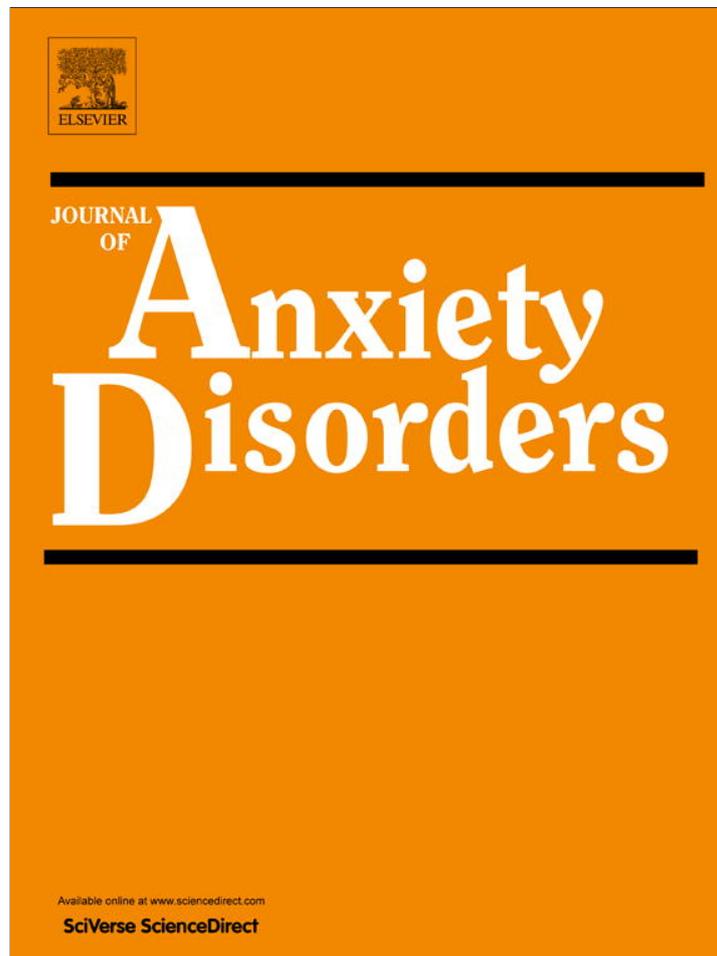


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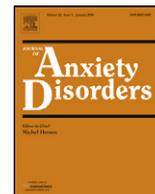
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Journal of Anxiety Disorders



Emotion regulation difficulties as a prospective predictor of posttraumatic stress symptoms following a mass shooting



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ARTICLE INFO

Article history:

Received 6 August 2012

Received in revised form 14 January 2013

Accepted 16 January 2013

Keywords:

Emotion regulation

Posttraumatic stress disorder (PTSD)

Trauma

Disaster

Mass violence

Cross-lagged panel design

Longitudinal

ABSTRACT

A strong positive association between emotion regulation difficulties (ERD) and posttraumatic stress symptoms (PTSS) has been consistently evidenced in cross-sectional research. However, a lack of prospective research has limited hypotheses regarding the temporal relationship between trauma exposure, ERD, and PTSS. The present prospective study investigated the role of pre-trauma difficulties with emotion regulation in the development of PTSS following exposure to a potentially traumatic event. Between Time 1 (T1) and Time 2 (T2), a mass shooting occurred at the participants' ($n = 691$) university campus. ERD and PTSS were assessed prior to the shooting (T1), in the acute aftermath of the shooting (T2), and approximately eight months later (T3). Using a cross-lagged panel design, ERD was found to prospectively predict PTSS from T1 to T2 and T2 to T3. Additionally, PTSS prospectively predicted ERD from T1 to T2. However, T2 PTSS failed to predict T3 PTSS. Results indicate that ERD and PTSS are reciprocally influential from pre- to post-shooting. Further, results suggest that emotion dysregulation in the aftermath of a potentially traumatic event influences one's ability to recover from PTSS over time, even after accounting for the effects of existing symptomatology. To examine the specificity of temporal relations between ERD and PTSS a second cross-lagged panel design, in which a general distress construct was substituted for PTSS, was conducted. Results of this analysis, as well as conceptual and clinical implications, will be discussed.

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1. Introduction

Posttraumatic stress disorder (PTSD) is comprised of symptoms including intense distress at exposure to trauma-related reminders, avoidance of internal and external cues associated with the traumatic experience, emotional numbing, and hyperarousal (American Psychiatric Association [APA], 2000). The symptom constellation of PTSD reflects a broad range of difficulties regulating emotions, spanning from hyperreactivity in the form of re-experiencing symptoms to emotional hyporeactivity in the form of anhedonia and restricted range of affect (Ehring & Quack, 2010; Frewen & Lanius, 2006; Litz, Orsillo, Kaloupek, & Weathers, 2000; Litz, 1992). Deficits in adaptive emotion regulation have been identified as a central feature of PTSD (e.g., Frewen & Lanius, 2006; Litz et al., 2000), and results of field trials for the DSM-IV indicated that over 70% of those with PTSD endorsed difficulties in regulating emotions (van der Kolk, Roth, & Pelcovitz, 1993). Theoretical perspectives regarding factors that promote and maintain posttraumatic stress symptoms (PTSS) indicate a disruption in

cognitive, physiological, or behavioral affect regulatory processes (Brewin, Dalgleish, & Joseph, 1996; Ehlers & Clark, 2000; Foa & Kozak, 1986; Frewen & Lanius, 2006). However, despite a growing body of literature indicating a link between emotion regulation deficits and PTSD symptomatology (Eftekhari, Zoellner, & Vigil, 2009; Ehring & Quack, 2010; Tull, Barrett, McMillan, & Roemer, 2007), the precise nature of the relationships among emotion dysregulation and PTSD is unclear.

Gratz and Roemer (2004) identify emotion regulation as the ability to monitor, evaluate, and modulate emotional reactions within the context of goal-directed behavior. Gratz and Roemer's integrated conceptualization of emotion regulation has been shown to involve awareness and clarity of emotional responses, emotional acceptance, access to effective emotion regulation strategies, and control of impulses and engagement in goal-directed behaviors when experiencing negative emotion. Difficulties with emotion regulation have been associated with a range of psychopathology, including depression (Gross & Munoz, 1995), borderline personality disorder (Gratz, Rosenthal, Tull, Lejuez, & Gunderson, 2006), anxiety disorders in general (for a review, see Cisler, Olatunji, Feldner, & Forsyth, 2010), and PTSD (Eftekhari, Zoellner, & Vigil, 2009; Ehring & Quack, 2010; Tull, Barrett, McMillan, & Roemer, 2007).

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Although emotion regulation deficits are associated with multiple forms of psychopathology, they may be specifically salient in the context of PTSD. The current diagnostic classification of PTSD indicates that the subjective experience of intense peritraumatic fear, helplessness, or horror in response to a traumatic stressor (Criterion A2) is a vital etiological component (APA, 2000). However, at present, Criterion A2 is the topic of much debate, with some suggesting that the criterion has limited clinical utility (Breslau & Kessler, 2001; Brewin, Lanius, Novac, Schnyder, & Galea, 2009), and others suggesting an expansion of Criterion A2 (Bovin & Marx, 2011; Kilpatrick, Resnick, & Acierno, 2009) to include additional acute trauma-related emotional reactions (e.g., shame, guilt, anger; Rubin, Berntsen, & Bohni, 2008).

Regardless, the appraisal of a stressor as threatening and in excess of an individual's coping resources, as well as the subjective experience of intense peritraumatic emotion, has been proposed as increasing risk of developing PTSD (Bovin & Marx, 2011). Deficits in emotion regulation would likely lead to greater appraisals of threat, diminished coping resources, and more intense emotional responding upon exposure to a traumatic stressor, and thus, these deficits may function in the etiology of PTSD. Consistent with this hypothesis, threat appraisal has emerged as a more reliable predictor of PTSD symptoms than objective measures of danger in some samples (Bernat, Ronfeldt, Calhoun, & Arias, 1998; Ehlers, Mayou, & Bryant, 1998; King, King, Gudanowski, & Vreven, 1995; Ullman & Filipas, 2001). Additional empirical evidence, however, suggests complex relationships between emotion regulation, trauma exposure, and PTSS (Ehring & Quack, 2010) when examined concurrently. Available research relies on cross-sectional designs, therefore, the precise nature of these associations is unknown.

Intense emotional reactions to trauma-related cues are common in the aftermath of traumatic events (e.g., Rothbaum, Foa, Riggs, Murdock, & Walsh, 1992; Shalev, Sahar, Freedman, Peri, Glick, & Pitman, 1998); however, these reactions diminish for most trauma survivors (Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995; Rothbaum et al., 1992; Shalev et al., 1998). Thus, peritraumatic emotional responding alone is not sufficient to account for the development of PTSD. Possessing adequate emotion regulation skills may be characteristic of those able to recover in the aftermath of a traumatic experience, while individuals lacking the emotion regulation capacity necessary to attenuate their arousal and distress may be at risk of chronic disruptions in emotional responding associated with PTSD (Frewen & Lanius, 2006). Deficits in emotion regulation likely contribute to the maintenance of PTSD in multiple ways. Individuals may perceive their emotions as uncontrollable (Frewen & Lanius, 2006) and subsequently learn to fear internal and external cues that elicit emotional reactions. A lack of access to adaptive emotion regulation strategies may then lead to avoidance of trauma-related experiences, thus preventing exposure to trauma relevant reminders that would otherwise facilitate habituation (Foa & Kozak, 1986).

Despite a growing body of literature implicating emotion dysregulation in the development and maintenance of PTSD, it is currently unknown whether emotion dysregulation is primarily a cause or consequence of PTSS. Cross-sectional research demonstrated that severity of PTSS was positively associated with self-reported difficulties with multiple components of emotion regulation (i.e., lack of emotional acceptance, impulse control difficulties, and lack of access to effective emotion-regulation strategies), even when overall level of negative affect was taken into account (Tull et al., 2007). Furthermore, individuals reporting PTSS suggestive of a diagnosis of PTSD endorsed subjective deficits in overall emotion regulation, as well as specific components of impulse control difficulties, lack of access to effective emotion regulation strategies, and lack of emotional clarity when controlling for the effects of negative affect (Tull et al., 2007). Severity of PTSS has

also been linked cross-sectionally to reduced subjective capacity and self-reported frequency of emotion regulation strategies, suggesting that deficits in emotion regulation flexibility play a role in PTSS (Eftekhari et al., 2009). Psychophysiological research also has demonstrated an association between PTSD and deficits in emotional processing and positive emotional expression (Litz et al., 2000). Individuals with PTSD exhibit heightened negative emotion and diminished positive emotion in response to emotionally evocative stimuli (Amdur, Larsen, & Liberzon, 2000) and exposure to trauma-related cues has been associated with enhanced negative affective reactions and subsequent defensive responding to negatively valenced stimuli in the context of PTSD (Miller & Litz, 2004). Interestingly, in an experimental study by Badour and Feldner (2013) PTSS were found to be predictive of emotional reactivity to trauma cue exposure, but only among individuals who had relatively higher levels of emotion regulation difficulties (ERD).

Although the sum of the available literature supports a meaningful link between deficits in emotion regulation and PTSD, the lack of prospective studies examining to what extent emotion regulation deficits are a risk factor for, or a consequence of, psychological distress in the aftermath of a traumatic experience limits directional conclusions. One proposition within the literature is that trauma exposure creates a disruption in emotion regulation capacity (e.g., Frewen & Lanius, 2006; van der Kolk, 1996). Marked disruptions in emotion regulation have been noted in individuals exposed to chronic interpersonal trauma, such as prolonged childhood abuse (e.g., Cloitre, Miranda, Stovall-McClough, & Han, 2005; van der Kolk, 1996), suggesting that trauma exposure itself may degrade the capacity for emotion regulation. However, recent research suggests ERD are not unique to survivors of early-onset interpersonal trauma and that the relationship between emotion regulation and trauma type is a function of PTSD symptom severity (Ehring & Quack, 2010), raising further questions regarding the role of emotion dysregulation in the course of PTSD. Moreover, Ehring and Quack (2010) voiced concerns regarding the high degree of overlap between definitions of emotion regulation and symptoms of PTSD, which may lead to inflated associations between the constructs. This would be of particular concern when symptomatology and emotion regulation are measured concurrently.

To address limitations in the available research and clarify associations between ERD and PTSS, the present prospective study investigates the role of pre-trauma difficulties with emotion regulation in the development of PTSS following exposure to a potentially traumatic event. Initially, we had intended to examine prospective predictors of sexual victimization in a sample of undergraduate women enrolled in a longitudinal study. However, the course of the longitudinal study took an unexpected turn in February of 2008, when a gunman opened fire on a classroom of students on the Northern Illinois University (NIU) campus, in DeKalb, Illinois. It was the fourth deadliest school shooting in U.S. history; before taking his own life, the gunman left five students dead and 21 wounded. Due to the trauma-focused nature of the initial longitudinal study, we were in a unique position to examine the effects of ERD in the development of PTSS following an episode of mass-violence.

The extant literature provides important clues about the relationships between ERD and PTSS; however, a number of questions remain. Due to a lack of prospective research examining predictive relationships between ERD and PTSS, the temporal relationship between these constructs remains unclear. Research that includes pre-trauma assessments of emotion regulation and accounts for the effects of previously reported PTSS is needed for the affirmation of temporal precedence. As suggested by some (e.g., Cloitre et al., 2005; van der Kolk, 1996), trauma exposure itself may degrade the capacity for emotion regulation. On the other hand, a deficit in one's ability to down-regulate emotional arousal may lead one to perceive emotions as uncontrollable and increase the likelihood

of fear acquisition (Bouton, Mineka, & Barlow, 2001). Individuals with emotion regulation deficits may be more likely to associate trauma cues with the negative affect and arousal experienced during the event, thus resulting in avoidance of feared situations and the subsequent development of PTSS. The prospective nature of the present study provides a unique opportunity to clarify the temporal nature of the association between these constructs.

The lack of prospective research in this area precluded a priori hypotheses regarding temporal specificity of the relationship between emotion regulation and PTSS. That is, as described above, it may be that deficits in emotion regulation are the result of trauma exposure and prior PTSS, or ERD may be a vulnerability factor in the development of PTSS. Additionally, in order to examine the specificity of observed temporal relations between ERD and PTSS, a second analysis was conducted in which a general distress construct was substituted for PTSS.

2. Method

2.1. Participants and procedure

One thousand and forty-five female undergraduate students from Northern Illinois University (NIU), aged 18 or older, were recruited for participation (T1) in a longitudinal study originally designed to examine trauma and sexual revictimization. In order to participate, the sole requirements were that women were over the age of 18 and fluent in English; although the study focused on sexual revictimization, participants were not selected based on the presence or absence of trauma history of any nature. At T1, participants provided written informed consent and completed a battery of measures including a subset of measures assessing PTSS and ERD. T1 data collection began during the Fall of 2006 (via rolling admission), and was halted when the shooting occurred on February 14th, 2008. We implemented a longitudinal study design in which participants from the pre-shooting assessment session (T1) completed two post-shooting assessment sessions (T2 & T3). PTSS (Distressing Events Questionnaire), ERD (Difficulties in Emotion Regulation Scale), and anxiety, depression, and stress (Depression, Anxiety, Stress Scale) were measured at all three time-points (see below for a description).

Eighty-five percent ($n=885$) of T1 participants consented to follow-up contact. Of these participants, 92% ($n=812$) were determined to be current students at the time of the mass shooting, and thus, members of the exposed community. This subsample was invited via e-mail to complete an on-line battery of questionnaires (T2), which took approximately 30 min to complete. Of those invited, 691 (85%) responded to the e-mail invitation and completed the post-shooting assessment. The interval between T1 and the mass shooting was variable, particularly given that T1 data collection began in the Fall 2006 semester and ended in Spring 2008, although the majority of participants completed T1 in Fall 2007 ($M=191.0$ days; $SD=149.1$; range 0–519 days). Additionally, the time elapsed between the mass shooting and completion varied ($M=29.5$ days; $SD=16.0$; range 17–100 days).

Participants from the T2 sample ($n=691$) were invited via e-mail to complete an additional follow-up survey online approximately seven months post-shooting. Of the T2 sample, 588 (85%) participants completed the T3 session, which took approximately 30 min. The average time elapsed between the shooting and completion of the T3 assessment varied ($M=241.78$ days; $SD=21.79$; range 227–346 days); however, approximately 90% of the T3 sample completed the survey within 9 months of the shooting.

The mean age of T2 completers at T1 ($n=691$) was 19.6 years ($SD=2.7$), and most were freshman (73%). In terms of race, 67.9% self-identified as White, 20.1% as Black, 3.0% as Asian, 0.1% as

American Indian or Alaska Native, 0.1% as Native Hawaiian or other Pacific Islander, 7.4% endorsed “other”, while 1.3% preferred not to respond. Additionally, 7.1% of T2 participants identified as Hispanic. The T3 sample had a similar demographic makeup, with a mean age at T1 of 19.6 years ($SD=2.2$), most were freshman (74%) and 70.7% self-identified as White, 17.5% as Black, 3.0% as Asian, 0.2% as American Indian or Alaska Native, 0.2% as Native Hawaiian or other Pacific Islander, 7.3% endorsed “other”, while 1.2% preferred not to respond. Additionally, 7.5% of T3 participants identified as Hispanic.

2.2. Measures

2.2.1. Descriptives and potential covariates

Age and race/ethnicity were evaluated as potential covariates in our analyses. Race and ethnicity were assessed according to the National Institute of Health policy on reporting race (five categories plus “other”) and ethnicity (Hispanic or Latina) data. Race and ethnicity were collapsed into a single dummy coded variable (coded as White and Non-Hispanic [$n=459$, 66.9%] versus all others [$n=227$, 33.1%]). Additionally, to account for duration effects, the intervals between (a) T1 and T2, (b) the mass shooting and T2, and (c) T2 and T3 were calculated for use in longitudinal analysis.

2.2.2. Difficulties in Emotion Regulation Scale

The Difficulties in Emotion Regulation Scale (DERS; Gratz & Roemer, 2004) is a 36-item self-report measure used to assess six dimensions of emotion regulation: Nonacceptance of Emotional Responses [Nonacceptance], Difficulty Engaging in Goal-Directed Behavior [Goals], Impulse Control Difficulties [Impulse], Lack of Awareness of Emotions [Awareness], Limited Access to Strategies for Regulation [Strategies], and Lack of Emotional Clarity [Clarity]. Items are rated on a 5-point scale based on how often participants believe each item pertains to them (1 = *almost never* to 5 = *almost always*). At T1 and T3, the instructions read, “Please indicate how often the following statements apply to you.” At T2, the instructions were slightly modified to cue the participants to the mass shooting; instructions at T2 read, “Since the mass shooting at NIU on February 14, 2008, please indicate how often the following statements apply to you.” Although the DERS has exhibited adequate internal consistency and good test retest reliability (Gratz & Roemer, 2004), recent evidence suggests that the DERS-Awareness dimension may not belong to the same higher order emotion regulation construct as the other five DERS dimensions (Bardeen, Fergus, & Orcutt, 2012). Thus, in the present study, the DERS-Awareness subscale was not used as an indicator of the latent construct of ERD in longitudinal analysis. All DERS subscales evidenced adequate internal consistency across assessment sessions (α values ranging from .77 to .92).

2.2.3. Traumatic Life Events Questionnaire

The Traumatic Life Events Questionnaire (TLEQ; Kubany, Haynes, et al., 2000) assesses exposure to 22 potentially traumatic events (e.g., natural disasters, combat or warfare, assault, sexual abuse) consistent with Criterion A1 and A2 of PTSD as specified in the DSM-IV-TR (APA, 2000; i.e., Criterion A1: exposure to a potentially traumatic event, and A2: the subjective experience of intense fear, helplessness, or horror). For each potentially traumatic event that is endorsed, follow-up questions assess lifetime frequency and whether participants experienced intense fear, helplessness, or horror in response to the event.

2.2.4. Distressing Events Questionnaire

The Distressing Events Questionnaire (DEQ; Kubany, Leisen, Kaplan, & Kelly, 2000) is a self-report measure used to assess PTSS. The DEQ was designed to assess the three clusters of PTSD symptomatology (i.e., reexperiencing, avoidance, arousal) via 17 items. Response options are rated on a 5-point scale, indicating the extent

to which participants have experienced each symptom in the past month (1 = *almost never* to 5 *almost always*). At T2 and T3, participants were instructed to respond to DEQ items based on the mass shooting. The DEQ has demonstrated good convergent and discriminant validity (Kubany, Leisen, et al., 2000). Additionally, the DEQ has good short-term test-retest reliability and excellent internal consistency (Kubany, Leisen et al., 2000). All DEQ cluster scores evidenced adequate internal consistency across assessment sessions (α values ranging from .82 to .90).

2.2.5. Shooting exposure

At T2, participants completed a 12-item self-report measure of physical exposure adapted from a similar measure used following the shooting at Virginia Polytechnic Institute and State University in April 2007 (Littleton, Grills-Taquechel, & Axsom, 2009). Participants were asked 12 yes/no questions about their exposure to various aspects of the shooting (e.g., saw individuals who had been wounded or killed, heard gunfire, on campus). Scale items were summed to create an exposure total count score, reflecting dose of exposure.

2.2.6. Depression Anxiety Stress Scale-21

The Depression Anxiety Stress Scale-21 (DASS-21; Lovibond & Lovibond, 1995a) is a brief measure of depression (7 items; e.g., I felt that life was meaningless), anxiety (7 items; e.g., I was aware of dryness of my mouth), and stress (7 items; e.g., I found it difficult to relax). The DASS-21 Anxiety and Depression subscales show good reliability (Henry & Crawford, 2005), and good convergent and discriminant validity when compared to other measures of anxiety and depression (e.g., Beck Depression Inventory; Beck & Steer, 1987; Beck Anxiety Inventory; Beck & Steer, 1990; Lovibond & Lovibond, 1995b). Additionally, this measure is appropriate for use in non-clinical samples; Lovibond and Lovibond (1995b) note that the factor structure and performance of items are consistent across clinical and non-clinical samples. All DASS-21 subscale scores evidenced adequate internal consistency across assessment sessions (α values ranging from .77 to .89). To examine the specificity of the temporal relationship between emotional regulation difficulties and PTSS, the DASS-21 subscales were used as indicators of a general distress construct in longitudinal analysis.

3. Results

3.1. Preliminary analyses

Bivariate correlations were calculated in order to examine associations among descriptive statistics (i.e., age, race/ethnicity, physical exposure to the shooting [exposure]) and variables of interest (i.e., DERS subscales, DEQ cluster scores, DASS-21 subscales) for covariate inclusion. Among potential covariates, age was associated with T1 DERS-Nonacceptance ($r = -.08, p < .05$), and race/ethnicity (0 = all others/1 = White and Non-Hispanic) was significantly associated with the following: T1, T2, and T3 DERS-Nonacceptance (T1 $r = .20, p < .001$; T2 $r = .11, p < .01$; $r = .12, p < .01$), T1, T2, and T3 DERS-Goals (T1 $r = .14, p < .001$; T2 $r = .09, p < .05$; T3 $r = .10, p < .05$), T1 and T2 DERS-Clarity (T1 $r = .09, p < .05$; T2 $r = .09, p < .05$), T1 DERS-Strategies ($r = .09, p < .05$), and T1, T2, and T3 DASS-21 Stress (T1 $r = .10, p < .01$; T2 $r = .08, p < .05$, T3 $r = .09, p < .05$). In addition, exposure was positively associated with all T2 and T3 DERS subscales ($ps < .05$), as well as all of the T2 and T3 DEQ cluster scores and DASS-21 subscales ($ps < .001$). Accordingly, age, race/ethnicity, and exposure were included as covariates in longitudinal analysis.

Of the 691 participants from the present study, 20.4% ($n = 141$) had a score of 18 or above on the DEQ at T1. At T2, 49.4% ($n = 341$) of participants had a score of 18 or above on the DEQ. A score of 18

or higher on the DEQ was identified by Kubany, Leisen et al. (2000) as the optimal cut point for diagnosing PTSD in women. Based on responses to the TLEQ, 651 (94.2%) participants endorsed experiencing a potentially traumatic event prior to T1 (Criterion A1) and 571 (82.6%) endorsed experiencing intense fear, helplessness, or horror in response to a traumatic event prior to T1 (Criterion A2). Moreover, participants reported having experienced an average of 4.12 ($SD = 2.93$) different types of potentially traumatic events prior to T1. Of those who experienced at least one potentially traumatic event in their lifetime, the most frequently endorsed potentially traumatic events included the sudden unexpected death of a loved one, a loved one surviving a life threatening illness, natural disaster, witnessing family violence, stalking, and sexual abuse or assault. In response to the mass shooting that occurred between T1 and T2, 622 (90%) participants reported experiencing intense fear, helplessness, or horror.

In regard to shooting exposure, 524 participants (76%) of the T2 sample were on campus at the time of the shooting, 474 (69%) saw emergency responders or police, and 334 (48%) were locked down in a campus building. One-hundred-and-fifty-two (22%) participants saw individuals who had been wounded or killed, 44 (6%) heard gunfire, 24 (3%) were in the building where the shooting took place, 15 (2%) saw the gunman, 11 (2%) saw the gunman firing his weapon, and 3 (.4%) were wounded in the shooting. Two-hundred-and-thirty-five (34%) participants knew someone who had been wounded in the shooting and 159 (23%) knew someone who had died in the shooting.

T2 responders ($n = 691$) were compared to eligible nonresponders ($n = 121$) on demographics (i.e., age, race/ethnicity) and variables measured at T1 (i.e., DERS subscales, DEQ cluster scores, DASS-21 subscales) in order to examine differences due to attrition. In comparison to all other participants, Non-Hispanic White participants were more likely to complete T2, $\chi^2(1, N = 793) = 5.1, p < .05, \phi = .08$. In regard to ERD, those who attrited, in comparison to those who completed T2, reported greater difficulties with emotional clarity (i.e., T1 DERS-Clarity, $t[808] = 2.6, p < .01, d = .18$).

Further, T3 responders ($n = 588$) were compared to T3 nonresponders ($n = 103$) on demographics (i.e., age, race/ethnicity) and T2 variables (i.e., DERS subscales, DEQ cluster scores, DASS-21 subscales) in order to examine differences due to attrition between T2 and T3. In comparison to all other participants, Non-Hispanic White participants were more likely to complete T2, $\chi^2(1, N = 674) = 12.4, p < .01, \phi = .14$. In regard to ERD at T2, those who attrited, in comparison to those who completed T3, reported greater impulse control difficulties (i.e., T2 DERS-Impulse, $t[808] = 2.2, p < .05, d = .16$). With regard to attrition from T1 to T2 and from T2 to T3, those who attrited did not significantly differ from those who completed in terms of PTSS, pre-shooting trauma exposure, and DASS-21 subscales (i.e., depression, anxiety, and stress), or on any of the DERS subscales not mentioned above.

3.2. Structural equation modeling

All models were estimated using Amos software (Version 19; Arbuckle, 2010). A three time-point cross-lagged panel design was used to examine the temporal relationship between ERD and PTSS (see Fig. 1). The ERD construct was modeled using five of the six subscales of the DERS as indicators and the PTSS construct was modeled with the three DEQ cluster scores serving as indicators. Covariates (i.e., age, race/ethnicity, exposure, interval variables) were modeled as manifest variables. To account for the autoregressive effects of measurement occasion (i.e., using the same measures at each time-point), we allowed the error terms for identical indicators to correlate across time (Cole & Maxwell, 2003). To examine the specificity of the temporal relationship between ERD and PTSS, the model was estimated a second time with general distress modeled

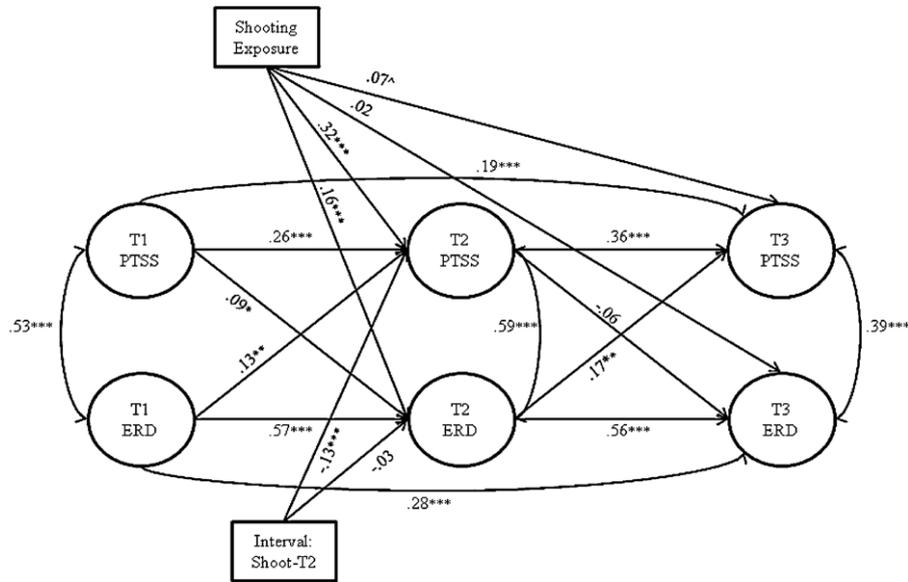


Fig. 1. Cross-lagged panel design for examining longitudinal relations between emotion regulation difficulties (ERD) and posttraumatic stress symptoms (PTSS). Coefficients are standardized. $N = 691$. $\chi^2(276) = 668.84$, $p < .001$, RMSEA = .05, CFI = .96, TLI = .95. T1 = Time 1; T2 = Time 2; T3 = Time 3. Race/ethnicity was included as a covariate in analysis; however, for the sake of clarity, the race/ethnicity variable has been removed from this graphic representation. * $p < .05$. ** $p < .01$. *** $p < .001$.

as a latent construct (i.e., the three subscales of the DASS-21 served as indicators replacing the latent construct of PTSS at all time points (see Fig. 2).

3.2.1. Measurement model with PTSS

Covariances between the variables were freely estimated, with the paths between manifest indicators and error terms constrained to one. Age, and two interval variables (i.e., T1 to T2, T2 to T3) were not associated with any of the latent constructs, and were thus trimmed from the measurement model and the model was reestimated. The DEQ factor loadings for each indicator were adequate with standardized coefficients of .82, .92, and .79 at T1, .84, .89, and .83 at T2, and 80, .92, and .79 at T3 (representing the reexperiencing, avoidance, and hyperarousal DEQ cluster scores, respectively). The DERS factor loadings ranged from poor to

excellent with standardized coefficients of .68, .68, .75, .93, and .56 at T1, .77, .66, .78, .93, and .60 at T2, and .77, .68, .80, .94, and .61 at T3 (representing the Nonacceptance, Goals, Impulse, Strategies, and Clarity subscales of the DERS, respectively). The model fit was adequate, $\chi^2(267, N = 691) = 635.99$, $p < .001$, RMSEA = .05 with a 90% confidence interval of .04 to .05, CFI = .97, and TLI = .95.

3.2.2. Structural model with PTSS

Maximum likelihood estimation was used to test the model. Parameters were estimated using all available data (incomplete data were assumed to be missing at random, and thus included in the parameter estimates). The model fit was adequate, $\chi^2(276, N = 691) = 668.84$, $p < .001$, RMSEA = .05 with a 90% confidence interval of .04–.05, CFI = .96, and TLI = .95. As seen in Fig. 1, T1 ERD significantly predicted T2 PTSS ($\beta = .13$, $p < .01$), with higher levels

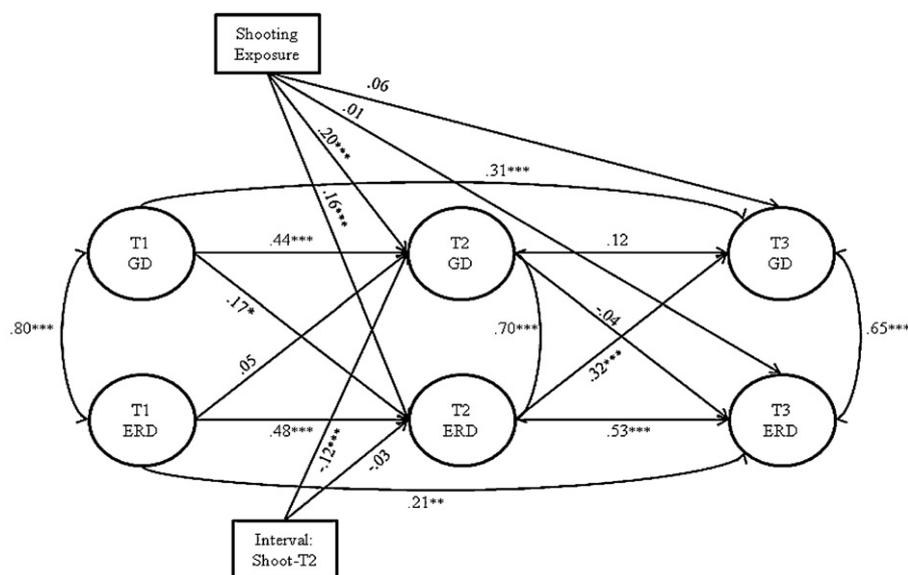


Fig. 2. Cross-lagged panel design for examining longitudinal relations between emotion regulation difficulties (ERD) and general distress (GD). Coefficients are standardized. $N = 691$. $\chi^2(276) = 640.73$, $p < .001$, RMSEA = .04, CFI = .97, TLI = .96. T1 = Time 1; T2 = Time 2; T3 = Time 3. Race/ethnicity was included as a covariate in analysis; however, for the sake of clarity, the race/ethnicity variable has been removed from this graphic representation. * $p < .05$. ** $p < .01$. *** $p < .001$.

of pre-shooting ERD predicting higher levels of post-shooting PTSS. Similarly, higher levels of T1 PTSS were associated with higher levels of T2 ERD ($\beta = .09, p < .05$); however, this directional relationship was not maintained at T3. That is, T2 PTSS did not predict T3 ERD ($\beta = -.06, ns$). Importantly, T2 ERD predicted T3 PTSS above and beyond the effects of T2 PTSS ($\beta = .17, p < .01$), with higher levels of ERD at T2 predicting higher levels PTSS at T3.

T1, T2, and T3 PTSS were all significantly associated, thus demonstrating continued associations between PTSS across time ($ps < .001$). Similarly, T1, T2, and T3 ERD were all significantly associated ($ps < .001$). In addition, exposure was a significant predictor of PTSS at T2 ($\beta = .32, p < .001$), and a marginally significant predictor of PTSS at T3 ($\beta = .07, p = .055$). Thus, greater physical exposure to the shooting was associated with higher PTSS in the acute aftermath of the shooting; however, this effect was greatly reduced over the course of approximately eight months. In a similar vein, the interval between the shooting and T2 significantly predicted T2 PTSS ($\beta = -.13, p < .001$), with a longer duration of time between the shooting and the T2 assessment being associated with lower reported PTSS at T2. The race/ethnicity variable predicted only one of the latent constructs. Non-Hispanic Whites, in comparison to all other participants, reported significantly higher levels of ERD at T1 ($\beta = .12, p < .01$).

3.2.3. Measurement model with general distress

Covariances between the variables were freely estimated, with the paths between manifest indicators and error terms constrained to one. As above, age, and two interval variables (i.e., T1 to T2, T2 to T3) were not associated with any of the latent constructs, and were thus trimmed from the measurement model and the model was reestimated. The DASS factor loadings for each indicator were adequate with standardized coefficients of .81, .73, and .87 at T1, .87, .81, and .89 at T2, and .82, .79, and .89 at T3 (representing the depression, anxiety, and stress subscales of the DASS-21, respectively). The model fit was adequate, $\chi^2 (267, N = 691) = 608.66, p < .001$, RMSEA = .04 with a 90% confidence interval of .04–.05, CFI = .97, and TLI = .96.

3.2.4. Structural model with general distress

Maximum likelihood estimation was used to test the model. Parameters were estimated using all available data (incomplete data were assumed to be missing at random, and thus included in the parameter estimates). The model fit was adequate, $\chi^2 (276, N = 691) = 640.73, p < .001$, RMSEA = .04 with a 90% confidence interval of .04–.05, CFI = .97, and TLI = .96. As seen in Fig. 2, T1 general distress significantly predicted T2 ERD ($\beta = .17, p < .05$), but T1 ERD did not predict general distress at T2 ($\beta = .05, ns$). Thus, higher levels of pre-shooting general distress predicted higher levels of post-shooting ERD above and beyond the effects of pre-shooting ERD. Interestingly, this pattern of effects was reversed from T2 to T3. That is, T2 ERD predicted T3 general distress above and beyond the effects of T2 general distress ($\beta = .32, p < .001$), with higher levels of ERD at T2 predicting higher levels general distress at T3.

4. Discussion

The present study sought to clarify the temporal nature of relations between ERD and PTSS from pre- to post-trauma. We utilized a three time-point prospective investigation with a sample of 691 undergraduate women who were involved in an ongoing longitudinal study at the time of a campus shooting. Further, a cross-lagged panel design was implemented in order to strengthen causal inferences about the directional relations between ERD and PTSS. A reciprocal model, in which ERD and PTSS mutually influenced one another, provided an adequate fit to the data. However, although reciprocal causality was evidenced from pre-shooting

(T1) to post-shooting (T2), with T1 PTSS predicting T2 ERD and T1 ERD predicting T2 PTSS, this directional relationship was not maintained from T2 to T3 (approximately eight months post-shooting). Instead, T2 ERD predicted T3 PTSS, but T2 PTSS failed to predict T3 ERD.

To our knowledge, this is the first time that a temporal relationship between emotion regulation difficulties, trauma, and posttraumatic stress symptoms has been demonstrated in an empirical study. Given the seemingly complex nature of the observed associations, it is important to interpret the results in the light of what is already known about the time-frame of post-traumatic reactions. More specifically, intense emotional reactions to trauma-related cues are common in the aftermath of traumatic events (e.g., Rothbaum et al., 1992; Shalev et al., 1998). This is consistent with the results of the present study in which the endorsement of clinically significant levels of PTSS jumped from 20% at T1 to 49% at T2 (Kubany, Leisen, et al., 2000). In the acute aftermath of the mass shooting, almost half of the sample reported intense post-traumatic reactions. Consistent with the extant literature (Kessler et al., 1995; Rothbaum et al., 1992; Shalev et al., 1998), these reactions diminished for the majority of participants over the course of several months; only 11% of the sample reported clinically significant levels of PTSS at T3. Thus, as previously noted, peri-traumatic emotional responding alone is not sufficient to account for the development of PTSD. The present results suggest that emotion dysregulation in the aftermath of a potentially traumatic event influence's one's ability to reduce PTSS over time. In contrast, one's level of PTSS following a potentially traumatic event does not appear to influence one's ability to regulate emotion over time.

Consistent with the cross-lagged ERD-PTSS model, the reciprocal ERD-general distress model, in which general distress and ERD mutually influence one another, provided an adequate fit to the data. However, reciprocal causality was not evidenced among the directional paths in the model. That is, T1 general distress predicted T2 ERD, but T1 ERD did not predict T2 general distress. Conversely, T2 ERD predicted T3 general distress, but T2 general distress did not predict T3 ERD. In other words, higher levels of pre-shooting distress predicted higher levels of emotion dysregulation in the acute aftermath of the shooting, and higher levels of emotion dysregulation in the acute aftermath of the shooting predicted higher levels of general distress approximately eight months post-shooting. Thus, pre-shooting ERD do not appear to be a risk factor for acute post-shooting general distress. However, acute post-shooting ERD do appear to maintain general distress over time. In fact, not only was T2 ERD a better predictor of T3 general distress than T2 general distress, T2 general distress failed to predict T3 general distress with ERD in the model. Therefore, although ERD do not appear to be a causal risk-factor for acute post-shooting general distress, ERD may be an extremely important etiological agent in the maintenance and exacerbation of general distress.

The results of the present study aid in elucidating the role of emotion regulation in the development of PTSD by demonstrating that ERD prospectively predict posttrauma symptom severity following exposure to a potentially traumatic event when accounting for the effects of existing symptomatology. The study's prospective design provides a substantial contribution to the literature, as existing research is largely cross-sectional and relies on concurrent examinations of emotion dysregulation and PTSS. Findings are consistent with a vulnerability model of PTSD (Elwood, Hahn, Olatunji, & Williams, 2009), in which individuals with higher levels of PTSS and deficits in the ability to regulate affective states demonstrate increased vulnerability to PTSS following trauma exposure. Moreover, results suggest a degree of specificity in the ERD-PTSS relationship. That is, ERD, measured prior to shooting exposure, prospectively predicted PTSS, but not general distress. This is consistent with the current diagnostic classification of PTSD

in the DSM-IV which indicates that the subjective experience of intense peritraumatic fear, helplessness, or horror in response to a traumatic stressor is a vital etiological component (APA, 2000).

Theoretical perspectives suggest this vulnerability is associated with appraisal of threat and peritraumatic emotional response during a traumatic experience and use of avoidant coping strategies in the aftermath of trauma exposure (Bovin & Marx, 2011; Olff, Langeland, & Gersons, 2005). Consistent with these theories, individuals with higher PTSS have been shown to demonstrate an attentional bias to threat (e.g., Bardeen & Orcutt, 2011), which recent evidence suggests is closely related to enhanced fear responding in the context of stress exposure (Fani et al., 2012). Additionally, greater threat appraisal and increased affective reactivity when exposed to trauma has been shown to be a risk factor for the development of PTSD (e.g., Bovin & Marx, 2011; Dunmore, Clark, & Ehlers, 1999).

Deficits in emotion regulation also may increase risk for PTSD via similar mechanisms, with the greatest vulnerability being among those with existing PTSS and substantial deficits in emotion regulation. Previous research suggests that physiological processes (e.g., difficulty in one's ability to control autonomic nervous system responses) may be a key element in maladaptive responding when exposed to a potentially traumatic event. Olff et al. (2005) propose a useful conceptual model of PTSD that implicates appraisal of threat, coping style, and associated neuroendocrine responses in the development and maintenance of PTSD. It is likely that a diminished emotion regulation capacity, whether due to existing deficits in emotion regulation or as a result of PTSS, impairs the ability for vulnerable trauma-exposed individuals to access effective flexible coping strategies both in the immediate context and aftermath of trauma exposure. These deficits may be associated with chronic HPA-axis dysregulation and sympathetic nervous system hyper-reactivity (Olff et al., 2005). Subsequent cognitive and physiological disruptions in emotional responding, paired with a reliance on avoidant coping strategies, likely function to maintain PTSD across time (Frewen & Lanius, 2006).

Although the present study advances our understanding of the relationship between difficulties with emotion regulation and symptoms of posttraumatic stress, limitations should be acknowledged. First, the findings presented may not generalize to symptoms consistent with a PTSD diagnosis. A portion of the sample may not have had a level of exposure to the mass shooting consistent with current definitions of a traumatic event, thus precluding a diagnosis of PTSD. Despite this limitation, existing research suggests that many individuals without direct exposure to mass trauma experience significant distress following these events (e.g., North, Smith, & Spitznagel, 1994; Schwarz & Kowalski, 1991). In addition, Criterion A2 was endorsed by 90% of the present sample, which suggests that the shooting had a sizeable psychological impact. There was also a relatively short latency between the occurrence of the shooting and the assessment of associated symptoms, with a large portion of the sample completing the post-shooting assessment within 30 days. Thus, the findings presented are better viewed as acute reactions to the shooting rather than symptoms of PTSD, and additional research among clinical samples would be of benefit. Given evidence that PTSD appears to have a dimensional structure where differences in symptomatology reflect variations along a continuum of posttraumatic stress responses, rather than representing a qualitatively distinct syndrome (e.g., Broman-Fulks et al., 2006; Ruscio, Ruscio, & Keane, 2002), the present investigation is likely of benefit even in light of existing limitations.

Because the sample included only female undergraduate students originally enrolled in an introductory psychology course, the ability to generalize the current findings to other groups is limited. Existing evidence suggests differences in prevalence of PTSD symptoms between females and males (e.g., Norris, Foster,

& Weisshaar, 2002; Tolin & Foa, 2006) and mechanisms for such differences are not well understood. Generalizability may also be limited as a result of the single traumatic experience of focus in the present study. All participants in the study reported PTSS in reference to the mass shooting, thus, results are limited to reactions to that particular index event. Additionally, because ERD was modeled as a latent construct, the individual domains of ERD, as identified by Gratz and Roemer (2004), were not examined as individual predictors of PTSS. In future studies, researchers may wish to examine prospective differential relations between the ERD domains and PTSS in order to identify the relative importance of each domain as a potential treatment target.

Taken together, the methodological issues of the present study highlight the importance of additional research designed to replicate and extend the results. The availability of research capable of identifying potential risk factors for PTSS is limited because of the challenges associated with conducting prospective, trauma-focused research. Thus, despite the disadvantages of the present study, the findings offer substantial contribution to the literature examining the relationships between difficulties with emotion regulation and PTSS. Given the reliance on cross-sectional designs and the lack of longitudinal research in this area, the current prospective design and availability of pretrauma assessments are unique strengths of the study.

Our findings may have important clinical implications. The present results may inform decisions regarding treatment of PTSD in clinical settings. Because existing PTSS appear to be associated with an increased vulnerability to exacerbations in symptomatology and ERD in the aftermath of trauma exposure, it may be most beneficial to target PTSD symptoms directly in treatment. There is evidence to suggest that exposure therapy for PTSD (i.e., Prolonged Exposure; Foa, Hembree, & Rothbaum, 2007) improves individuals' sense of self competence and control over negative outcomes and results in habituation to trauma-related stimuli (Foa & Rauch, 2004), which may be associated with concurrent improvement in emotion regulation capacity. The potential for exposure therapy to improve emotion regulation is further supported by evidence that reduction in novelty and increased competence/control have been linked with psychophysiological responses (Abelson, Liberzon, & Young, 2005; Lang, Melamed, & Hart, 1970; Olff et al., 2005).

In contrast, additional research has suggested that exposure therapy may not adequately address difficulties in regulating emotion, particularly among individuals with PTSD who have greater emotion regulation deficits (Cloitre et al., 2010). There is conflicting evidence as to whether these individuals may be less likely to show improvement following exposure-based PTSD treatment due to high rates of treatment dropout (Cloitre et al., 2005; McDonagh, Friedman, McHugo, Ford, S, & Descamps, 2005), exacerbation of PTSD symptoms during exposure (Pitman et al., 1991; Foa, Zoellner, Feeny, Hembree, & Alvarez-Conrad, 2002), or worsening of PTSD at the end of treatment (Devilley & Foa, 2001). Although additional research is needed to better understand the precise role of emotion regulation in the treatment of PTSD, therapies that specifically address regulation of emotion (e.g., Dialectical Behavior Therapy; Linehan, Schmidt, Dimeff, Craft, Kanter, & Comtois, 1999; Skills Training in Affect and Interpersonal Regulation: Cloitre, Koenen, Cohen, & Han, 2002), in combination with exposure therapy, may enhance treatment effectiveness for PTSD sufferers with more severe emotion regulation difficulties.

Acknowledgements

This research was funded by grants to the third author from the Joyce Foundation, the National Institute for Child and Human

Development (1R15HD049907-01A1), and the National Institute of Mental Health (5R21MH085436-02).

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