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The Effects of Prolonged Exposure and Sertraline on Emotion Regulation in Individuals with Posttraumatic Stress Disorder

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Abstract

The effects of current posttraumatic stress disorder (PTSD) interventions on emotion regulation are relatively unknown. Many conceptualize PTSD as a disorder of emotion dysregulation, and clinicians often fear that emotion regulation impairments will not change with stand-alone PTSD treatments, particularly for individuals with pre-existing emotion regulation difficulties. The present study examined changes in emotion regulation (expressive suppression, cognitive reappraisal, negative mood regulation) with prolonged exposure (PE) therapy or sertraline, specifically examining whether those with higher pre-existing emotion regulation difficulties improve over treatment on these indices. Individuals with chronic PTSD ($N = 200$) received 10 weeks of PE or sertraline and were followed through 6-month follow-up. Emotion regulation was assessed at pre- and post-treatment and at 3- and 6-month follow-up. Individuals with poorer initial emotion regulation showed greater improvement on all indices of emotion regulation, regardless of which treatment they received. Changes occurred during active treatment and were maintained over follow-up. These findings have both theoretical and clinical implications, arguing that emotion regulation is not impaired across all individuals with PTSD and that PE and sertraline effectively address emotion regulation difficulties.

Keywords

emotion regulation; PTSD; prolonged exposure; sertraline; treatment

Posttraumatic stress disorder (PTSD) is a chronic and impairing condition that can develop in the wake of trauma, with a U.S. lifetime prevalence rate of 6.8% (Kessler et al., 2005). One potential mechanism underlying the development and maintenance of PTSD is emotion regulation (e.g., Benoit, Bouthillier, Moss, Rousseau, & Brunet, 2010; Ehling & Quack, 2010; Frewen & Lanius, 2006; Weiss, Tull, Anestis, & Gratz, 2013; Weiss et al., 2012).

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Emotion regulation is defined as “the process by which individuals influence which emotions they have, when they have them, and how they experience and express these emotions” (p. 275, Gross, 1998). It is often considered a key component of mental health (Gross, 2001; Gross & Munoz, 1995). Impaired emotion regulation is thought to be present in most personality disorders and in most DSM-IV Axis I disorders (Gross & Levenson, 1997), including PTSD (e.g., Benoit et al., 2010; Cloitre, Miranda, Stovall-McClough, & Han, 2005; Eftekhari, Zoellner, & Vigil, 2009; Ehling & Quack, 2010; Frewen, Dozois, Neufeld, & Lanius, 2012; Kashdan, Breen, & Julian, 2010; Litz & Gray, 2002; McDermott et al., 2009; Moore, Zoellner, & Mollenholt, 2008; New et al., 2009; Tull, Barrett, McMillan, & Roemer, 2007; Weiss et al., 2012, 2013; Wisco, Sloan, & Marx, 2013).

In fact, it has been argued that PTSD is a disorder characterized primarily by emotion dysregulation (Frewen & Lanius, 2006). However, very few randomized control trials (RCTs) in the PTSD literature have examined emotion regulation outcomes and whether efficacious PTSD treatments without a separate, emotion regulation component can effectively improve emotion regulation. We are aware of only four PTSD RCTs that report emotion regulation outcomes and one RCT that examined the moderating role of childhood abuse on emotion regulation.

Hinton and colleagues compared emotion regulation-focused cognitive behavioral treatment (CBT) to a wait-list control in a small sample of Cambodian refugees with comorbid PTSD and orthostatic panic (Hinton, Hoffman, Pollack, & Otto, 2009). Emotion regulation, measured with a single questionnaire assessing the ability to distance oneself from painful emotions, improved over the course treatment. Cloitre and colleagues (2002) reported large improvements in emotion regulation difficulties using an emotion regulation-focused intervention plus imaginal exposure in comparison to a minimal attention waitlist (Cloitre, Koenen, Cohen, & Han, 2002). More recently, Cloitre and colleagues showed large improvement in emotion regulation across both an emotion regulation-focused intervention with imaginal exposure and a supportive counseling intervention with imaginal exposure (Cloitre et al., 2010). They also found marginally better negative mood regulation outcomes at 3- and 6-month follow-up for the individuals receiving the emotion regulation-focused intervention with imaginal exposure compared to individuals receiving the supportive counseling with imaginal exposure (Cloitre et al., 2010).

Additionally, in a small study reporting the use of a PTSD treatment not directly targeting emotion regulation, Wisco and colleagues (Wisco et al., 2013) found greater decreases in rumination for written exposure therapy (WET) compared to a waitlist control; however, these improvements occurred during follow-up rather than active treatment and no significant effects of WET were found on other cognitive strategies. Contrary to the potentially adaptive function of reappraisal, baseline positive reappraisal was associated with less improvement in PTSD symptoms for those receiving WET. In a final study, Jerud and colleagues (Jerud, Zoellner, Pruitt, & Feeny, 2014) examined the hypothesis that individuals with a history of childhood abuse differed in their emotion regulation outcomes from those without such a history, and found that they did not. However, this study did not examine specific differential treatment effects on emotion regulation nor did it examine emotion regulation changes for individuals with higher or lower emotion regulation

difficulties prior to the start of treatment. These latter questions are largely unaddressed in PTSD clinical trials to date.

Effective PTSD treatments, whether or not they directly target emotion regulation, may have a beneficial effect on emotion regulation. Selective serotonin reuptake inhibitors (SSRIs) may facilitate effective emotion regulation through their effect on the serotonin (5-HT) system. Indeed, genetic polymorphisms that are key to serotonin signaling in the brain have been associated with increases in anxiety, depression, and other disorders of emotion regulation (Lesch, 2010; Murakami, Matsunaga, & Ohira, 2009), for which SSRIs show general efficacy. Additionally, therapies such as prolonged exposure (PE) may improve emotion regulation by promoting inhibitory learning of fear responses and enhancing distress tolerance skills (Craske et al., 2008).

Given that extant PTSD treatments without a separate emotion regulation component may be sufficient for improving emotion regulation impairments, the effects of empirically-supported, stand-alone PTSD treatments on emotion regulation need to be examined. Notably, PTSD interventions without separate emotion regulation components require fewer sessions and less therapist training than treatments with added emotion regulation modules, making dissemination less complicated. Our previous work showed that childhood abuse histories did not moderate changes in emotion regulation in a heterogeneous trauma-exposed sample with chronic PTSD (Jerud et al., 2014). Here, using the same sample, we investigate whether PE and sertraline differentially influence emotion regulation and how initial emotion regulation impairments affect emotion regulation outcomes. Individuals receiving PE and sertraline were compared at pre- and post-treatment and at 3- and 6-month follow-up, with emotion regulation examined as a potential effect modifier. We hypothesized that individuals with greater initial emotion regulation difficulties would experience the largest improvements in emotion regulation (reduced expressive suppression, increased cognitive reappraisal, and increased negative mood regulation) from pre-treatment to 6-month follow-up. There was no strong *a priori* hypothesis regarding the comparative efficacy of PE or sertraline in improving emotion regulation.

Method

Participants

Participants ($N = 200$) were recruited from two large, metropolitan communities. Eligible participants were adults aged 18-65 years with a primary DSM-IV diagnosis of chronic PTSD (APA, 2000). Exclusion criteria included: a current diagnosis of schizophrenia or delusional disorder; medically unstable bipolar disorder, depression with psychotic features, or depression severe enough to require immediate psychiatric treatment; a current diagnosis of alcohol or substance dependence within the previous three months; an ongoing intimate relationship with an assault perpetrator; an unwillingness to discontinue current cognitive-behavioral psychotherapy or antidepressant medication; a previous, failed trial of either PE (8 sessions or more) or sertraline (150 mg/day or greater for at least 8 weeks); or a medical contraindication for the initiation of sertraline. Data presented here are emotion regulation outcomes associated with a PTSD clinical trial (NCT00127673) utilizing a doubly

randomized preference design to compare PE and sertraline (Youngstrom, Feeny, Zoellner, Mavissakalian, & Roy-Byrne, 2013).

A total of 426 individuals were assessed for eligibility, of whom 172 were ineligible and 54 were eligible but not interested in study participation. The final sample was predominantly female (75.5%), Caucasian (65.5%), and not college-educated (70%). Rates of current and lifetime comorbid DSM-IV Axis I disorders were 67.1% and 91.3%, respectively. Index DSM-IV Criterion A trauma exposures reported were sexual assault (31%), non-sexual assault (22.5%), childhood assault (24%), motor vehicle accident or natural disaster (13.5%), having a loved one who died or had been exposed to violence (6.5%), and combat/war (2.5%). The mean time since index trauma was 11.97 years ($SD = 12.69$). The mean number of different lifetime DSM-IV Criterion A traumatic events was 9.05 ($SD = 6.23$).

Measures

Structured Clinical Interview for DSM-IV Axis I Disorders With Psychotic Screen (SCID-IV; First, Spitzer, Gibbon, & Williams, 1995)—The SCID-IV is a semi-structured clinical interview that assessed DSM-IV diagnostic comorbidity and exclusion criteria. In the present study, over 10% of the cases were rerated for diagnostic reliability, with good agreement for major depressive disorder ($\kappa = .68$, $p_{pos} = .88$, $p_{neg} = .80$), current anxiety disorders ($\kappa = 1.00$, $p_{pos} = 1.00$, $p_{neg} = 1.00$), substance abuse disorders ($p_{pos} = .00$, $p_{neg} = 1.00$), and other diagnoses ($p_{pos} = .00$, $p_{neg} = 1.00$).

PTSD Symptom Scale-Interview (PSS-I; Foa, Riggs, Dancu, & Rothbaum, 1993)—The PSS-I is a 17-item interview measuring diagnosis and severity of DSM-IV PTSD. PTSD symptoms were assessed for the worst Criterion A traumatic event using a 0 (*not at all*) to 3 (*5 or more times per week/very much*) scale, within the last two weeks. The PSS-I has good concurrent validity with the Clinician-Administered PTSD Scale (Blake, Weathers, Nagy, & Kaloupek, 1995) and the SCID-IV (Foa & Tolin, 2000). In the present study, internal consistency was acceptable ($\alpha = .65$). Approximately 10% of the cases were rerated for diagnostic reliability. Inter-rater reliability was high ($ICC = .985$).

Emotion Regulation Questionnaire (ERQ; Gross & John, 2003)—The ERQ is a 10-item self-report measure of current levels of expressive suppression (i.e., active suppression of emotional experience) and cognitive reappraisal (i.e., reinterpretation of an emotional experience's meaning). Items are scored on a 7-point Likert scale from 1 (*strongly disagree*) to 7 (*strongly agree*) and are averaged. Test-retest reliability is good for both scales (.69; Gross & John, 2003). Normative means for expressive suppression are $M = 3.31$ ($SD = 1.00$) for men and $M = 3.14$ ($SD = 1.18$) for women. Normative means for cognitive reappraisal are $M = 4.60$ ($SD = 0.94$) for men and $M = 4.61$ ($SD = 1.02$) for women (Gross & John, 2003). Internal consistency in this sample was good (cognitive reappraisal: $\alpha = .85$; expressive suppression: $\alpha = .75$).

Negative Mood Regulation Scale (NMR; Catanzaro & Mearns, 1990)—The NMR is a 30-item inventory assessing current beliefs about one's ability to improve negative

moods. Items are assessed using a 5-point Likert scale from 1 (*strong disagreement*) to 5 (*strong agreement*), with higher scores reflecting better emotion regulation. In the present study, internal consistency was good ($\alpha = .91$). Normative means are $M = 99.68$ ($SD = 14.33$) for men and $M = 99.14$ ($SD = 14.33$) for women (Catanzaro & Mearns, 1990).

Prolonged Exposure (PE)

PE consisted of 10 weekly, 90-120 min individual sessions, following a standardized manual (Foa, Hembree, & Dancu, 2002). Procedures included education about common reactions to trauma, breathing retraining, imaginal exposure, processing, *in vivo* exposure, and homework. PE therapists were Masters or Doctoral level clinicians, had CBT experience, and were trained in PE. Weekly supervision included case discussion and session videotapes. Trained raters reviewed 10% of the videotapes, providing integrity and competence ratings using a 3-point scale from 1 (*inadequate*) to 3 (*adequate or better*). PE providers completed 90% of the essential components, and overall competence was very good ($M = 2.73$, $SD = .32$). No protocol violations were observed.

Sertraline

Pharmacotherapy with sertraline was based on a treatment manual (Marshall et al., 2001) and consisted of 10 weekly sessions with a board certified psychiatrist, lasting up to 30 min, with the first lasting 45 min. Neither exposure nor anti-exposure instructions were given. Using a titration algorithm, patients started at 25 mg/day, with the final goal of 200 mg/day if indicated and tolerated. Final dosage ranged from 0 to 300 mg/day, with an average final dosage of 115 mg/day ($SD = 78.00$). Trained raters reviewing 10% of the session videotapes found that sertraline providers completed 96% of the essential components. No protocol violations were observed.

Procedures

Independent evaluators blind to eventual treatment assignment conducted interviews using the SCID-IV and PSS-I. Prior to randomization, potential patients underwent a physical exam, laboratory panel, and a pregnancy test (if applicable), and completed self-report questionnaires including the ERQ and NMR. Randomization was stratified according to PTSD severity and current antidepressant status. Following randomization, patients began 10 weekly sessions of either PE or sertraline. Independent evaluations and self-report measures were again completed at post-treatment and at 3- and 6-month follow-up.

Data Analytic Strategy

To examine whether baseline emotion regulation (ERQ, NMR) moderates the effect of treatment (PE vs sertraline) and time (pre-, post-, 3-month, 6-month follow-up), multilevel linear models were used with a random intercept, fixed slope model, which provided the best fit for the data. Predictor variables were baseline emotion regulation (ERQ, NMR), treated as continuous variables, and treatment (PE, sertraline). Dependent variables were emotion regulation (ERQ, NMR) at post-, 3-month, and 6-month follow-up. Missing data was handled using restricted maximum likelihood estimation. Based on our *a priori* hypotheses, we were particularly interested in the interaction among baseline emotion

regulation, treatment, and time. Significant interactions were followed by examining phase of treatment in terms of acute (pre- to post-) or early (post- to 3-month) or later (3-month to 6-month) follow-up. Effect sizes for the multilevel models were approximated using parameter estimates of t and converting to Cohen's d .

Results

Means and standard deviations, by treatment and over time, are presented in Table 1 for the total sample and for individuals who were 25% below and 75% above the measure's mean at pre-treatment.

Changes in Emotion Regulation

For expressive suppression (ERQ), where higher scores reflect worse emotion regulation ability, there was a main effect of time, $F(1, 536.48) = 9.72, p = .002$, and a baseline expressive suppression $\times$ time interaction, $F(1, 541.90) = 35.51, p < .001$. See Figure 1a. There was no treatment $\times$ time interaction. When examining the baseline expressive suppression $\times$ time interaction, between pre- and post-treatment, for those with worse emotion regulation (higher expressive suppression) at pre-treatment, there was a larger reduction in expressive suppression over time ($b = .46, SE = .08, t(457.58) = 6.03, p < .001; d = 0.56$). This interaction was not present between post-treatment and 3-month follow-up ($d = 0.07$) or between 3- and 6-month follow-up ($d = 0.04$), suggesting that the bulk of reduction in expressive suppression occurred during active treatment for those with higher initial expressive suppression.

For cognitive reappraisal (ERQ), where higher levels of cognitive reappraisal indicate greater emotion regulation, there was a main effect of time, $F(1, 535.74) = 42.61, p < .001$, and a baseline emotion regulation $\times$ time interaction, $F(1, 539.83) = 39.14, p < .001$. See Figure 1. There were no other main effects or interactions. When examining the baseline emotion regulation $\times$ time interaction, between pre- and post-treatment, for those with lower pre-treatment scores, there was a greater increase in cognitive reappraisal over time ($b = .54, SE = .07, t(457.24) = 6.51, p < .001; d = 0.61$). This interaction was not present between post-treatment and 3-month follow-up ($d = 0.01$) or between 3- and 6-month follow-up ($d = 0.08$), suggesting that the majority of improvement occurred during active treatment for those with lower initial cognitive reappraisal abilities.

For negative mood regulation (NMR), where higher scores reflect better perceived ability to alter negative mood states, there was a main effect of time, $F(1, 463.06) = 44.25, p < .001$, and a baseline negative mood regulation $\times$ time interaction, $F(1, 465.12) = 26.92, p < .001$. See Figure 1. There were no other main effects or interactions. When examining the baseline negative mood regulation $\times$ time interaction, between pre- and post-treatment, for those with lower pre-treatment scores, there was a greater increase in negative mood regulation over time ($b = .45, SE = .08, t(393.20) = 5.91, p < .001; d = 0.60$). This interaction was not present between post-treatment and 3-month follow-up ($d = 0.17$) or between 3- and 6-month follow-up ($d = 0.15$), again suggesting that most of the improvement occurred during active treatment for those with lower initial negative mood regulation.

Association Between Emotion Regulation and PTSD Severity

To examine the relationship between pre-treatment emotion regulation (ERQ, NMR) and PTSD severity (PSS-I) over time, correlations were examined. As can be seen in Table 2, only negative mood regulation was moderately associated with PTSD severity over time.

Discussion

Consistent across emotion regulation measures, particularly for those with initial impairments, both PE and sertraline produced clinically meaningful improvements in emotion regulation in a highly comorbid PTSD sample. This improvement was most clear during active treatment, and initial gains were maintained through 6-month follow-up. The changes that occurred were not differentiated by treatment type, suggesting that PE and sertraline both yield improvements in emotion regulation. The present findings suggest that emotion regulation improvements occur over the course of PTSD treatments that do not have a separate emotion regulation component, even in those who present for treatment with initial emotion regulation impairments.

Importantly, patients were not selected *a priori* for emotion regulation deficits but instead entered treatment with a range of emotion regulation abilities. As can be seen by visually inspecting the observed and normative means and standard deviations, some of our sample reported emotion regulation abilities well within the normative range while others reported definite impairments in emotion regulation. It is not surprising then that although there were general improvements in emotion regulation over the course of treatment, these improvements were most noticeable for those with greater initial emotion regulation deficits. This is clinically important because it indicates that individuals with PTSD who have emotion regulation deficits show improvements in emotion regulation with PE or sertraline, even without a specific emotion regulation-focused intervention. Conversely, those that enter treatment with a strong emotion regulation repertoire stand to gain little in regard to emotion regulation from any intervention, as there is no room for further improvement.

The present findings call into question the notion that impaired emotion regulation is a key, maintaining factor in PTSD. Pre-treatment emotion regulation was not consistently associated with PTSD over time, and the pattern varied between emotion regulation measures. This divergence between measures and with some previous studies (e.g., Moore et al., 2008; Tull et al, 2007) may reflect overlapping constructs, particularly for the NMR where some items (e.g., “I won't be able to put it out of my mind.”) are similar to PTSD symptoms, as well as varying levels of PTSD severity seen in treatment-seeking versus non-treatment-seeking samples. Regardless, clinicians should not assume that patients with PTSD have poor emotion regulation skills nor should poor emotion regulation be used as a reason to forego the use of trauma-focused CBT interventions or SSRIs when treating PTSD. It is also encouraging that both of these PTSD treatments improved emotion regulation, for those with initial impairments, despite the fact that neither treatment explicitly teaches emotion regulation skills.

Several caveats should be noted. As expected in a clinical trial, we did not include a psychiatrically healthy control group; thus, it is unclear whether patients differed from

healthy individuals in terms of emotion regulation abilities. That said, when examining normative data (Catanzaro & Mearns, 1990; Gross & John, 2003), patients in the present study who reported poor initial emotion regulation (i.e., upper 75% on expressive suppression and lower 25% on cognitive reappraisal and negative mood regulation) were impaired at baseline, falling more than one standard deviation outside of the normative mean, but not at post-treatment or follow-up. We also cannot definitively say that PE or sertraline directly caused the observed effects, as their changes may be related to the reduction in other symptoms rather than a direct effect of the intervention. Regression to the mean is also an unlikely explanation given that emotion regulation does not improve in PTSD trials with waitlist conditions (e.g., Cloitre et al., 2002) and given that the pattern of improvement altered between active treatment and follow-up. Finally, the present findings likely hold for DSM-5 given that 94% ($n = 188$) of our sample met DSM-5 PTSD diagnostic criteria without including the three new DSM-5 PTSD symptoms.

In summary, both PE and sertraline were associated with sustained improvements in emotion regulation in individuals presenting for PTSD treatment, particularly for those with initial emotion regulation impairments. These gains occurred during active treatment. Brief forms of PTSD treatment, particularly PE and sertraline, may therefore be sufficient for producing emotion regulation improvements among individuals with chronic PTSD.

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Highlights

- Examined changes in emotion regulation for patients undergoing PTSD treatment.
- Not all patients with PTSD reported poor emotion regulation skills.
- Emotion regulation improved with prolonged exposure (PE) and with sertraline.
- Those with worse initial emotion regulation showed largest improvements.
- Poor emotion regulation is not a contraindication for PE or sertraline.

a) Expressive Suppression

b) Cognitive Reappraisal

c) Negative Mood Regulation

Note. To facilitate interpretation, expressive suppression (ERQ) (a), cognitive reappraisal (ERQ) (b), and negative mood regulation (NMR) (c) are presented for individuals +1 SD/-1 SD above or below the mean at pre-treatment. Lower scores indicate better expressive suppression, and higher scores indicated better cognitive reappraisal and negative mood regulation.

Figure 1.

Trajectories for Individuals Higher (+1 SD) and Lower (-1 SD) in Pre-Treatment Emotion Regulation from Pre- to Post-Treatment and through 6 Month Follow-up.

Table 1
Emotion Regulation Means, Standard Deviations, and Effect Sizes from Pre- to Post-Treatment and from Pre- to 6-Month Follow-up

	Sertraline (<i>n</i> = 84)					Prolonged Exposure (<i>n</i> = 116)				
	Pre-Tx	Post-Tx	6-Mo	ES (pre-post)	ES (pre-6 mo)	Pre-Tx	Post-Tx	6-Mo	ES (pre-post)	ES (pre-6 mo)
	<i>M</i> (<i>SD</i>)	<i>M</i> (<i>SD</i>)	<i>M</i> (<i>SD</i>)	<i>Cohen's d</i>	<i>Cohen's d</i>	<i>M</i> (<i>SD</i>)	<i>M</i> (<i>SD</i>)	<i>M</i> (<i>SD</i>)	<i>Cohen's d</i>	<i>Cohen's d</i>
Mean Suppression (ERQ)	3.96 (<i>1.38</i>)	3.55 (<i>1.40</i>)	3.27 (<i>1.31</i>)	0.29	0.51	4.15 (<i>1.25</i>)	3.63 (<i>1.27</i>)	3.32 (<i>1.42</i>)	0.41	0.62
Lower 25%	3.03 (<i>0.95</i>)	3.01 (<i>1.20</i>)	2.81 (<i>1.14</i>)	0.02	0.21	3.27 (<i>0.87</i>)	3.48 (<i>1.18</i>)	2.90 (<i>1.09</i>)	0.20	0.38
Upper 75%	5.25 (<i>0.65</i>)	4.42 (<i>1.27</i>)	3.99 (<i>1.28</i>)	0.82	1.24	5.27 (<i>0.60</i>)	3.78 (<i>1.36</i>)	3.85 (<i>1.61</i>)	1.42	0.50
Mean Reappraisal (ERQ)	4.35 (<i>1.24</i>)	4.64 (<i>1.44</i>)	4.59 (<i>1.39</i>)	0.22	0.18	4.30 (<i>1.12</i>)	4.69 (<i>1.11</i>)	4.55 (<i>1.20</i>)	0.35	0.22
Lower 25%	3.79 (<i>0.84</i>)	4.33 (<i>1.42</i>)	4.27 (<i>1.33</i>)	0.46	0.43	3.74 (<i>0.81</i>)	4.49 (<i>1.05</i>)	4.39 (<i>1.14</i>)	0.80	0.66
Upper 75%	5.94 (<i>0.70</i>)	5.41 (<i>1.20</i>)	5.55 (<i>1.15</i>)	0.54	0.41	5.53 (<i>0.60</i>)	5.15 (<i>1.14</i>)	4.91 (<i>1.28</i>)	0.42	0.42
Mean Negative Mood Regulation (NMR)	96.08 (<i>18.99</i>)	107.53 (<i>21.55</i>)	109.64 (<i>19.76</i>)	0.56	0.70	94.14 (<i>18.72</i>)	105.58 (<i>19.86</i>)	107.68 (<i>18.97</i>)	0.59	0.72
Lower 25%	72.06 (<i>8.91</i>)	100.64 (<i>24.77</i>)	103.34 (<i>20.86</i>)	1.54	1.95	70.35 (<i>9.18</i>)	93.66 (<i>17.67</i>)	99.08 (<i>18.86</i>)	1.66	1.94
Upper 75%	104.55 (<i>13.50</i>)	111.75 (<i>19.21</i>)	113.93 (<i>18.15</i>)	0.43	0.59	102.28 (<i>13.43</i>)	112.51 (<i>17.77</i>)	112.06 (<i>17.64</i>)	0.65	0.62

Note. Overall means and standard deviations for the full sample are presented. To better quantify observed effects, the sample was divided in quartiles, with upper and lower quartile data presented, reflecting those with better and worse reported emotion regulation. Data was analyzed treating these variables as continuous.

TX = Treatment; ES = Effect Size; ERQ = Emotion Regulation Questionnaire; NMR = Negative Mood Regulation. Higher scores on the ERQ Cognitive Reappraisal subscale and NMR are indicative of better emotion regulation, while higher scores on the ERQ Expressive Suppression subscale are indicative of worse emotion regulation.

Table 2

Correlations Between Pre-treatment Emotion Regulation and PTSD Severity at Pre- and Post-treatment and at 6-month Follow-up

	Interview-rated PTSD Severity (PSS-I)		
	Pre-treatment	Post-treatment	6-Mo Follow-up
Expressive Suppression (ERQ)	.14	.09	.06
Cognitive Reappraisal (ERQ)	-.16	-.12	-.20
Negative Mood Regulation (NMR)	-.32 *	-.27 *	-.27 *

Note.

ERQ = Emotion Regulation Questionnaire; NMR = Negative Mood Regulation

* = $p < .05$, based on Holm's step-down procedure for multiple comparisons (Holm, 1979).