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Emotion dysregulation mediates the relationship between trauma type and PTSD symptoms in a diverse trauma-exposed clinical sample☆

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ABSTRACT

Background: Research has explored the influence of trauma type on emotion dysregulation and the role of emotion dysregulation in posttraumatic stress disorder (PTSD). However, it remains unclear whether trauma types differentially impact emotion dysregulation, and whether this in turn contributes to elevated PTSD. The current study tested whether trauma type is related to PTSD symptoms via emotion dysregulation.

Methods: Trauma-exposed community members (n = 209) completed a semi-structured clinical interview and self-reported on emotion regulation, trauma exposure, PTSD symptoms, and negative affect.

Results: Interpersonal trauma, sexual assault in particular, is associated with greater emotion dysregulation. Furthermore, emotion dysregulation mediates the effects of trauma type on PTSD symptoms for sexual assault but not other trauma types, and effects remained significant after covarying for negative affectivity. More recent and chronic trauma was not associated with greater emotion dysregulation.

Conclusions: This study underscores the importance of emotion dysregulation in PTSD development and maintenance. Findings may be used for the development of interventions targeting emotion regulation as a malleable risk factor for PTSD, especially for sexual assault victims.

1. Introduction

Posttraumatic stress disorder (PTSD) is a chronic disorder with serious individual and economic burdens (Kessler, 2000). Up to 90% of people experience a trauma; yet, a minority of individuals will develop PTSD, making it crucial to identify vulnerability factors (Kilpatrick et al., 2013). One risk factor for PTSD is the type of trauma experienced, with increased PTSD symptoms observed in individuals exposed to interpersonal trauma compared to non-interpersonal trauma (Ford, Stockton, Kaltman, & Green, 2006; LeBouthillier, McMillan, Thibodeau, & Asmundson, 2015). However, it is unclear why certain trauma types lead to the development of PTSD more often than others, in part due to limited empirical research.

One promising mechanism underlying the development of PTSD, and differentially associated with trauma type, is emotion dysregulation. Gratz and Roemer (2004) broadly define emotion regulation as an ability to modulate emotional arousal, act in desired ways despite negative emotions, and understand one's emotions. Theoretical and empirical research highlight the role of difficulties with emotion regulation (i.e., emotion dysregulation) in PTSD (O'Bryan, McLeish, Kraemer, & Fleming, 2015; Seligowski, Lee, Bardeen, & Orcutt, 2015), and relevant clinical implications (Cloitre, Koenen, Cohen, & Han, 2002; Wolfsdorf & Zlotnick, 2001). Specifically, individuals who perceive themselves as unable to effectively downregulate negative emotions triggered by trauma reminders may view their emotions as uncontrollable and threatening. This may exacerbate fear of trauma cues, making individuals more likely to avoid trauma reminders that might provoke distress. Emotion regulation is especially important because individuals with difficulties are more likely to engage in maladaptive coping behaviors including avoidance, substance use, and other risky behaviors (Weiss, Tull, Viana, Anestis, & Gratz, 2012). Paradoxically, these avoidant coping strategies maintain PTSD symptoms by preventing individuals from learning accurate information about their ability to handle negative PTSD-related emotions (Naifeh, Tull, & Gratz, 2012).

While the association between emotion dysregulation and PTSD is well-established, it is unclear how emotion dysregulation develops. Developmental psychology research indicates that trauma can cause difficulties regulating emotions (Cloitre, Miranda, Stovall-McClough, & Han, 2005; Glaser, Van Os, Portegijs, & Myin-Germeys, 2006). Early life and interpersonal trauma in particular may disrupt the development and consolidation of adaptive emotion regulation strategies, potentially due to impaired brain development (for a review, see Pollak, 2008). Research

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founded on evolutionary and developmental theory suggests that interpersonal trauma often involves victimization from people that victims otherwise rely on (e.g., family members, partners); in order to maintain a relationship with the perpetrator, victims may respond in ways such as emotional numbing that may be adaptive in the situation as an attempt to decrease tension with the perpetrator and prevent continued or worsened traumatization (Freyd, DePrince, & Zurbriggen, 2001). However, over time, emotional numbing as an emotion regulation strategy may lead to overall difficulties with recognizing and regulating emotions (Feeny, Zoellner, Fitzgibbons, & Foa, 2000). Furthermore, research suggests that sexual assault differs from physical assault, as indicated by elevated self-reported distress and PTSD symptoms (Charuvastra & Cloitre, 2008; Ullman, Peter-Hagene, & Relyea, 2014). Accordingly, additional research is needed to assess the impact of various trauma types on the development of emotion dysregulation, including among vulnerable populations such as veterans who often experience emotional deficits (Price, Monson, Callahan, & Rodriguez, 2006).

While empirical work has been limited, one web-based study by Ehring and Quack (2010) found that early onset chronic interpersonal trauma had the greatest negative impact on emotion regulation compared to non-interpersonal, late onset interpersonal, and early onset singular interpersonal trauma among 616 trauma survivors. However, this study did not examine the effects of emotion dysregulation on subsequent PTSD symptoms. Next, another web-based study by Goldsmith, Chesney, Heath, and Barlow (2013) found that emotion regulation mediates high betrayal trauma (i.e., perpetrator and victim are close), but not low betrayal trauma (i.e., no perpetrator or perpetrator and victim are not close), and posttraumatic stress symptoms among 593 undergraduates. However, this study only focused on betrayal trauma types within a non-clinical sample, combining non-interpersonal with low betrayal interpersonal traumas. Thus, it is unknown whether other trauma types impact emotion regulation difficulties, and whether these difficulties then contribute to PTSD. Better understanding the role of trauma exposure in the development of emotion dysregulation could suggest a modifiable risk factor for reducing PTSD symptoms among individuals exposed to high-risk traumas.

The current study integrates research on the influence of trauma type on emotion dysregulation, and the role of emotion dysregulation in PTSD, by testing the mediating role of emotion dysregulation in the relationship between trauma type and PTSD symptoms. This investigation adds to the literature by further parsing interpersonal trauma into physical and sexual assault while also including combat trauma (Ehring & Quack, 2010). Furthermore, this is the first study to our knowledge to test whether various trauma types influence PTSD symptoms via emotion dysregulation. Hypotheses were tested cross-sectionally in a mixed clinical sample reporting diverse trauma exposure. First, based on previous findings, we hypothesized that interpersonal trauma types (e.g., physical and sexual assault) would be associated with greater emotion dysregulation compared to combat or non-interpersonal trauma (e.g., natural disaster, serious accident, life-threatening illness; Ehring & Quack, 2010). Second, based on research indicating that PTSD symptoms are more severe with more recent and chronic trauma, we hypothesized a similar pattern for the effects of length of time since and chronicity of trauma on emotion dysregulation (Cloitre et al., 2009; Hartley, Sarkisian, Violanti, Andrew, & Burchfiel, 2013). Third, we hypothesized emotion dysregulation would mediate the association between trauma type and PTSD symptoms. We hypothesized effects would hold after covarying for negative affectivity to confirm specificity to emotion dysregulation, rather than generally experiencing increased negative affect.

2. Method

2.1. Participants

Participants were community members recruited from a larger randomized clinical trial investigating a computerized intervention for reducing suicide risk (N = 304; Allan, Boffa, Raines, & Schmidt, 2018). For the current study, participants (n = 209) were selected if they endorsed exposure to a trauma on the Posttraumatic Diagnostic Scale (PDS; Foa, Cashman, Jaycox, & Perry, 1997). Trauma-exposed participants did not significantly differ by emotion regulation compared to participants with no trauma exposure. Eligible participants had complete data for measures of interest, were at least 18 years of age, English speakers, and demonstrated elevated risk for suicide (i.e., elevated levels of anxiety sensitivity cognitive concerns, perceived burdensomeness or thwarted belongingness; Reiss, Peterson, Gursky, & McNally, 1986; van Orden et al., 2010). Participants were not required to demonstrate elevated suicidality (e.g., ideation, intent or prior attempts). Exclusion criteria included psychotic or bipolar-spectrum disorder, psychiatric medication unstable for 6 weeks before study initiation, and serious suicidal intent.

Participant age ranged from 18 to 79 (M = 37.93, SD = 16.00) and 55.0% identified as female. The sample was White (53.1%), Black (34.0%), Asian (1.9%), Pacific Islander (0.5%) and Other (e.g., biracial; 10.5%), with 10% identifying as Hispanic. Most participants were single (55.5%), while 19.6% were divorced, 12.9% married, 4.8% separated, 4.8% widowed and 2.4% cohabitating. Most participants completed some college (54.5%), followed by four-year college (20.1%), graduate school (11.0%), high school or equivalent (11.0%), non-college trade school (2.4%) and 1.0% did not complete high school. Most participants presented with at least one psychiatric diagnosis (92.8%). The distribution of primary diagnoses is as follows: 37.3% anxiety disorder, 23.9% depressive disorder, 20.1% PTSD or other specified trauma, 4.3% obsessive-compulsive and related disorder, 2.4% substance-related disorder, 2.4% bipolar disorder, 0.5% somatic disorder, and 1.9% other (e.g., psychotic). The most common index trauma was sexual assault by familial or known person (20.6%), followed by serious accident, fire or explosion (17.7%), non-sexual assault by familial or known person (15.3%), childhood sexual contact (8.1%), sexual assault by stranger (7.2%), combat (7.2%), life-threatening illness (7.2%), natural disaster (6.2%), non-sexual assault by stranger (5.7%), imprisonment (3.8%) and torture (1.0%). Participants (n = 40) who selected “other” as their index trauma type were excluded from analyses due to unclear trauma descriptions (e.g., “I can't go into that now it will make my head start to hurt”).

2.2. Procedure

Participants were recruited from the community via newspaper advertisements, flyers and other media outlets from 2013 to 2016. Local veteran organizations were prioritized to increase military relevance. Individuals completed a telephone screen to determine eligibility. Participants presented for a baseline appointment involving written informed consent, self-report questionnaires and a clinical interview for the DSM-5 (SCID; First, Williams, Karg, & Spitzer, 2015). This study utilizes data collected prior to randomization and treatment onset. Procedures were approved by the university's institutional review board.

2.3. Measures

2.3.1. Structured clinical interview for DSM-5 disorders- research version (SCID-V-RV)

The SCID-V-RV (First et al., 2015) was used to determine psychiatric diagnoses. SCIDs were administered by trained graduate students and reviewed by a licensed clinical psychologist. A random sample of 20 SCID interviews from this study demonstrated high inter-rater agreement (i.e., a kappa value of 0.86; Schmidt, Norr, Allan, Raines, & Capron, 2016).

2.3.2. Posttraumatic diagnostic scale (PDS)

The PDS (Foa et al., 1997) includes a 12-item checklist of traumatic event exposure. Participants indicate which traumatic event was most

Table 1
Means, standard deviations, and zero-order correlations of all variables.

Variable	Overall sample				Trauma groups (means & *SD*s)			
	Mean	*SD*	Min	Max	Combat	Non-interpersonal	Physical assault	Sexual assault
PCL-C	47.45	17.69	17	85	51.60 (17.34)	42.60 (16.90)	46.65 (17.00)	51.40 (18.11)
DERS	98.42	26.30	37	166	85.13 (22.92)	91.88 (23.94)	99.26 (23.83)	106.13 (28.42)
PANAS-NA	26.47	9.58	10	50	23.20 (8.52)	25.08 (8.26)	26.06 (9.84)	28.63 (10.35)
PDS 15	5.25	1.25	1	6	5.60 (0.74)	4.96 (1.60)	5.41 (0.96)	5.33 (1.14)
PDS 16	1.89	0.92	1	3	2.40 (0.91)	1.54 (0.79)	1.87 (0.89)	2.11 (0.94)

Note: $n = 209$; Combat $n = 15$; Non-interpersonal $n = 65$; Physical assault $n = 54$; Sexual assault $n = 75$. PCL-C = Posttraumatic Stress Disorder Checklist–Civilian Version total score; DERS = Difficulties in Emotion Regulation Scale; PANAS-NA = Positive and Negative Affect Schedule–Trait–Negative Affect Subscale; PDS 15 = Posttraumatic Diagnostic Scale question 15, length since trauma, with 1 = *< 1 month*, 2 = *1 to 3 months*, 3 = *3 to 6 months*, 4 = *6 months to 3 years*, 5 = *3 to 5 years*, and 6 = *> 5 years*; PDS 16 = Posttraumatic Diagnostic Scale question 16, chronicity, with 1 = *Once*, 2 = *Twice*, and 3 = *Three or more times*.

bothersome. Additionally, PDS question 15 assesses how long ago the traumatic event happened (i.e., length since trauma) with 1 = *< 1 month*, 2 = *1 to 3 months*, 3 = *3 to 6 months*, 4 = *6 months to 3 years*, 5 = *3 to 5 years*, and 6 = *> 5 years*. PDS question 16 assesses the frequency of the event (i.e., chronicity), with 1 = *Once*, 2 = *Twice*, and 3 = *Three or more times*. Traumatic events were categorized as non-interpersonal (i.e., natural disaster, serious accident, life-threatening illness), physical assault (i.e., non-sexual assault by familial or known person, non-sexual assault by a stranger, imprisonment, torture), sexual assault (i.e., sexual assault by familial or known person, sexual assault by a stranger, sexual contact under the age of 18 with someone 5 or more years older) and combat (i.e., military combat or war zone). Trauma type was coded as one variable. These categories are consistent with the limited research implying that whether or not trauma is interpersonal is an important predictor of emotion dysregulation (Ehring & Quack, 2010). A combat trauma type category was created due to the elevated number of veterans recruited. The PDS has demonstrated strong psychometric properties in previous research (Powers, Gillihan, Rosenfield, Jerud, & Foa, 2012).

2.3.3. Posttraumatic stress disorder checklist–civilian version (PCL-C)

The PCL-C (Weathers, Litz, Herman, Huska, & Keane, 1993) is a 17-item measure of PTSD symptom severity corresponding with the DSM-IV. Participants rate symptoms in the past month, as related to the trauma self-identified in the PDS. Items are rated on a 5-point Likert scale, with higher scores indicating increased PTSD severity. The PCL-C has been demonstrated to be psychometrically sound (Blanchard, Jones-Alexander, Buckley, & Forneris, 1996) and demonstrated excellent reliability at baseline ($\alpha = 0.95$).

2.3.4. Difficulties in emotion regulation scale (DERS)

The DERS (Gratz & Roemer, 2004) is a 36-item measure of emotion dysregulation. Based on research suggesting that general emotion dysregulation has large effect sizes (Seligowski et al., 2015), this study only used a total score. Items are rated on a 5-point Likert scale, with higher scores indicative of greater emotion dysregulation. The DERS has been shown to be psychometrically sound (Gratz & Roemer, 2004) and demonstrated excellent reliability at baseline ($\alpha = 0.94$).

2.3.5. Positive and negative affect schedule–trait–negative affect subscale (PANAS-NA)

The PANAS-NA (Watson, Clark, & Tellegen, 1988) is a 10-item measure of negative affect. Participants indicate to what extent they experienced particular negative emotions in the past week using a 5-point Likert scale ranging from 1 (*Very slightly*) to 5 (*Extremely*). The PANAS-NA has demonstrated excellent psychometric properties in previous research (Watson & Clark, 1994) and demonstrated excellent reliability at baseline ($\alpha = 0.91$).

2.4. Data analytic plan

To test our first hypothesis that emotion dysregulation would differ by trauma type, an ANOVA was conducted in which total DERS was associated with trauma type. A series of contrast tests were then used to determine which trauma types significantly differed by DERS. Next, a mediational model testing the indirect effect of trauma type on PTSD symptoms via DERS, covarying for NA, was conducted. We used Preacher and Hayes' PROCESS macro for SPSS (Preacher & Hayes, 2008), which utilizes 5000 bootstrap resamples to compute estimates for the paths from independent variable to mediator (α) and the path(s) from mediator to outcome (β), as well as the indirect effect (α β), yielding 95% confidence intervals of the indirect effect based on the bootstrap distribution. PROCESS can handle categorical variables (e.g., trauma type), and we used the indicator coding system. Combat exposure was set as the indicator trauma type because this group had the lowest relative mean score for the DERS. All effects are reported as unstandardized coefficients.

3. Results

3.1. Descriptive statistics and correlations

Descriptive statistics and intercorrelations are found in Tables 1 and 2. No threats to or violations of skewness, kurtosis, homoscedasticity and multicollinearity were found. All variables were positively and significantly correlated. Distributions for trauma type are as follows: 7.2% combat, 31.1% non-interpersonal, 25.8% physical assault, and 35.9% sexual assault. Participants presented with clinical levels of PTSD as suggested by PCL-C scores averaging above 44 (Ruggiero, Del Ben, Scotti, & Rabalais, 2003). The overall mean DERS score was higher compared to means of 86.3–87.5 found in trauma-exposed and clinical samples (Tull, Barrett, McMillan, & Roemer, 2007; Weiss et al., 2012). Additionally, the overall mean PCL-C score was higher compared to a mean of 40.0 found among trauma-exposed samples (Tull et al., 2007),

Table 2
Zero-order correlations for variables of interest.

	1	2	3	4	5
1. PCL-C	1	–	–	–	–
2. DERS	0.63**	1	–	–	–
3. PANAS-NA	0.66**	0.67**	1	–	–
4. PDS 15	0.11	0.06	0.05	1	–
5. PDS 16	0.16*	0.10	0.13	0.09	1

Note: $n = 209$; * = $p < .05$; ** = $p < .01$. PCL-C = Posttraumatic Stress Disorder Checklist–Civilian Version total score; DERS = Difficulties in Emotion Regulation Scale; PANAS-NA = Positive and Negative Affect Schedule–Trait–Negative Affect Subscale; PDS 15 = Posttraumatic Diagnostic Scale question 15, length since trauma; PDS 16 = Posttraumatic Diagnostic Scale question 16, chronicity.

while the mean PCL-C score for the combat trauma group was comparable to a mean of 52.4 found in military samples (Kearney, McDermott, Malte, Martinez, & Simpson, 2012).

3.2. Primary analyses

To determine whether levels of emotion dysregulation differed by trauma type, an ANOVA was conducted in which trauma type was associated with DERS total. The overall model was significant ($F(3, 205) = 5.07$, $p = .002$). Next, all trauma types were tested against one another sequentially using contrast tests. Individuals with histories of combat exposure did not significantly differ from those with histories of non-interpersonal ($p = .358$) or physical assault exposure ($p = .060$), but had significantly lower DERS scores than sexual assault survivors ($F(1, 205) = 8.44$, $p = .004$). Individuals with histories of non-interpersonal trauma did not significantly differ from individuals with histories of physical assault trauma ($p = .118$), and had significantly lower DERS scores than sexual assault survivors ($F(1, 205) = 10.84$, $p = .001$). Finally, those reporting physical assault did not significantly differ in DERS from those reporting sexual assault ($p = .133$).

Subsequently, we examined whether length of time since trauma and chronicity of trauma were associated with elevated emotion dysregulation. Two multiple regressions were used with time or chronicity and negative affectivity (as a covariate) as independent variables associated with DERS total scores. Neither length of time since trauma ($B = 0.49$, SE = 0.93, $t = 0.53$, $p = .598$), or chronicity of trauma ($B = 0.02$, SE = 1.36, $t = 0.01$, $p = .991$) was significantly associated with emotion dysregulation.

3.3. Mediation model

Next, we conducted a mediation model to test the indirect effects of trauma type on PTSD symptoms via emotion dysregulation, covarying for negative affectivity (see Fig. 1). We did not include length of time since or chronicity of trauma in the model since these variables were not associated with DERS. The entire model was significant and accounted for 72.8% of the variance in PTSD symptoms ($F(5, 203) = 45.74$, $p < .001$). First, in terms of the α path, only the sexual assault trauma type was significantly associated with increased DERS ($B = 11.42$, SE = 5.53, $t = 2.06$, $p = .040$), while other trauma types did not ($ps > .110$). Negative affectivity was also significantly associated with DERS ($B = 1.77$, SE = 0.14, $t = 12.38$, $p < .001$). Regarding the ß path, DERS was significantly associated with PTSD symptoms ($B = 0.23$, SE = 0.04, $t = 5.21$, $p < .001$), as did negative affectivity ($B = 0.81$, SE = 0.12, $t = 6.77$, $p < .001$). The indirect effect of trauma type on PTSD symptoms via DERS was significant for sexual assault ($B = 2.64$, 95% CI[0.49, 5.23]), but not other trauma types ($B = 0.79$, 95% CI[−1.52, 3.19], and $B = 2.10$, 95% CI[−0.08, 4.52]).

4. Discussion

This study is the first to show that emotion dysregulation mediates the effects of trauma type on PTSD symptoms within a diverse clinical sample. Results show that interpersonal trauma, sexual assault in particular, is associated with greater emotion dysregulation. Findings highlight the importance of emotion dysregulation in PTSD development and maintenance (Seligowski et al., 2015).

Consistent with our first hypothesis and previous research, we found that interpersonal trauma is associated with greater emotion dysregulation compared to non-interpersonal or combat trauma (Ehring & Quack, 2010; Goldsmith et al., 2013). Findings add to the literature by suggesting that sexual assault in particular is associated with greater emotion dysregulation. Previous research has found that childhood and adult sexual assault is associated with increased emotion dysregulation (Cloitre et al., 2005; Ullman et al., 2014). Experiencing a trauma, especially interpersonal, may interfere with the development of adaptive emotion regulation strategies and/or disrupt neural circuitry underlying emotion regulation (Pollak, 2008). It is plausible that victims of sexual assault specifically may have experienced a more intimate betrayal from a perpetuator who victims otherwise rely on, and thus experience more intense distress following a trauma. This could lead to increased maladaptive ways to cope, including emotional responses (e.g., emotional numbing, avoidance) and risky behavior (e.g., substance use, sexual behavior, self-injury) that paradoxically perpetuate difficulties regulating negative trauma-related emotion and chronic PTSD (Feeny et al., 2000; Weiss et al., 2012). Indeed, prior studies indicate that interpersonal trauma, especially rape, is frequently self-reported for men and women as the worst trauma type and is associated with increased PTSD symptoms (Charuvastra & Cloitre, 2008). Future research should investigate behavioral and neurobiological effects of trauma that may lead to changes in emotion regulation, specifically those unique to sexual assault.

Contrary to our second hypothesis, we did not find that more recent and chronic trauma was associated with greater emotion dysregulation after covarying for negative affectivity. While previous research has found that PTSD symptoms are more severe with recent and chronic trauma (Cloitre et al., 2009; Hartley et al., 2013), this study is the first to explore this pattern in relation to emotion dysregulation. Findings imply that individuals may develop varying levels of emotion dysregulation that persist regardless of length of time since and chronicity of trauma. These findings fail to replicate research suggesting that early

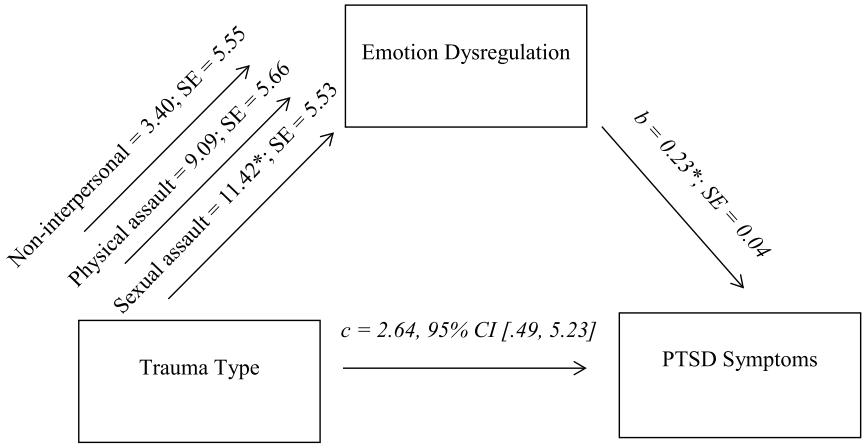

Fig. 1. Mediational model examining the effects of trauma type on PTSD symptoms through emotion dysregulation. This model covaries for PANAS-NA. * = $p < .05$.

onset trauma leads to increased emotion dysregulation compared to late onset trauma, potentially due to qualitative differences between childhood and adult trauma that cannot be captured by only assessing length of time since trauma (Ehring & Quack, 2010). Additional research identifying factors that influence emotion dysregulation can aid the development of emotion regulation interventions.

Consistent with our third hypothesis, a key finding is that emotion dysregulation mediates the effects of trauma type on PTSD symptoms. Interestingly, this association shows specificity since mediational effects were significant for sexual assault but not for other trauma types. These findings bring together two lines of research: the effects of trauma type on emotion dysregulation, and the effects of emotion dysregulation on PTSD symptoms. This study highlights the devastating influence of trauma on emotion dysregulation, and corroborates research identifying emotion dysregulation as a risk factor for elevated PTSD (Seligowski et al., 2015).

These findings have important clinical implications. A strength of this study includes utilizing a representative clinical sample. Findings call for studies to test interventions targeting emotion regulation in trauma survivors, which may be especially useful for sexual assault victims. Clinicians should consider trauma type for PTSD treatment. Some studies call for a phase-based treatment approach to PTSD in which affective regulation skills are delivered prior to trauma-related treatment (Cloitre et al., 2002; Wolfsdorf & Zlotnick, 2001). However, additional research is needed to test this treatment approach compared to first line PTSD treatments (e.g., exposure therapy; Cahill, Zoellner, Feeny, & Riggs, 2004). This study may be useful for explaining mixed findings regarding the administration of emotion regulation treatment prior to trauma-related treatment.

Results of this study should be considered in light of its limitations. First, this study relied on self-reported emotion regulation. Although we used a valid and reliable measure of emotion dysregulation (i.e., DERS; Gratz & Roemer, 2004), it is possible that some individuals may not be able to accurately self-report on emotion regulation difficulties. Future research could incorporate physiological measures of emotional arousal (e.g., skin conductance response) that is related to use of emotion regulation strategies in real-time (Shepherd & Wild, 2014). Second, while PTSD symptoms were assessed using the PCL-C, a well-validated and widely used measure of PTSD symptoms based on the DSM-IV (Blanchard et al., 1996), we were unable to utilize the more recent PCL-5 (Weathers et al., 2013) that was not developed at the time of study onset. Yet, prior research indicates a high association between the PCL-C and PCL-5 (Rosellini et al., 2015). Third, this study relied on cross-sectional associations. It is also possible that PTSD symptoms contributed to the development of emotion dysregulation. Future research should prospectively test the temporal associations between emotion dysregulation, trauma exposure, and PTSD symptoms. Fourth, we were unable to distinguish between childhood and adult trauma because the PDS assesses length of time since trauma, not whether trauma occurred during childhood or adulthood. Since previous research suggests that early onset trauma may lead to greater emotion dysregulation, future research should assess these differences within a clinical sample (Ehring & Quack, 2010). Fifth, recent trauma was infrequently reported, thus limiting conclusions about the role of emotion dysregulation in the immediate response to trauma. Future research should actively recruit individuals shortly following a trauma. Sixth, while this study was evenly distributed in terms of gender, findings might be confounded because men are less likely to report sexual assault (Sable, Danis, Mauzy, & Gallagher, 2006). Additional research should investigate the role of emotion dysregulation in a gender-balanced sample of sexual assault victims to better assess gender differences. Seventh, while this study highlights the role of emotion regulation, there may be other variables that could account for the association between trauma and PTSD (e.g., personal efficacy) that were not assessed.

Despite these limitations, the current study adds to the literature on emotion dysregulation in PTSD by demonstrating that emotion dysregulation mediates the effects of trauma type on PTSD symptoms within a diverse clinical sample. Findings suggest that emotion dysregulation may be uniquely associated with interpersonal trauma, especially sexual assault. Knowledge from these findings may be applied to the development of interventions targeting emotion regulation as a malleable risk factor in individuals with PTSD, particularly for sexual assault victims.

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