

Emotion Regulation in Adults with a History of Child Abuse Following PTSD Treatment

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Abstract

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Objective: The findings that adults with a history of childhood abuse (CA) have greater emotion regulation difficulties than adult trauma survivors without a history of CA (e.g., Cloitre, Scarvalone, & Difede, 1997) has led to the hypothesis that existing PTSD treatments may not be adequate for improving the emotion regulation deficits of adults with a history of CA (Cloitre et al., 2010). This study compared changes in emotion regulation over the course of PTSD treatment with either prolonged exposure (PE) therapy or sertraline in adults with and without a history of CA.

Method: Two hundred adults with PTSD received 10 weeks of either PE or sertraline. Emotion regulation and trait affect were assessed pre- and post-treatment with the Emotion Regulation Questionnaire (Gross & John, 2003), the Negative Mood Regulation Scale (Catanzaro & Mearns, 1990), and the Positive and Negative Affect Scale (Watson, Clark, & Tellegen, 1988).

Results: Individuals with a history of CA did not differ from individuals without a history of CA

at pre-treatment on PTSD severity, emotion regulation, or positive/negative affect. In addition, treatment was effective at improving emotion regulation and trait affect in those with and without a history of CA, and no significant differences in emotion regulation or trait affect emerged at post-treatment between adults with and without a history of CA. Furthermore, non-inferiority analyses indicated that the emotion regulation and trait affect outcomes of those with a history of CA were not inferior to the emotion regulation and trait affect outcomes of those without a history of CA.

Conclusion: Contrary to clinical lore regarding clients presenting for PTSD treatment, the current findings suggest that those with a history of CA do not differ from those without a history of CA in terms of emotion regulation and trait affect. Further, these findings cast doubt on the assumption that CA predicts worse emotion regulation outcomes following PTSD treatment.

Keywords: child abuse, emotion regulation, posttraumatic stress disorder, prolonged exposure, sertraline

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Emotion Regulation in Adult Survivors of Child Abuse Following PTSD Treatment

Posttraumatic stress disorder (PTSD) is a severe and debilitating disorder that develops in some individuals following exposure to a traumatic life event. Although selective serotonin reuptake inhibitors (SSRIs) and cognitive behavioral therapies, including prolonged exposure (PE) therapy, have been found to be efficacious in the treatment of chronic PTSD (e.g., Davis, Frazier, Williford, & Newell, 2006; Powers, Halpern, Ferenschak, Gillihan, & Foa, 2010), these treatments do not work with all clients. In fact, some claim that exposure therapy is inadequate for addressing the complex sequelae of childhood abuse (CA; e.g., Cloitre, Koenen, Cohen, & Han, 2002), including childhood sexual abuse, physical abuse, or both (Goldberg, Cloitre, Whiteside, & Han, 2003; Levitt & Cloitre, 2005; Stovall-McClough & Cloitre, 2006). One of the key problems associated with CA is impaired emotion regulation (e.g., Cloitre, Miranda, Stovall-McClough, & Han, 2005), which is the process that allows individuals to “influence which emotions they have, when they have them, and how they experience and express these emotions” (Gross, 1998, p. 275). For many with a history of CA, these emotion regulation deficits include difficulty recognizing and identifying emotions, managing emotional arousal, calming down, and letting go of distressing affective states (Roth, Newman, Pelcovitz, van der Kolk, & Mandel, 1997; Cloitre et al., 2005; Shipman et al., 2007).

Explanations for this association between CA and impairments in emotion regulation come from the developmental, behavioral neuroscience, and psychopathology literatures. Research from these areas suggests that there may be critical periods during childhood for learning how to properly respond to emotional stress (see Sánchez, Ladd, & Plotsky, 2001; Cicchetti & Toth, 1995, for reviews). Further, early life stress may be associated with neurobiological changes that may lead to later emotion dysregulation (see Heim & Nemeroff, 2001, for a review). Notably, pre-clinical research with animals has been instrumental in

furthering understanding of the effects of early stress on emotion regulation. For example, in rodents, postnatal stress at two-weeks, but not three- or 10-weeks of age, has been shown to alter emotional responses to stress exhibited later in life (Matsumoto et al., 2005). These findings have led to the hypothesis that emotion regulation skills are acquired in humans initially during infancy and the preschool years (Widom, Kahn, Kaplow, Sepulveda-Kozakowski, & Wilson, 2007) and that CA during these early years may affect children's abilities to engage the cortex in regulating the responses of other areas of the brain to emotional or stressful stimuli (e.g., Bremner, 2002). Moreover, it has been posited that CA also interferes with key learning processes that are necessary for achieving the developmental task of learning to regulate one's emotions (van der Kolk, 2003).

These disturbances in emotion regulation are often observed in adult survivors of CA (Roth et al., 1997; Cloitre et al., 2005; Frewen, Dozois, Neufeld, & Lanius, 2012), suggesting that CA may have long-lasting effects on emotion regulation abilities. Further, adults with a history of CA have been found to have moderately to substantially greater emotion regulation difficulties than those of individuals who have experienced trauma only in adulthood (e.g., Kulkarni, Pole, & Timko, 2012; Ehring & Quack, 2010; Cloitre et al., 1997). Although Ehring and colleagues also found that these differences between those with and without a history of CA became non-significant after controlling for PTSD severity, it has been posited that the symptoms of adults with PTSD and a history of CA extend beyond the symptoms of PTSD and may include severe disturbances in emotion regulation (e.g., Ford, Courtois, Steele, van der Hart, & Nijenhuis, 2005). These disturbances may serve as critical barriers to the successful implementation of existing PTSD treatments (Zlotnick et al., 1997).

Given that PTSD treatment often encourages individuals to address disturbing emotions

related to traumatic memories, some assume that a basic foundation of emotion regulation skills must be present in order to benefit from these treatments (Cloitre et al., 2002). Indeed, treatment guidelines have been developed for treating complex PTSD resulting from CA and other chronic forms of trauma (Cloitre et al., 2011). These guidelines build off of the concerns of some that traditional forms of exposure therapy with survivors of CA might overwhelm these emotionally unstable individuals, resulting in unsatisfactory emotion regulation outcomes (e.g., Zlotnick et al., 1997; Cloitre, Stovall-McClough, & Levitt, 2004; Ford et al., 2005). Consistent with this hypothesis is the finding that individuals with a history of CA, specifically child sexual abuse (CSA), have more PTSD symptoms following treatment with exposure therapy than do those without a history of CSA (Hembree, Street, Riggs, & Foa, 2004).

Three randomized controlled trials have shown that the addition of an emotion regulation skills component to exposure therapy for PTSD is effective at reducing PTSD symptoms and other difficulties in women with a history of CA. In the first two studies, Cloitre and colleagues found that a phase-based treatment consisting of both skills training in affect and interpersonal regulation (STAIR) and a modified form of prolonged exposure therapy (MPE) was more effective at improving PTSD symptoms and negative mood regulation than no treatment (Cloitre et al., 2002) and than both supportive counseling (SC) followed by MPE and STAIR followed by SC (Cloitre et al., 2010). In the third study, Steil and colleagues found that a residential form of dialectical behavior therapy for PTSD that included group skills training in mindfulness and in managing trauma-related emotions, as well as individual exposure treatment, was effective at reducing PTSD symptoms, trait anxiety, and depression (Steil, Dyer, Priebe, Kleindienst, & Bohust, 2011).

However, although promising results have been obtained in these trials, it remains

unclear whether phase-based treatments contribute to treatment effects on emotion regulation above and beyond those of exposure therapy (Cahill, Zoellner, Feeny, & Riggs, 2004; Ehring & Quack, 2010), as these treatments have yet to be compared to a full course of ten sessions of prolonged exposure therapy involving both imaginal and *in vivo* exposures. Furthermore, it is not clear that CA predicts worse outcome at post-treatment in the absence of additional targeted emotion regulation interventions. Specifically, Resick and colleagues found that women with a history of CSA fared as well as women without a history of CSA in terms of PTSD and other trauma reactions, including anxious arousal and anger-irritability, following treatment with either cognitive-processing therapy or prolonged exposure therapy (Resick, Nishith, & Griffin, 2003). Similarly, McDonagh and colleagues found that, among women with PTSD and a history of CSA, those treated with cognitive behavioral therapy (CBT) were as likely as those treated with a problem-solving therapy tailored specifically for survivors of CSA to no longer meet criteria for PTSD following treatment (McDonagh et al., 2005). Although the dropout rate for CBT was higher than for problem-solving therapy, those in CBT were also less likely to have a diagnosis of PTSD at follow-up assessments than were those treated with problem-solving therapy. Additionally, individuals with PTSD secondary to CA fared just as well as individuals without a history of CA in treatment with sertraline, showing marked improvement in PTSD symptom severity at post-treatment (Stein, van der Kolk, Austin, Fayyad, & Clary, 2006). Thus, these results suggest that non phase-based PTSD treatments may be effective in treating PTSD in adults with a history of CA.

Remarkably, however, no studies to date have examined whether PTSD treatment with CBT or selective serotonin reuptake inhibitors (SSRIs) can improve the emotion regulation difficulties of adults with a history of CA. From a theoretical perspective, both treatments may

be adequate for addressing the PTSD symptoms and emotion regulation difficulties of these individuals. The prolonged exposure (PE) therapy protocol outlined by Foa, Hembree, and Dancu (2002) directly and indirectly addresses many facets of emotion regulation, equipping patients with tools that might help them deal with intense emotions. Moreover, PE allows patients to experience *in vivo* that intense emotions associated with anxiety do not last indefinitely, but eventually subside, potentially teaching distress tolerance skills and facilitating inhibitory learning (Craske et al., 2008). Similarly, sertraline may improve emotion regulation by acting through various neural pathways and brain structures to broadly enhance mood and reduce anxiety (e.g., Schoenfeld, Marmar & Neylan, 2004; Quidé, Witteveen, El-Hage, Veltman, & Olff, 2012). Sertraline has been found to improve emotion regulation (Davidson, Landerman, Farfel, & Clary, 2002; Simmons & Allen, 2011) and therefore may be an effective treatment for individuals suffering from both PTSD and emotion regulation difficulties.

The present study compared patients with and without a history of CA on key indices of emotion regulation and trait affect before and after receiving PTSD treatment with either PE or sertraline. Notably, we took a multi-faceted approach to our examination of emotion regulation, assessing general emotion regulation and specific negative mood regulation, as well as positive and negative affect, which we assumed would co-vary with emotion regulation. First, if CA impairs emotion regulation, then those with CA should have greater emotion regulation difficulties and worse trait affect (i.e., lower positive affect, higher negative affect) than those without CA at pre-treatment. However, if CA does not impair emotion regulation, then those with and without a history of CA should be similar in emotion regulation and trait affect at pre-treatment. Second, if CA interferes with PTSD treatment, then those with CA should have worse emotion regulation and trait affect outcomes than those without CA. Alternatively, if CA does

not interfere with PTSD treatment, then post-treatment emotion regulation and trait affect outcomes of those with CA should not be worse than the post-treatment emotion regulation and trait affect outcomes of those without CA.

Method

Participants

Two hundred participants were recruited from two large, metropolitan communities via referrals from providers in these communities and through advertising in buses, newspapers, and flyers placed in community centers, churches, grocery stores, convenience shops and libraries, and on campus message boards. Eligible participants were English-speaking men (24.5%, $n = 49$) and women (75.5%, $n = 151$) between the ages of 18-65 with a primary *DSM-IV* diagnosis of chronic PTSD related to a trauma that occurred at least three months prior to the initial evaluation. Exclusion criteria included: a current diagnosis of schizophrenia or delusional disorder; medically unstable bipolar disorder, depression with psychotic features, or depression requiring immediate psychiatric treatment; a diagnosis of alcohol or substance abuse within the previous three months; an ongoing intimate relationship with the perpetrator (in cases of sexual or physical assault); an unwillingness to discontinue current antidepressant medication or psychotherapy; current use of sertraline; a previous, failed trial of either PE or sertraline; or a medical contraindication for taking sertraline, such as pregnancy or lactation.

A total of 426 individuals were screened for eligibility, of whom 172 were ineligible and 54 were eligible but not interested in participating further in the study. The final sample thus consisted of 200 participants and was primarily Caucasian (65.5%), female (75.5%) and not college educated (70%). The index trauma reported by participants included sexual assault (31%), non-sexual assault (22.5%), childhood sexual abuse (17.5%), childhood non-sexual abuse

(6.5%), a motor vehicle accident or some other kind of accident (13.5%), having a loved one die or be exposed to violence (6.5%), combat or war (2.5%)

Although a minority of participants reported CA as their index trauma (24%), 65% ($n = 170$) of the participants were survivors of CA, defined as either child sexual abuse or child physical abuse. Child sexual abuse was defined as one or more episodes of unwanted sexual contact (hand to genital or genital to genital contact) prior to the age of 13 by an individual five or more years older than the client. Child physical abuse was defined as one or more episodes prior to the age of 13 in which physical contact by an individual at least five years older than the client resulted in bruises or marks. For the full sample, the mean time since trauma was 11.97 years ($SD = 12.69$) and the mean number of traumatic events participants reported experiencing over the course of their lives was 9.05 ($SD = 6.23$).

Measures

PTSD Symptom Scale-Interview Version (PSS-I; Foa et al., 1993). The PSS-I is an interviewer-administered instrument that assesses the severity of the 17 *DSM-IV* PTSD symptoms on a 0 (*not at all*) to 3 (*5 or more times per week/very much*) scale. The PSS-I has good inter-rater reliability (diagnosis: $r = .91$; symptom severity: $r = .97$) and test-retest reliability ($r = .80$; Foa & Tolin, 2000). In the current study, diagnostic reliability was examined by rerating over 10% of the cases. Inter-rater reliability was excellent ($ICC = .98$).

Structured Clinical Interview for *DSM-IV* Axis I Disorders with Psychotic Screen (SCID-IV; First et al., 1995). The SCID-IV is a semi-structured clinical interview that was used in the present study to assess for exclusion criteria and to assess for comorbid disorders. Over 10% of the cases from the current study were rerated for diagnostic reliability. There was good diagnostic agreement for current anxiety disorders ($\kappa = 1.00$, $p_{pos} = 1.00$, $p_{neg} = 1.00$), major

depressive disorder ($\kappa = .68$, $p_{pos} = .88$, $p_{neg} = .80$), substance abuse disorders ($p_{pos} = .00$, $p_{neg} = 1.00$), and other diagnoses ($p_{pos} = .00$, $p_{neg} = 1.00$).

Emotion Regulation Questionnaire (ERQ; Gross & John, 2003). The ERQ is a ten-item self-report measure of both cognitive reappraisal (i.e., cognitive attempts to dampen the emotional impact of situations) and expressive suppression (i.e., attempts to inhibit the expression of emotions). Items from both scales are assessed using 7-point Likert scales with scores ranging from 1 (*strongly disagree*) to 7 (*strongly agree*), with the mean score of the items on each scale being used as an indicator of reappraisal and suppression, respectively. Higher reappraisal is associated with more adaptive functioning, whereas higher suppression is associated with less adaptive functioning (Gross & John, 2003). The two subscales are only weakly correlated ($r = -.01$) and test-retest reliability is .69 for both scales (Gross & John, 2003).

Negative Mood Regulation (NMR; Catanzaro & Mearns, 1990). The NMR is a self-report measure that assesses beliefs about one's ability to effectively improve negative mood states. The inventory consists of 30 items scored on a 5-point Likert scale. Scores range from 1 (*strong disagreement*) to 5 (*strong agreement*), with higher scores reflecting better self-efficacy beliefs. The measure has both good internal consistency (.86 - .90) and temporal stability (.67 - .78; Catanzaro & Mearns, 1990).

Positive and Negative Affect Schedule (PANAS; Watson, Clark, & Tellegen, 1988). The PANAS is a measure that consists of both positive affect (PA) and negative affect (NA) scales. In the present study, the trait version of the PANAS was used. The PA scale assesses the degree to which one experiences pleasant mood states such as excitement, inspiration, and pride; whereas, the NA scale assesses the extent to which one experiences unpleasant mood states such as nervousness, fear, anger, guilt, contempt and disgust. Each of the subscales contains 10 items,

and items are assessed using a 5-point Likert scale ranging from 1 (*slightly or not at all*) to 5 (*extremely*). Test-retest reliability is .68 for PA and .71 for NA (Watson et al., 1988).

Treatment: Prolonged Exposure (PE)

Treatment with PE followed a standardized treatment manual, consistent with Foa and Rothbaum (1998), and consisted of 10 weekly, 90-120 min sessions. Procedures included the following: education about common reactions to trauma; breathing retraining; repeated *in vivo* exposure; repeated imaginal exposure to the client's trauma memory; processing of the trauma memory; and *in vivo* and imaginal exposure homework, which was delivered at the end of each session. All PE therapists were masters or PhD level clinicians who received standardized training in the delivery of PE. Weekly supervision occurred and included case discussions and video or audiotape review of the treatment sessions. Trained raters reviewed 10% of the session videotapes, providing integrity ratings, as well as PE therapist competence ratings (e.g., engaged in interactive exchange with the client) using a 3-point scale (1 = *Inadequate*, 3 = *Adequate or Better*). PE therapists completed 85% of the essential components of PE and no protocol violations were observed. Additionally, overall PE therapist competence ratings were very good ($M = 2.71$, $SD = 0.56$).

Treatment: Sertraline

Pharmacotherapy with sertraline consisted of 10 weekly medication management sessions with board certified psychiatrists who were experienced in the treatment of anxiety disorders. Based on a treatment manual (Marshall, Beebe, Oldham, & Zaninelli, 2001), sessions lasted up to 30 min, with the first lasting up to 45 min. No exposure or anti-exposure instructions were given. Dosage started at 25 mg/day and if indicated and tolerated, increased to the goal of 200 mg/day, using a standard titration algorithm (Brady et al., 2000). Final average dosage was

115 mg/day ($SD = 78.00$). Medication adherence was documented with pill counts and medication diaries, and all sessions were recorded. Trained raters reviewed 10% of the videotapes, providing integrity ratings using the same scale as was used by Marshall et al. (2001). For essential components, psychiatrists completed 96% and no protocol violations were observed.

Procedures

After completing informed consent procedures, potential participants were interviewed by trained raters using the SCID-IV and the PSS-I. Eligible participants completed self-report questionnaires including the ERQ, NMR, and PANAS prior to randomization and then began acute treatment with either PE or sertraline. After treatment, participants again completed emotion regulation and trait affect self-report measures (ERQ, NMR, and PANAS).

Data Analytic Strategy

Statistical analyses were conducted using SPSS Statistics version 19.0 (SPSS inc., Chicago). Pre-treatment continuous variables were analyzed using independent samples t-tests. To examine whether CA moderates the effects of treatment (PE vs SER) and time (pre-, post-treatment) on emotion regulation (ERQ, NMR) and trait affect (PANAS), linear mixed effects models were used with a random intercept model, which provided the best fit for the data. Missing data was handled using Restricted Maximum Likelihood (REML).

All non-significant findings were followed up with non-inferiority analyses (Pocock, 2003), which allowed us to examine whether the emotion regulation and trait affect outcomes of those with a history of CA were clinically non-inferior to the emotion regulation and trait affect outcomes of those without a history of CA (Wellek, 2010). Non-inferiority margins (Wellek, 2010) were determined using the standard deviations of healthy samples, providing a

conservative benchmark for healthy post-treatment responding. For the ERQ reappraisal and suppression scales, the non-inferiority margins were set at $[-\infty, 1]$ and $[-1.16, \infty]$, respectively. These values were derived by weighting the standard deviations by gender of the healthy sample (Gross & John, 2003). The non-inferiority margins were similarly set for the NMR $[-\infty, 14.33]$, positive affect $[-\infty, 6.40]$, and negative affect $[-5.90, \infty]$ (Catanzaro & Mearns, 1990; Watson et al., 1988). One-sided 95% confidence intervals, $\alpha = .05$, were calculated for the difference between the emotion regulation outcomes for no CA and CA at post-treatment. For cognitive reappraisal, negative mood regulation, and positive affect, positive values would indicate that those with a history of CA had worse outcomes than those without a history of CA. Conversely, for expressive suppression and negative affect, negative values would indicate that those with a history of CA had worse outcomes than those without a history of CA.

Results

Pre-treatment Differences on Childhood Abuse

We first examined whether those with CA differed from those without CA in terms of baseline severity, as measured by the PSS-I, ERQ, NMR, and PANAS. Individuals with a history of CA ($M = 29.76$, $SD = 6.73$) did not differ from those without a history of CA ($M = 29.21$, $SD = 6.64$) in terms of baseline PTSD severity; $t(198) = -0.55$, $p = .62$. As seen in Table 1, there also were no differences between groups on emotion regulation, as assessed by cognitive reappraisal, $t(192) = 0.09$, $p = .37$, and expressive suppression, $t(192) = -0.57$, $p = .63$, and negative mood regulation, $t(169) = -1.12$, $p = .52$. In addition, there were no differences between groups on trait affect, as assessed by positive affect, $t(191) = -1.70$, $p = .75$, and negative affect, $t(191) = 0.05$, $p = .64$. Thus, there were no pre-treatment differences between those with and without a history of CA on PTSD severity, emotion regulation, or trait affect. See Table 1 for

pre-treatment emotion regulation and trait affect means, as well as for effect sizes of the pre-treatment differences between those with and without a history of CA.¹

Childhood Abuse as a Moderator of Time and Treatment Outcome

Next, to test the hypothesis that CA moderates treatment outcome, we used multilevel modeling to examine the two-way interaction between CA (present vs absent) and Time (pre- vs post-treatment). There was no CA x Time interaction for emotion regulation, as assessed by cognitive reappraisal, $F(1, 166.149) = 1.94, p = .17$, expressive suppression, $F(1, 163.92) = 0.85, p = .36$, and negative mood regulation, $F(1, 143.37) = 0.20, p = .66$. Further, there was no CA x Time interaction for trait affect, as assessed by positive affect, $F(1, 170) = 0.20, p = .65$, and negative affect, $F(1, 166.97) = 0.02, p = .89$. Thus, CA did not moderate the effect of time on emotion regulation or trait affect.² See Table 1 for post-treatment emotion regulation and trait

¹ We re-ran these analyses for individuals with and without an index trauma of CA. Here, an index trauma refers to the traumatic event from which the individual's current PTSD symptoms developed. The results from these analyses did not differ from the main analyses. Individuals with an index trauma of CA ($M = 29.15, SD = 6.99$) did not differ significantly from those without an index trauma of CA ($M = 29.69, SD = 6.61$) in terms of baseline PTSD severity; $t(198) = 0.48, p = .63$. Moreover, individuals with an index trauma of CA ($M = 4.36, SD = 1.17$) did not differ from individuals without an index trauma of CA ($M = 4.31, SD = 1.17$) in terms of baseline cognitive reappraisal, $t(192) = 0.81, p = .81$. Individuals with an index trauma of CA ($M = 4.22, SD = 1.22$) also did not differ from individuals without an index trauma of CA ($M = 4.03, SD = 1.33$) in terms of expressive suppression, $t(192) = -0.81, p = 0.42$. There was also no difference between individuals with an index trauma of CA ($M = 92.86, SD = 14.51$) and individuals without an index trauma of CA ($M = 95.49, SD = 19.82$), in terms of negative mood regulation, $t(169) = 0.75, p = .45$. Similarly, individuals with an index trauma of CA ($M = 33.14, SD = 8.28$) did not differ from individuals without an index trauma of CA ($M = 30.80, SD = 7.86$) in terms of positive affect, $t(191) = -1.70, p = .09$. Further, individuals with an index trauma of CA ($M = 31.33, SD = 8.48$) did not differ from individuals without an index trauma of CA ($M = 29.08, SD = 8.49$), in terms of negative affect, $t(191) = 0.05, p = .13$.

² We also re-ran the linear mixed effect models for individuals with and without an index trauma of CA. An index trauma of CA did not significantly affect the relationship between time from pre- to post-treatment and emotion regulation and trait affect outcomes, as assessed by cognitive reappraisal, $F(1, 164.62) = 0.24, p = .63$, expressive suppression, $F(1, 162.48) = 0.58, p = .45$, negative mood regulation, $F(1, 153.82) = 0.21, p = .65$, positive affect, $F(1, 166.09) = 0.21, p = .65$, and negative affect, $F(1, 160.85) = 0.29, p = .59$.

affect effect sizes comparing No CA to CA.

To examine whether CA moderated the effect of either PE or sertraline over time, we examined the three-way interaction among CA (CA vs no CA), treatment (PE vs sertraline), and time (pre- vs post-treatment), on emotion regulation and trait affect. There were no three-way interactions for cognitive reappraisal, $F(1, 166.15) = 1.44, p = .23$, expressive suppression, $F(1, 163.92) = 0.00, p = .99$, or negative mood regulation, $F(1, 143.37) = 0.80, p = .37$. Further, there were no three-way interactions for positive affect, $F(1, 170.00) = 0.10, p = .75$, or negative affect, $F(1, 166.97) = 0.90, p = .34$. Thus, CA did not moderate the emotion regulation or trait affect treatment outcomes following treatment with either PE or sertraline.³

However, as seen in Table 1, the pre- to post-treatment effect sizes were moderate to large for those with a history of CA and small to large for those without a history of CA. Thus emotion regulation and trait affect improved over the course of treatment for individuals with and without a history of CA.

Non-inferiority of Childhood Abuse at Post-Treatment

Given that CA did not moderate change in post-treatment emotion regulation or trait affect according to conventional superiority tests, we further examined whether the emotion regulation outcomes of those with a history of CA were non-inferior to the outcomes of those without a history of CA. That is, were the emotion regulation and trait affect outcomes of individuals with a history of CA no worse than those of individuals without a history of CA at

³ An index trauma of CA also did not moderate the effect of PE or sertraline as assessed by a three-way interaction among treatment (PE vs sertraline), time (pre- to post-treatment) and index trauma (CA or no CA) on emotion regulation or trait affect. There were no three way interactions for cognitive reappraisal, $F(1, 164.62) = 1.42, p = 0.24$, expressive suppression, $F(1, 162.48) = 0.02, p = .90$, negative mood regulation, $F(1, 153.82) = 1.11, p = .29$, or positive affect, $F(1, 166.09) = 2.20, p = .14$. However, there was a three-way interaction for negative affect, $F(1, 160.85) = 3.92, p = .049$.

post-treatment? To do so, we examined the one-sided, 95% confidence intervals of the difference between the emotion regulation and trait affect outcomes for No CA and CA at post-treatment. As seen in Figure 1, which depicts the mean difference between No CA and CA, as well as the 95% confidence intervals and the non-inferiority margins for cognitive reappraisal, expressive suppression, negative mood regulation, positive affect, and negative affect, the emotion regulation and trait affect outcomes for individuals with a history of CA were not inferior to those of individuals without a history of CA. More specifically, the confidence values fell within the pre-set, non-inferiority margins for reappraisal, 95% CI $[-\infty, 1.22]$, suppression, 95% CI $[-0.382, \infty]$, negative mood regulation, 95% CI $[-\infty, 2.681]$, positive affect, 95% CI $[-\infty, 1.380]$, and negative affect, 95% CI $[-3.689, \infty]$. Thus, across all measures, individuals with CA were not worse in emotion regulation or trait affect at post-treatment than individuals without a history of CA.

Discussion

The present study examined emotion regulation and trait affect in individuals with and without a history of CA before and after PTSD treatment with either PE or sertraline. There were no baseline differences in PTSD severity, emotion regulation, or trait affect between those with and without a history of CA. Further, emotion regulation and trait affect both improved from pre- to post-treatment for those with and without a history of CA, and the post-treatment emotion regulation and trait affect outcomes of those with a history of CA were not worse than those without a history of CA. Notably, these findings were seen across measures of general and negative mood emotion regulation, as well as measures of positive and negative trait affect. Thus, contrary to clinical lore, emotion regulation and trait affect improve over the course of standard, evidenced-based PTSD treatment, regardless of whether clients have a history of CA or

not.

The current study is novel in that it is, to our knowledge, the first to examine whether individuals with and without a history of CA differ in emotion regulation and trait affect before and after receiving treatment for PTSD. Although prior studies have focused on the effects of CA on post-treatment PTSD symptoms rather than on emotion regulation and trait affect, the results of the present study fit with previous findings suggesting that clients with and without a history of CA benefit similarly from treatment with either CBT or SSRIs (e.g., Resick et al., 2003; Stein et al., 2006). Further, the results of this study are consistent with previous findings suggesting that CA is not a reliable predictor of poorer PTSD treatment outcome (van Minnen, Arntz, & Keijsers, 2002; Karatzias et al. 2007). Accordingly, the current study's findings lend support to the notion that CA should not be viewed as a contraindication for exposure therapy (Cahill et al., 2004; Hembree & Brinen, 2009).

Conversely, the findings of the present study contrast with previous research suggesting that those with a history of CA have elevated PTSD symptoms and greater emotion regulation impairments compared to trauma survivors without a history of CA. However, unlike previous studies, which examined PTSD symptoms and emotion regulation in trauma-exposed individuals who may or may not have had PTSD (Cloitre et al., 1997; Ehring & Quack, 2010; Kulkarni et al., 2012), the current study's findings are based on a sample of individuals who had a primary diagnosis of PTSD and who were seeking treatment for their PTSD symptoms. In addition, although the current findings are inconsistent with the finding that CSA predicts worse PTSD outcomes following treatment with PE (Hembree et al., 2004), this discrepancy in findings may be a reflection of the different methods used in the previous study and in our study. More specifically, given that Hembree and colleagues assessed PTSD rather than emotion regulation

and collapsed the treatment outcomes of those who received PE, stress inoculation training (SIT), and PE + SIT, it is unclear from their findings whether CA actually predicts worse outcomes following PE.

In addition, the moderate to large pre- to post-treatment effect sizes found in the present study for those with a history of CA contradict the hypothesis that existing PTSD treatments are insufficient for improving emotion regulation in individuals with a history of CA (e.g., Cloitre et al., 2004). Indeed, it is likely that we would have seen even greater changes in emotion regulation had we specifically selected for individuals with baseline deficits in emotion regulation. Relatedly, the study's main findings call into question claims that PTSD treatment for adults with a history of CA must explicitly include targeted emotion regulation interventions (e.g., Ford et al., 2005; Cloitre et al., 2011). After all, PE and SSRIs lead to positive changes in emotion regulation and trait affect, even though these treatments do not necessarily target either emotion regulation or trait affect. Furthermore, although it is possible that additional interventions could augment standard, evidenced-based PTSD treatment for individuals with a history of CA, there is no data to suggest that the addition of targeted emotion regulation interventions to PTSD treatment results in outcomes that are superior to those of stand alone PTSD treatments. Indeed, the only trial that has compared an augmented treatment of this nature to an existing PTSD treatment used a modified version of PE that did not include a key component of the standard PE protocol, namely *in vivo* exposure. Additionally, given that the post-treatment negative mood regulation means from the present study for those with a history of CA were within one standard deviation of the means that Cloitre and colleagues obtained following their phase-based treatment for CA-related PTSD (Cloitre et al., 2002; 2010), it further raises doubts regarding whether individuals with a history of CA benefit more from an emotion

regulation skills augmented PTSD treatment than from standard PTSD treatments.

Nevertheless, several limitations should be noted. First, it is possible that the sample we selected of individuals with a history of CA was not representative of the general population of survivors of CA but instead was positively skewed in terms of pre-treatment emotion regulation and trait affect. However, this seems unlikely given that the pre-treatment NMR means of those in the current study were similar to the pre-treatment NMR means found in previous studies of individuals with a history of CA presenting for PTSD treatment (e.g., Cloitre et al., 2002; 2010). Second, given that a primary diagnosis of PTSD was one of our inclusion criteria, individuals with the most severe impairments in emotion regulation, such as those with a primary diagnosis of borderline personality disorder (BPD), were excluded from the study. However, the study's inclusion criteria were chosen in order to ensure that clients received the most appropriate clinical care given their presenting problems, as we believe that those with a primary diagnosis of BPD should be treated initially with treatment that targets BPD (Harned, Jackson, Comtois, & Linehan, 2010). Third, we assessed CA as a homogenous construct despite theoretical arguments that CA during certain critical, early periods of development may lead to greater disturbances in emotion regulation than CA during later developmental periods (Widom et al., 2007). However, we chose to define CA as is commonly defined in the current the literature (e.g., Cloitre et al., 2010). Finally, this study lacked a wait-list condition, making it difficult to rule out the possibility that the observed improvements in emotion regulation and trait affect were an artifact of time. Yet, this seems unlikely given that the mean time since participants' index trauma was over 11 years and that previous studies have failed to show improvements in PTSD symptoms over time alone (e.g., Rothbaum, Astin, & Marsteller, 2005; Stein et al., 2006).

The current findings are important in that they provide evidence that counters three

common assumptions in the literature: 1) that individuals with PTSD and a history of CA have greater emotion regulation deficits than those without CA; 2) that individuals with a history of CA fare worse than individuals without a history of CA in terms of emotion regulation following standard, evidenced-based PTSD treatments; and 3) that existing PTSD treatments must be augmented with interventions that explicitly target emotion regulation. Additionally, the findings have potential clinical implications for clients with a history of CA who cannot afford the time or cost of longer, phase-based treatments, as well as for practitioners who worry that clients with a history of CA are not appropriate candidates for shorter, evidenced-based PTSD treatments. Additional research is needed, however, before any conclusions can be made regarding the superiority, equivalence, or non-inferiority of phase-based treatments to standard PTSD treatment. Furthermore, future research should move away from assessing CA as a monolithic entity and should instead assess whether and how the effects of CA on emotion regulation vary depending on the age at which the abuse occurred as well as the frequency and chronicity of the abuse. Doing so will be crucial in enabling us to respond to calls for the mental health field to find the best treatment approaches for individual patients given their particular characteristics and circumstances (Insel, 2009).

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Table 1

Emotion Regulation at Pre- and Post-treatment and Mean Effect Size Difference between No CA and CA at Pre- and Post-Treatment

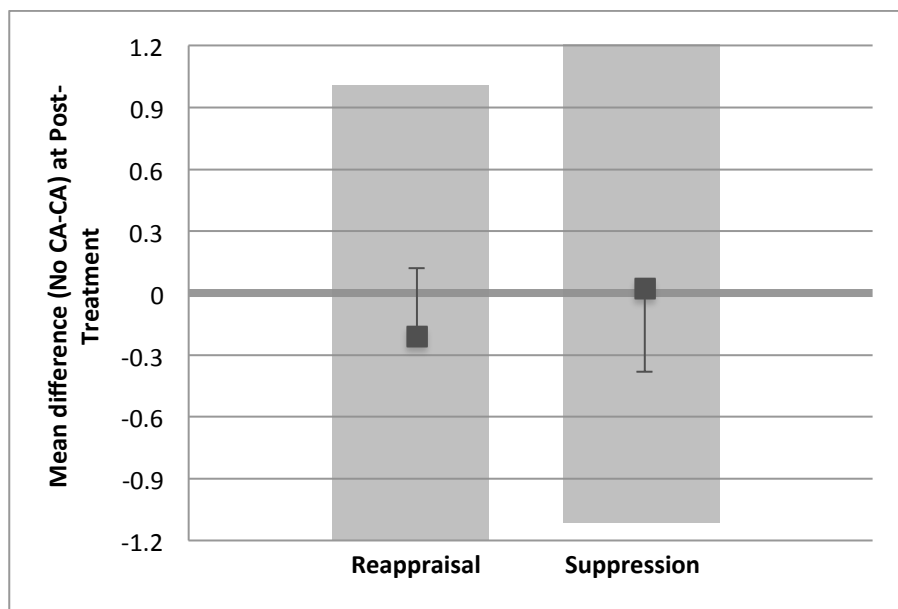
Measure	No CA (<i>n</i> = 70)					CA (<i>n</i> = 130)					No CA Minus CA		
	Pre-		Post-		Pre-Minus Post-	Pre-		Post-		Pre-Minus Post-	Post-	Pre-	Post-
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>Effect Size (d)</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>Effect Size (d)</i>	<i>M</i>	<i>Effect Size (d)</i>	<i>Effect Size (d)</i>
ERQ Rea	4.32	1.21	4.53	1.34	0.16	4.32	1.22	4.74	1.47	0.31	0.211	0.00	0.15
ERQ Sup	3.97	1.32	3.59	1.46	0.27	4.13	1.33	3.57	1.57	0.38	0.021	0.12	0.01
NMR	93.05	20.72	105.16	21.92	0.57	95.70	20.64	107.82	22.61	0.56	2.653	0.13	0.12
PA	30.07	8.26	35.22	9.35	0.58	32.09	8.40	36.14	9.83	0.44	0.914	0.24	0.10
NA	29.48	8.95	20.33	10.18	0.95	29.60	9.12	21.52	10.71	0.81	1.189	0.01	0.11

Note. ERQ Rea = Emotion Regulation Questionnaire, Cognitive Reappraisal Subscale; ERQ Sup = Emotion Regulation Questionnaire, Expressive Suppression Subscale; NMR = Negative Mood Regulation; PA = Positive and Negative Affect Scale, Positive Affect Subscale; NA = Positive and Negative Affect Scale, Negative Affect Subscale.

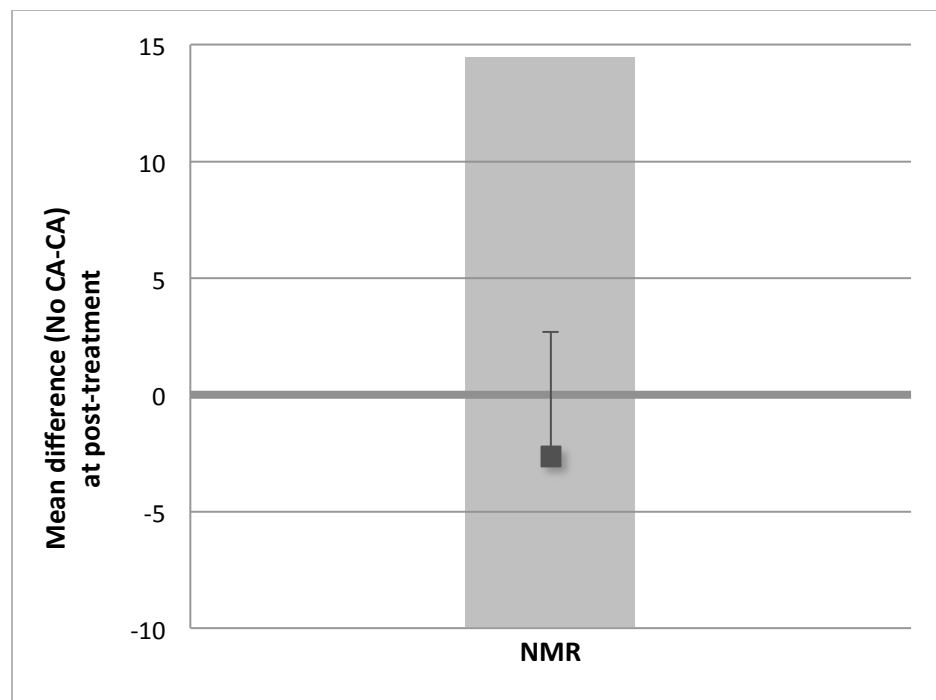
Figure 1

Non-inferiority Margins and One-sided 95% CIs for Differences in Outcome Between those with and without a history of CA

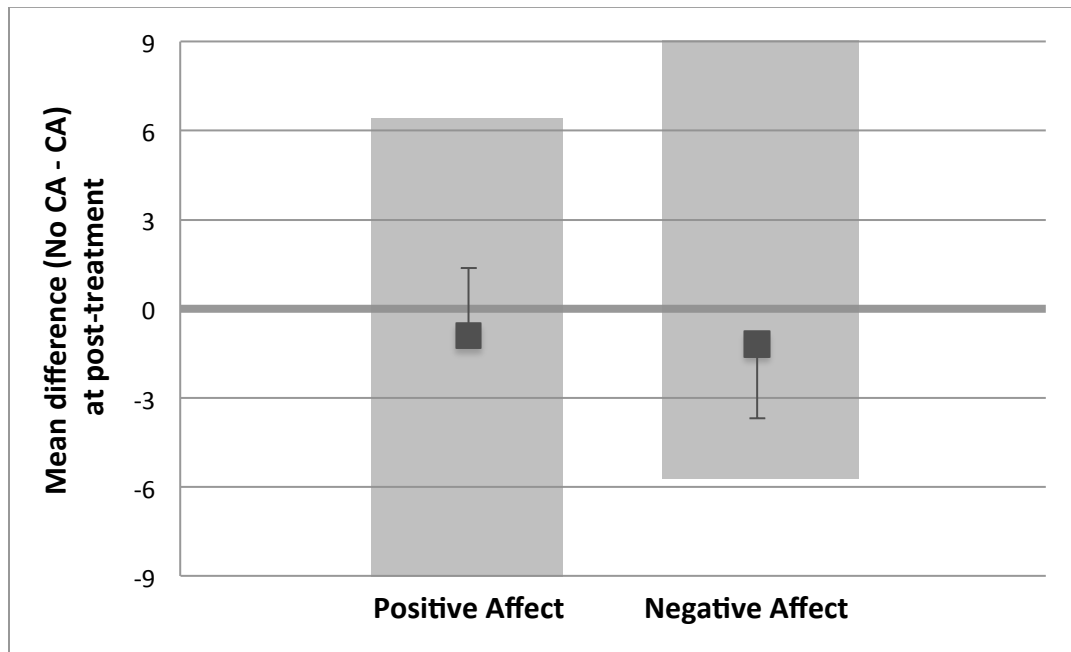
a)



b)



c)



Note. The line at zero represents where the mean difference (No CA-CA) would be if there were no difference between those with and without a history of CA at post-treatment. The error bars represent the 95% confidence interval of the mean difference and the shaded regions show the one-sided non-inferiority margins.