} = 4.27$, 95% CI 1.66–7.54. In contrast to expectation, adaptive ER repertoires did not attenuate the adverse effect of perceived stress on depression, and therefore did not sequentially mediate the effects of CSA on depression via stress sensitization.

Do emotion regulation deficits and stress sensitization mediate effects of CSA on depressive affects in daily life?

We examined this question in two ways, by testing the effects of CSA, stress sensitization, and ER repertoires on contemporaneous and prospective associations between stress and depressive affects (see Tables 3 and 4).

In the contemporaneous models, CSA predicted NA elevation, $\gamma_{CSA} = 1.38$, t (103) = 5.07, $p < .001$), but was unrelated to PA. In a similar vein, participants' average stress level during the measurement period predicted elevation in NA, $\gamma_{PmStress} = 1.02$, t (103) = 4.79, $p < .001$, and reduction in PA at a trend level, $\gamma_{PmStress} = -.31$, t (103) = 1.66, $p = .10$, as did momentary increases in stress at the time of the EMA prompt that significantly predicted both NA elevation, $\gamma_{wStress} = .41$, t (103) = 7.08, $p < .001$, and PA reduction, $\gamma_{wStress} = -.64$, t (103) = 11.81, $p < .001$.

In the prospective models, sexual abuse histories evidenced a trend for worsening NA over time, $\gamma_{CSA} = .41$, t (100) = 1.75, $p = .08$. Participants' average stress level during the measurement period also predicted elevation in NA, $\gamma_{PmStress} = .79$, t (100) = 4.89, $p < .001$, as did momentary increases in stress at the time of the prior EMA prompt at a trend level, $\gamma_{wStress} = .07$, t (100) = 1.88, $p = .06$. Neither CSA nor the two stress indices predicted change in PA levels over time.

Stress sensitization

In support of their stress sensitization effect, sexual abuse histories evidenced a cross-level interaction with participants' momentary deviations from their average stress levels to predict contemporaneous NA, $\gamma_{CSAxwStress} = .41$, t (103) = 3.42, $p < .001$ (see Figure 2, Panel A). Simple slopes analysis revealed a stronger association between stress and concurrent NA levels for those with sexual abuse histories, $\gamma_{CSA} = .82$, t (103) = 3.78, $p < .001$, than for their peers, $\gamma_{NoCSA} = .41$, t (103) = 6.83, $p < .001$. Sexual abuse histories did not moderate the relationship between participants' average stress levels and NA, nor between both stress indices and PA.

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Table 2. Multiple and sequential mediation of child sexual abuse history's effect on depression symptoms via stress sensitization and emotion regulation

	DV: depression symptoms			
Variables	*B*	*SE*	*B*	*SE*
Age	.01	.09	.01	.09
Sex	−1.12	1.32	−1.10	1.32
Stress	1.00***	.09	1.00***	.08
CSA	7.37**	2.73	7.00†	3.98
CSA*Stress	—	—	.05	.08
	Med.$_1$ Adaptive ER		Med.$_2$ Maladaptive ER	
	B	*SE*	*B*	*SE*
Age	.05	.09	−.25**	.07
Sex	−1.88	2.10	−3.33*	1.54
CSA	−4.78*	2.25	6.97***	1.96
	DV: depression symptoms			
	B	*SE*	*B*	*SE*
Age	.10	.07	.10	.07
Sex	−.71	1.27	−.71	1.27
Stress	.79***	.09	.79***	.09
CSA	7.40*	3.39	7.38*	3.39
CSA*Stress	−.31	.38	−.31	.38
aER	−.18**	.06	−.18**	.06
mER	.36**	.12	.37**	.12
aER*Stress	—	—	.00	.01
mER*Stress	—	—	.02*	.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.
***$p \leq .001$, **$p \leq .01$, *$p \leq .05$, †$p \leq .10$.

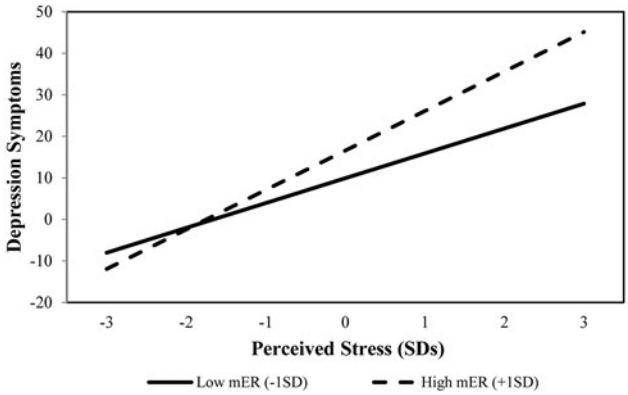

Figure 1. Maladaptive emotion regulation (mER) moderation of perceived stress effects on depression symptoms.

In the prospective models, sexual abuse histories and deviations from participants' average stress levels during the prior EMA prompt jointly predicted significant change in subsequent PA, $\gamma_{CSAxwStress} = -.19$, t (98) = 2.13, $p = .03$, as well as change in NA at a trend level, $B_{CSAxwStress} = .13$, t (98) = 1.74, $p = .08$ (see Figure 2, Panels B and C). Consistent with stress sensitization, CSA was associated with reduced PA across successive EMA observations, $\gamma_{wStress} = -.13$, t (98) = 1.98, $p = .05$, and increased NA, $\gamma_{wStress} = .19$, t (98) = 2.89, $p = .005$. Increased stress relative to average levels was unrelated to change in PA and NA for those without sexual abuse histories, γs = .06, t (98) = 1.09–1.40, ps = .17–.28.

Emotion regulation

In support of emotion regulation's role in distress among those with CSA, sexual abuse histories predicted elevated maladaptive, B = 7.03, t (105) = 3.21, $p = .002$, and reduced adaptive repertoires, B = −4.81, t (105) = 1.99, $p = .05$, across contemporaneous and prospective models. In turn, adaptive ER repertoires predicted elevations in contemporaneous PA levels, γ = .07, t (96) = 3.41, $p = .002$, that increased over successive EMA prompts, γ = .06, t (96) = 4.55, $p < .001$, as well as mediated the effects of CSA on PA contemporaneously $\gamma_{indaER} = -.35$ 95% CI −.81–−.003, and prospectively, $\gamma_{indaER} = -.29$ 95% CI −.65–−.002. Adaptive ER repertoires were unrelated to NA indices. Though unrelated in contemporaneous models, maladaptive ER repertoires predicted NA in the prospective model, $\gamma_{mER} = .04$, t (96) = 3.61, $p < .001$, and mediated the effect of CSA on increased NA across successive EMA observations, $\gamma_{indbER} = .25$ 95% CI .11–.49. Maladaptive repertoires were unrelated to indices of PA.

Do emotion regulation deficits and stress sensitization sequentially mediate effects of CSA on depression affects in daily life?

We tested this possibility by adding second-order effects of the adaptive and maladaptive ER repertoires with indices of stress in the contemporaneous and prospective models. In support of sequential mediation, maladaptive ER moderated contemporaneous effects of participants' average stress levels and their momentary deviations on NA, $\gamma_{mERxPmStress} = .04$, t (95) = 2.08, $p = .04$, $\gamma_{mERxwStress} = .02$, t (95) = 2.44, $p = .02$, (see Figure 3, Panels A and B), and the effects of participants' average stress levels in the prospective NA model, $\gamma_{mER} = .04$, t (92) = 2.18, $p = .03$ (see Figure 3, Panel C). Post hoc probes of the interaction terms revealed that tendencies to deploy maladaptive ER responses exacerbated the effects of stress on contemporaneous NA across participants' average stress levels, $\gamma_{PmStress,+1SD} = 1.08$, t (95) = 4.25, $p < .001$, $\gamma_{PmStress,\ -1SD} = .41$, t (95) = 1.73, $p = .09$, and momentary deviations from those levels $\gamma_{wStress,+1SD} = .59$, t (95) = 5.78, $p < .001$, $\gamma_{wStress,\ -1SD} = .25$, t (95) = 4.08, $p < .001$. Similar effects emerged in the prospective model, whereby maladaptive ER potentiated the adverse effects of participants' average stress levels on NA over time, $B_{PmStress,+1SD} = .94$, t (92) = 4.29, $p < .001$, $\gamma_{PmStress,\ -1SD} = .28$, t (92) = 1.22, $p = .22$. Importantly, and as hypothesized, the frequent use of maladaptive ER responses mediated stress-sensitization effects of CSA on contemporaneous NA levels, PmStress: $\gamma_{indmER,+1SD} = 7.60$, 95% CI 2.46–14.22, $\gamma_{indmER,-1SD} = 2.85$, 95% CI −.34–7.20; wStress $\gamma_{indmER,+1SD} = 4.15$, 95% CI 1.49–7.35; $\gamma_{indmER,-1SD} = 1.78$, 95% CI .56–3.35, and their change over time, γmStress : $\gamma_{mER+1SD} = 6.64$, 95% CI 2.16–12.40; $\gamma_{mER-1SD} = 1.96$, 95% CI −1.16–5.93.

Surprisingly, though adaptive ER repertoires moderated the effects of within-participant stress fluctuations on PA, $\gamma_{aERxwStress} = -.01$, t (95) = 2.00, $p = .05$, the frequent use of adaptive responses was paradoxically associated with a reduction in PA

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Table 3. Multiple and sequential mediation of child sexual abuse history's effects on contemporaneous negative and positive affects in daily life via stress sensitization and emotion regulation

	DV: NA				DV: PA			
Variables	*γ*	*SE*	*γ*	*SE*	*γ*	*SE*	*γ*	*SE*
Age	−.01	.01	−.01	.01	−.03*	.02	−.03*	.01
Sex	−.02	.16	−.02	.16	.73*	.35	.73**	.35
wSt	.41***	.05	.41***	.06	−.64***	.05	−.64***	.06
PmSt	1.02***	.21	1.02***	.25	−.31†	.19	−.31	.22
CSA	1.38***	.27	1.38	.86	−.22	.38	−.29	1.00
CSA*wSt	—	—	.41***	.12	—	—	.00	.12
CSA*PmSt	—	—	−.42	.38	—	—	.03	.42

	Med.$_1$: aER		Med.$_2$: mER	
	B	*SE*	*B*	*SE*
Age	−.03	.10	−.24**	.08
Sex	−1.53	2.34	−2.81†	1.69
CSA	−4.81*	2.42	7.03***	2.19

	DV: NA				DV: PA			
	γ	*SE*	*γ*	*SE*	*γ*	*SE*	*γ*	*SE*
Age	.00	.01	.00	.01	−.03*	.01	−.03*	.01
Sex	.05	.15	.04	.15	.82**	.29	−.82**	.30
wSt	.41***	.06	.42***	.06	−.62**	.06	−.62***	.06
PmSt	.85***	.18	.74***	.18	−.17	.19	−.17	.20
CSA	1.09†	.64	1.45*	.64	−.06	.86	−.06	.94
CSA*wSt	.41***	.12	.33**	.12	−.05	.13	−.05	.13
CSA*PmSt	−.42	.30	−.59*	.30	.09	.37	.09	.42
aER	−.01	.02	−.03	.02	.07**	.02	.07†	.04
mER	.04	.04	−.05	.04	.00	.02	.00	.04
aER*wSt	—	—	.00	.00	—	—	−.01*	.00
aER*PmSt	—	—	.01	.01	—	—	.00	.02
mER*wSt	—	—	.02*	.01	—	—	.00	.01
mER*PmSt	—	—	.04*	.02	—	—	−.01	.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.
***$p \leq .001$, **$p \leq .01$, *$p \leq .05$, †$p \leq .10$

as participants' stress increased relative to average levels, $\gamma_{aER,+1SD} = -.73$, t (95) = 8.84, $p < .001$, $\gamma_{aER,-1SD} = -.51$, t (95) = 6.87, $p < .001$ (see Figure 4), and mediated stress-sensitization effects of sexual abuse on concurrent PA via contemporaneous stress fluctuations, wStress $\gamma_{indaER,+1SD} = 3.49$, 95% CI .05–7.18; $\gamma_{indaER,-1SD} = 2.47$, 95% CI .03–5.18. Adaptive ER did not moderate effects of either stress index on NA across contemporaneous or prospective models.

Discussion

CSA confers a considerable risk across the life span for developing depressive disorders (Lindert et al., 2014; Turner et al., 2017), with stress sensitization and emotion regulation deficits put forth as plausible mechanisms through which such risk is conferred (Heim et al., 2000; Ullman et al., 2014). Yet, though both are associated with CSA and depression (Andersen & Teicher, 2008; Lupien et al., 2009), it remains unclear whether stress sensitization and emotion regulation deficits contribute to depression risk independently or sequentially. We fit multiple and sequential mediation models to test whether stress sensitization and emotion regulation deficits mediate associations between histories of CSA and depression outcomes independently of one another, or whether the more pronounced effects of stress for those with CSA arise from a reliance on maladaptive responses and insufficient use of adaptive strategies.

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Table 4. Multiple and sequential mediation of child sexual abuse history's effects on prospective negative and positive affects in daily life via stress sensitization and emotion regulation.

	DV: NA				DV: PA			
Var.	γ	*SE*	γ	*SE*	γ	*SE*	γ	*SE*
Time	−.03	.04	−.09	.06	.03	.04	.02	.04
DV_{t-1}	.03	.02	.02	.03	.10***	.03	.10***	.03
Time*DV_{t-1}	.01	.01	.03	.02	−.02	.01	−.01	.01
Age	−.01	.01	−.01	.01	−.03**	.01	−.03**	.01
Sex	.00	.14	−.01	.14	.58*	.29	.59**	.29
wSt_{t-1}	.07†	.03	.06	.04	.01	.04	.06	.06
PmSt	.79***	.16	.85***	.19	−.20	.16	−.21	.17
CSA	.41†	.23	.86	.74	−.21	.30	−.28	.92
CSA*wSt_{t-1}	—	—	.13†	.07	—	—	−.19*	.09
CSA*PmSt	—	—	−.20	.33	—	—	.04	.40

	Med.$_1$ Adaptive ER		Med.$_2$ Maladaptive ER	
Var.	*B*	*SE*	*B*	*SE*
Age	−.03	.10	−.24**	.08
Sex	−1.53	2.34	−2.81†	1.69
CSA	−4.81*	2.42	7.03***	2.19

	DV: NA				DV: PA			
Var.	γ	*SE*	γ	*SE*	γ	*SE*	γ	*SE*
Time	−.03	.04	−.09	.06	.03	.04	.02	.04
DV_{t-1}	.03	.02	.01	.03	.10***	.03	.10***	.03
Time*DV_{t-1}	.01	.01	.03	.02	−.01	−.01	−.01	.01
Age	.00	.01	.00	.01	−.03**	.01	−.03**	.01
Sex	.07	.14	.07	.13	.69**	.24	.70**	.25
wSt_{t-1}	.03	.04	.06	.04	.06	.05	.05	.05
PmSt	.69***	.18	.62***	.15	−.11	.17	−.07	.18
CSA	.62	.57	.95†	.55	−.06	.78	−.12	.85
CSA*wSt_{t-1}	.14†	.07	.11	.07	−.19*	.09	−.20*	.09
CSA*PmSt	−.19	.28	−.35	.26	.07	.35	.09	.40
aER	−.01	.01	−.02	.02	.06***	.01	.07†	.04
mER	.04***	.01	−.04	.03	−.01	.02	.02	.04
aER*wSt_{t-1}	—	—	.00	.00	—	—	.00	.00
aER*PmSt	—	—	.01	.01	—	—	−.01	.02
mER*wSt_{t-1}	—	—	.00	.00	—	—	.01	.01
mER*PmSt	—	—	.04*	.02	—	—	−.01	.02

Note. NA = negative affect, PA = positive affect, Time = interval between current and prior EMA prompt, DV_{t-1} = NA or PA level at prior EMA prompt, Sex (0 = female, 1 = male), wSt_{t-1} = deviation in average stress level at prior 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.
***$p \leq .001$, **$p \leq .01$, *$p \leq .05$, $^{\dagger}p \leq .10$

Overall, results supported a sequential mediation between CSA and depression outcomes: histories of CSA predicted greater tendencies to deploy maladaptive emotion regulation responses that, in turn, exacerbated the effects of stress on depression severity. Further, maladaptive emotion regulation repertoires mediated the contemporaneous effects of daily life stress on NA levels and their increase across successive EMA observations. Though the reduction in adaptive emotion regulation repertoires mediated the direct effects of CSA on depression symptoms and PA in prospective EMA models, their intervening

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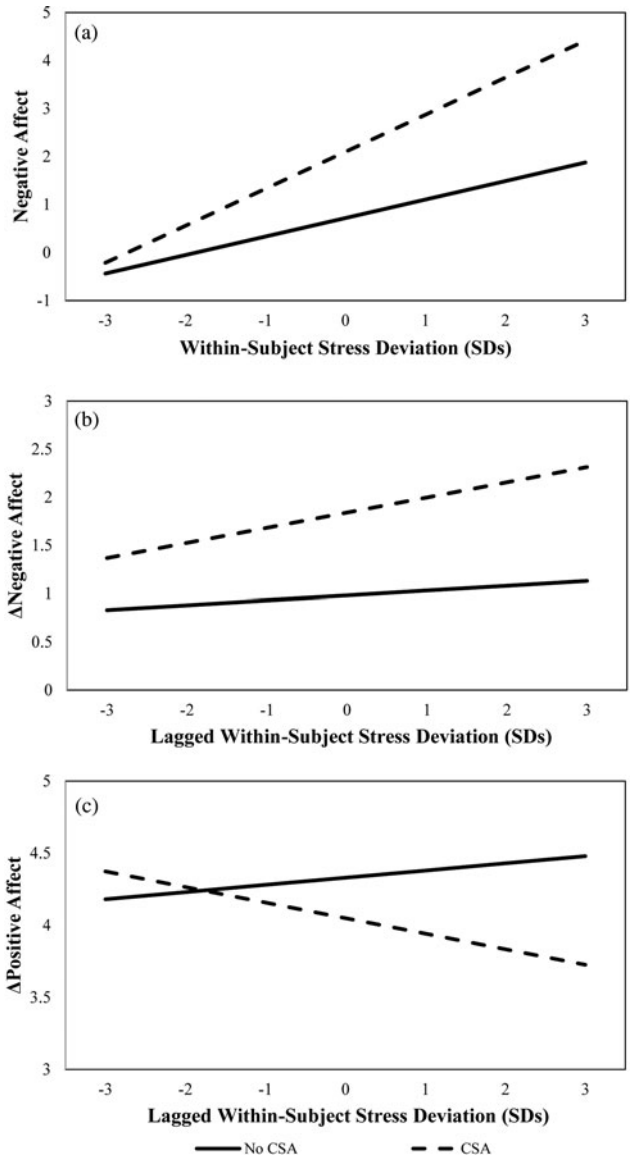

Figure 2. Childhood sexual abuse moderation of within-subject stress fluctuations on contemporaneous negative affect (NA) (Panel A), and change (Δ) in NA (Panel B) and positive affect (Panel C) across successive ecological momentary assessment (EMA) prompts.

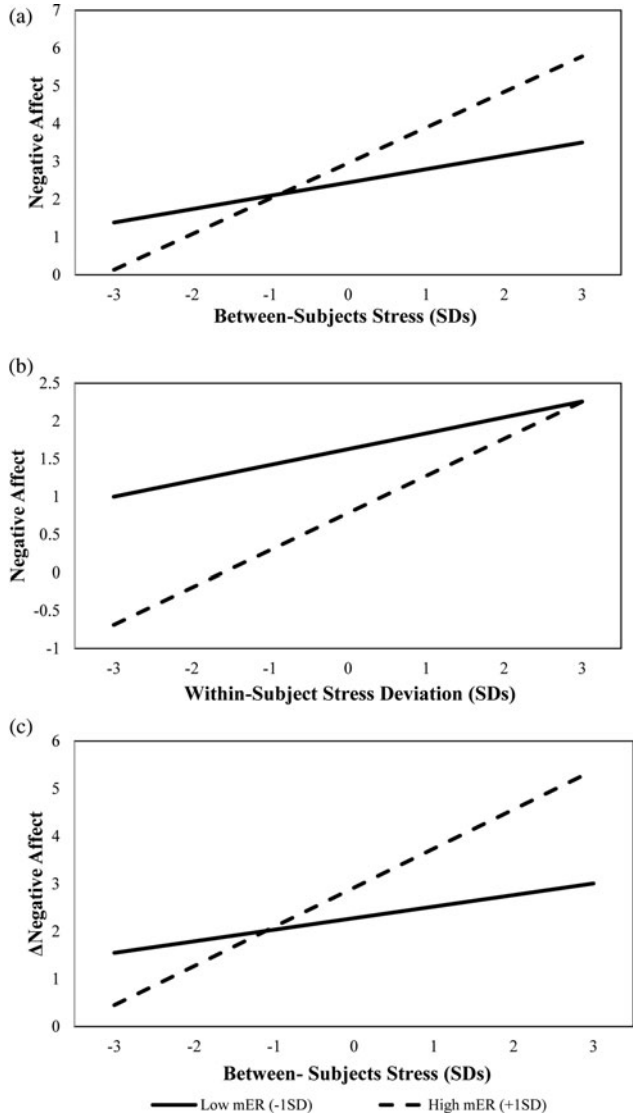

Figure 3. Maladaptive emotion regulation (mER) moderation of average stress level (Panel A) and within-subject stress fluctuations (Panel B) on concurrent negative affect (NA), and average stress levels on change (Δ) in NA (Panel C) across successive ecological momentary assessment prompts.

role between CSA and stress sensitization were inconsistent and paradoxical, as evidenced by the reduced PA in response to stress among those with CSA who frequently deployed adaptive responses.

Our results suggest that the relationship between CSA, stress, and depression is nuanced, as stress may have both transient and enduring components. Regarding the latter, we found higher perceived stress levels among those with CSA, for whom exploratory analyses also revealed higher average stress in daily life relative to their peers ($M_{CSA} = 2.17$ vs. $M_{NoCSA} = 1.75$, $p = .03$).[3] In turn, with few exceptions, elevation in stress levels predicted parallel increases in depression symptoms and depressive affects. These findings are in accord with results from a number of survey, laboratory, and experience sampling studies that connect CSA to elevated stress levels (Bandoli et al., 2017; Glaser et al., 2006; Luthar & Zigler, 1991), and with a large body of work associating stress withdepression (Hammen, 2005; Hankin, 2008). Though not of primary interest in this study, the strong relationship between CSA, stress, and depression outcomes may suggest that stress serves an intervening role between CSA and depression.

In contrast, and of primary importance, CSA-linked stress sensitization emerged only in response to fluctuations in participants' stress levels. That is, as compared to their peers, those with CSA whose stress increased relative to their average levels reported more NA and less PA that worsened over time. Importantly, some CSA stress-sensitization effects were maintained when the emotion regulation indices were added to the model, suggesting that the vulnerability for depression conferred by a history of

[3]In an exploratory analysis, we tested whether akin to perceived stress (see Table 1), histories of CSA were associated with elevated average stress levels across the EMA measurement period. Independent of the effects of age (B = −.01, $p = .08$) and gender (B = −.07. $p = .70$), those with histories of CSA reported higher average stress levels than their peers (B = .43, $p = .03$).

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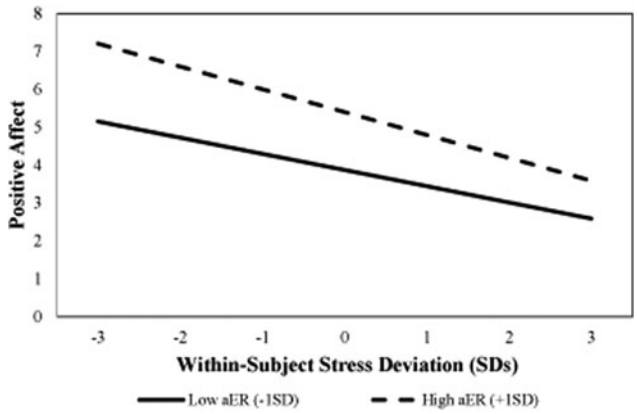

Figure 4. Adaptive emotion regulation (aER) moderation of within-subject stress fluctuations on contemporaneous hedonic affect.

CSA exceeds that which is accounted for by dispositional emotion regulation repertoires.

The distinct pattern of associations between CSA and stress has both substantive and methodological implications. Our findings imply that mechanisms predisposing those with CSA to experience ongoing stress may be distinct from those that potentiate transient stress responses. Consistent with this possibility, empirical findings differentiate basal stress levels and their reactivity across physiological and subjective dimensions (Henckens et al., 2016; Sliwinski, Almeida, Stawski, & Smyth, 2009). For example, at the physiological level, resting cortisol, a neuroendocrine marker of perceived stress (van Eck & Nicolson, 1994), evidenced an inverse pattern of amygdala activity and emotion processing relative to cortisol reactivity in response to a psychological stress (Henckens et al., 2016). Further, the two physiological stress indices were shown to be orthogonal and differed in associations with personality dimensions (Henckens et al., 2016). In a similar vein, day-to-day fluctuations in perceived stress levels have been shown to differentially predict dysphoric states in daily life from more enduring indices of perceived stress (Sliwinski et al., 2009). Given the complexity of the stress response, our results echo the call to take a multiple-levels-of-analyses perspective (Cicchetti & Dawson, 2002) to study shared and specific processes that underpin the stress experience of those with histories of CSA, both with respect to stress levels and their fluctuations.

Our findings also suggest that emotion regulation deficits play seminal, but complicated roles in the relationship between CSA and risk for depression. As in our study, CSA has been associated with the infrequent use of adaptive responses and reliance on maladaptive strategies and (Carvalho Fernando et al., 2014; Heleniak et al., 2016) that prognosticate new incidence of depression (Kovacs et al., 2009, 2016), and that meta-analytic reviews tie to depression severity (Aldao, Nolen-Hoeksema, & Schweizer, 2010; Webb, Miles, & Sheeran, 2012) and depression status (Visted, Vøllestad, Nielsen, & Schanche, 2018). However, as depression at the affective level may reflect a combination of high NA and low PA (Clark & Watson, 1991), global measures of depression symptoms feasibly obscure the unique ties between emotion regulation deficits and the two affective domains. Indeed, our results and those of others suggest some specificity between emotion regulation repertoires and affective states, with adaptive responses linked to PA (Brans, Koval, Verduyn, Lim, & Kuppens, 2013) and maladaptive ones with NA (Chaudhury et al., 2017).

As in our study, deploying adaptive strategies in response to distress predicted increased PA in the daily lives of healthy adults (Brans et al., 2013, Study 2; Brockman, Ciarrochi, Parker, & Kashdan, 2017) and patients with borderline personality disorder (BPD) (Chaudhury et al., 2017), but was less or unrelated with NA downregulation (Brockman et al., 2017; Chaudhury et al., 2017; but see Blalock, Kashdan, & Farmer, 2016 and Nezlek & Kuppens, 2008). Conversely, efforts to "find perspective," a strategy akin to rumination, and to suppress NA predicted NA upregulation (Chaudhury et al., 2017; Nezlek & Kuppens, 2008; but see Blalock et al., 2016 and Brockman et al., 2017), but not PA (Chaudhury et al., 2017).

We do not imply a strong specificity between emotion regulation repertoires and affective domains. Rather, as sadness and dysphoria are more commonly observed symptom in depression than anhedonia (Baji et al., 2009; Smith, Joiner, Pettit, Lewinsohn, & Schmidt, 2008), we believe that further inquiry into the link between adaptive strategies and PA may shed light on findings that suggest a weak and inconsistent association between adaptive emotion regulation repertoires and depression (Kovacs & Yaroslavsky, 2014; Kovacs et al., 2009, 2016; Yaroslavsky, Bylsma, Rottenberg, & Kovacs, 2013).

Of consequence, our results suggest that emotion regulation repertoires play a critical role in stress sensitization that is observed among those with sexual abuse histories. In particular, the consistent and pernicious effects of maladaptive repertoires on the relationship between stress and depression outcomes across reporting methods, along with their direct effects, signals their key role in depression risk (Aldao et al., 2010; Extremera & Rey, 2015). This observation is aligned with literature that ties maladaptive emotion regulation to concurrent and prospective depression symptom elevation (Aldao et al., 2010; Just & Alloy, 1997) and risk for incidents of depressive episodes (Nolen-Hoeksema, 2000) and their recurrence (Kovacs et al., 2009, 2016). These associations do not appreciably attenuate when the use of adaptive emotion regulation responses is statistically controlled.

The key mechanistic role of maladaptive repertoires is further bolstered by the limited and inconsistent predictive value of adaptive response repertoires that, though associated with depression severity (Aldao et al., 2010), are markedly weakened or drop below a level of significance in the presence of maladaptive responses. Our finding stress-potentiating effects of adaptive emotion regulation repertoires on concurrently measured PA adds to the uncertain role of adaptive responses in depression risk. How may stress-sensitizing effects of adaptive emotion regulation repertoires be understood? It is feasible that the observed effect masks a higher-order interaction wherein adaptive responses are iatrogenic rather than salubrious for those with CSA. For example, results from laboratory studies show that some vulnerable groups (e.g., depressed participants) experience worsening moods after deploying a putatively adaptive response (e.g., Joormann, Siemer, & Gotlib, 2007). The same may be true for individuals with CSA. Future works that examine whether CSA moderates the effects of adaptive emotion regulation on affective outcomes would shed light on our findings.

Our results should be interpreted in the context of several limitations. There is evidence that sexual abuse during the childhood years can vary in intensity, frequency, source, and developmental timing, parameters that may affect stress sensitization, emotion regulation development, and depression risk. For example, age of onset, severity, source, and frequency of sexual abuse differentially predict difficulties in emotional (Clemmons, Walsh, Dilillo, & Messman-moore, 2007; Kaplow & Widom, 2007) and behavioral

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difficulties (Ruggiero, McLeer, & Dixon, 2000) across adulthood. Therefore, it is feasible that those whose abuse began early in life, was more severe, and chronic would evidence greater stress sensitization and emotion regulation deficits than those who experienced a single instance of abuse in late adolescence. Though detailed accounts of trauma were collected during the clinical interview, some participants were circumspect in their disclosure, thereby precluding an examination of several important CSA parameters in this study. Further, as those with CSA frequently experience other forms of abuse (Finkelhor, Turner, Shattuck, & Hamby, 2015), it is feasible that accompanying histories of physical violence and neglect may have contributed to our results.

There is also some evidence that gender differences exist in the long-term consequences of CSA (Gray & Rarick, 2018), and in the stress response across physiological (Kudielka & Kirschbaum, 2005) and subjective levels (Kelly, Tyrka, Anderson, Price, & Carpenter, 2008). Although our analyses controlled for gender, it is feasible that the relationship between CSA, emotion regulation, stress sensitization, and depression-related outcomes may vary between men and women, a possibility that we could not examine due to our small sample size of participants reporting CSA. Relatedly, emotion regulation outcomes are known to be contextual (Aldao, Sheppes, & Gross, 2015) and it is feasible that measuring state emotion regulation responses during EMA would have provided greater insights into the relationship between CSA and stress sensitization.

Although we proposed a sequential relationship between emotion regulation deficits and stress sensitization, a reverse effect is possible; CSA stress sensitization may lead to emotion regulation deficits by undermining normative emotion regulation development. We did not test this possibility because emotion regulation repertoires appear to slow in their development by mid-adolescence (see Kovacs et al., 2019), and we used an adult sample in this study.

Finally, though not uncommon in EMA studies, our single-item measure of stress may have contributed to greater measurement error than what could be expected from the use of a longer survey. Future works that via longitudinal designs examine the effects of CSA parameters, co-occurring physical abuse and neglect histories, contextual effects of gender on emotion regulation across development, and measure stress during experience sampling via multiple items would do much to clarify the mechanisms through which CSA confers risk for depressive disorders.

Nevertheless, this study has a number of strengths. First, the use of mixed methodology enabled us to test concurrent and prospective effects of stress sensitization in the daily lives of adults with histories of CSA as they encountered ideographic stressors, which increased the generalizability of our findings beyond that offered through cross-sectional designs that are commonly seen in the literature. Second, by disambiguating daily life stress into its stable and time-varying components, we were able to show distinct ties between emotion regulation and stress sensitization, in particular, that stress sensitization emerges in response to perturbations in stress levels rather than the absolute stress magnitudes. Finally, to our knowledge, this study is the first to test the roles of putative adaptive and maladaptive emotion regulation response repertoires in stress sensitization among those with histories of CSA.

Conclusion

In summary, our results have methodological and clinical implications as they point to emotion regulation deficits as key mechanisms for stress sensitization and depression-related outcomes, as well as a need to measure stress as a dynamic process that may precipitate depressive states differently depending on the nature of said deficits. Further, our findings may generalize to other disorders as CSA, stress sensitization, and emotion regulation deficits are transdiagnostic risk factors. This, in particular, may be the case for PTSD, which is associated with all three risk factors and depression (Chang et al., 2018; John, Cisler, & Sigel, 2017; McLaughlin, Conron, Koenen, & Gilman, 2010). Experience sampling methods may therefore provide clinicians with an inexpensive means to detect and target specific emotion regulation deficits that likely differ across those with CSA. Such innovations may prove efficacious for treating PTSD and other emotional disorders that are associated with CSA.

Supplementary Material. The supplementary material for this article can be found at https://doi.org/10.1017/S095457942000098X.

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