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Trauma Symptoms and Deliberate Self-Harm Among Sexual Violence Survivors: Examining State Emotion Regulation and Reactivity as Dual Mechanisms

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Objective: Posttraumatic stress disorder (PTSD) symptoms have been associated with elevated rates of deliberate self-harm, including among women who have experienced sexual violence (SV); however, processes underlying this association have not been widely examined. Because a common function of deliberate self-harm is to reduce negative internal states, SV survivors may use self-harm to cope with impairments in broader affective processes associated with PTSD symptoms. To test this hypothesis, the present study examined the role of two aspects of emotional responding (i.e., state emotional reactivity and emotion dysregulation) as mechanisms between greater PTSD symptoms and risk for future deliberate self-harm among SV survivors. ***Method:*** Participants were 140 community women with a history of SV who completed two waves of data collection. At baseline, participants reported on their PTSD symptoms, as well as state emotional reactivity and state emotion dysregulation following a standardized laboratory stressor task (i.e., the Paced Auditory Serial Addition Task [PASAT-C]). Participants then completed a self-report measure of deliberate self-harm 4 months later. ***Results:*** Results from a parallel mediation analysis indicated that greater state emotion dysregulation, but not state emotional reactivity, mediated prospective associations between more severe PTSD symptoms at baseline and greater risk for deliberate self-harm 4-months later. ***Conclusions:*** Applied to the context of survivors' daily lives, these findings underscore the importance of deficits in emotion regulation during times of distress in predicting risk for later deliberate self-harm.

Keywords: self-injurious behavior, victimization, posttraumatic stress disorder, emotional regulation, negative emotions

Deliberate self-harm refers to the intentional, direct, self-inflicted destruction of body tissue without suicidal intent and for purposes not socially sanctioned (Chapman et al., 2006; Gratz, 2001). Deliberate self-harm includes behaviors such as cutting, burning, and self-hitting (Klonsky, 2007). In addition to the clinical and functional impairment associated with this behavior (Daukantaitė et al., 2021), deliberate self-harm is the strongest predictor of suicide attempts (Franklin et al., 2017), highlighting the clinical importance of understanding mechanisms underlying this behavior. Although several motives for deliberate self-harm have been identified, the most well-established motive is the avoidance or regulation of unwanted internal states (Chapman et al., 2006; Klonsky, 2007; Taylor et al., 2018). Indeed, a prominent model of deliberate self-harm, the experiential avoidance model (Chapman et al., 2006), posits that deliberate self-harm functions primarily to avoid or escape aversive or unwanted internal states that the individual is unwilling or unable to regulate in other (more adaptive) ways. Consistent with this theory, a recent meta-analysis indicated that 66%–81% of participants reported using deliberate self-harm to manage or change internal states (e.g., thoughts, feelings, bodily sensations), with the most common motive reflecting a desire to escape or avoid unwanted internal states (Taylor et al., 2018). Thus, the present study will examine risk factors for deliberate self-harm that are grounded in theoretical models of emotional avoidance—specifically, posttraumatic stress disorder (PTSD) symptoms and aspects of emotional responding.

PTSD Symptoms and Deliberate Self-Harm

Consistent with the theory that deliberate self-harm functions primarily to avoid or escape unwanted internal states (Chapman et al., 2006), rates of deliberate self-harm are elevated among

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individuals who experience PTSD symptoms following trauma exposure (Bentley et al., 2015; Smith et al., 2014). PTSD is characterized by several symptom clusters, including avoidance of internal and external stimuli, intrusions (e.g., unwanted memories, nightmares), hyperarousal, and negative alterations in cognition and mood (e.g., self-blame, negative affect [NA]; American Psychiatric Association, 2013). Because avoidance is a central symptom of PTSD, trauma survivors may be more likely to use avoidant strategies, such as deliberate self-harm, to escape the negative thoughts and emotions associated with PTSD symptoms (Chapman et al., 2006; Smith et al., 2014).

Although elevated rates of deliberate self-harm have been found across samples of trauma survivors (Dixon-Gordon et al., 2014; Holliday et al., 2018; Smith et al., 2014), one type of trauma that is particularly relevant to deliberate self-harm is sexual violence (SV) victimization, which includes both childhood sexual abuse and sexual assault in adolescence and adulthood. Compared to other types of trauma, SV confers the greatest risk for both PTSD (childhood sexual abuse, Bornovalova et al., 2011; adult SV, Kessler et al., 2017) and current deliberate self-harm (Jaquier et al., 2013). Moreover, women are disproportionately affected by SV across the lifespan (Breiding et al., 2014) and engage in deliberate self-harm at higher rates than men, with young women being at particularly high risk (Breiding et al., 2014; Bresin & Schoenleber, 2015). Finally, providing further support for the relevance of SV-related PTSD symptoms in particular to deliberate self-harm, greater PTSD symptoms have been found to relate to lifetime deliberate self-harm among veterans with military sexual trauma (Holliday et al., 2018) and greater SV-related PTSD symptoms at baseline have been found to predict deliberate self-harm 5 years later among women (Nada-Raja & Skegg, 2011).

Aspects of Emotional Responding as Intermediary Mechanisms

In addition to the likelihood that women who have experienced SV engage in deliberate self-harm specifically to avoid PTSD symptoms, it is also possible that the link between PTSD symptoms and deliberate self-harm occurs indirectly as a result of known consequences of PTSD on broader affective processes. Consistent with the experiential avoidance model (Chapman et al., 2006), emotional difficulties stemming from SV-related PTSD symptoms could exacerbate urges to engage in deliberate self-harm for emotion regulation related reasons. In particular, two domains of emotional responding commonly influenced by PTSD symptoms, emotional reactivity and emotion dysregulation (Seligowski et al., 2015; Vujanovic et al., 2013), may be especially relevant to deliberate self-harm (Chapman et al., 2006).

Emotional Reactivity

Emotional reactivity refers to the amplitude of one's emotional responses to internal and external stimuli (Rothbart & Derryberry, 1981), with greater emotional reactivity reflecting a larger increase in elicited emotion following a stimulus, such as a stressful event. Heightened emotional reactivity is prevalent among those with PTSD symptoms (Badour & Feldner, 2013) and could lead to deliberate self-harm as a method of reducing unpleasant and overwhelming levels of intense negative affect (e.g., anger, fear, arousal).

Supporting the potential intermediary role of emotional reactivity, prior empirical work suggests that emotional reactivity is closely related to both PTSD symptoms and deliberate self-harm. Compared to women with less severe PTSD symptoms, those with more severe PTSD symptoms exhibit greater responses to negative emotional stimuli (Vujanovic et al., 2013). Similarly, trauma survivors (e.g., women with a history of physical or sexual assault, military veterans) who endorse greater PTSD symptoms report greater subjective anxiety following trauma imagery tasks (Badour & Feldner, 2013) and greater frustration and irritability following a laboratory stressor (Brown et al., 2018). These self-report findings are corroborated by results from studies using physiological measures, which indicated interpersonal trauma survivors with greater PTSD symptoms exhibited greater arousal on measures of heart rate and skin conductance following trauma imagery tasks (Badour & Feldner, 2013; McDonagh-Coyle et al., 2001). Although more research is needed within samples of SV survivors, these findings converge to suggest that PTSD symptoms are related to greater emotional reactivity.

Notably, there is also some evidence that emotional reactivity is related to greater risk for deliberate self-harm. Some work suggests that deliberate self-harm is associated with greater trait negative emotional reactivity (Glenn et al., 2011), as well as physiological arousal (i.e., skin conductance) following a laboratory stressor (Nock & Mendes, 2008). Importantly, however, other research has not supported the link between emotional reactivity and deliberate self-harm. Compared to participants without a history of deliberate self-harm, participants with prior self-harm did not report greater negative emotional reactivity in general (Gratz, 2006) or in response to sad film clips (Davis et al., 2014). Likewise, participants' startle response (Glenn et al., 2011) and amygdala activation (Davis et al., 2014) while viewing unpleasant images did not differ in relation to one's history of deliberate self-harm. Thus, more research is needed to clarify the nature and strength of any potential links between emotional reactivity and deliberate self-harm.

Emotion Dysregulation

Along with changes in the magnitude of emotional reactions to internal and external stimuli (i.e., emotional reactivity), survivors with PTSD symptoms often face challenges *responding* effectively to their emotions (i.e., emotion dysregulation) and may engage in deliberate self-harm as a compensatory strategy. Emotion dysregulation is a multifaceted construct that refers to difficulties understanding, accepting, and modulating emotions (Gratz & Roemer, 2008). Although these difficulties can be more pronounced during times of intense negative affect (Selby & Joiner, 2013), and more reactive emotions can be more difficult to regulate effectively (Gross & Jazaieri, 2014; Lynch et al., 2001), how one responds to one's emotions is considered distinct from the nature or quality of those emotions, with individuals varying in the ability to respond adaptively to emotions regardless of their intensity or reactivity (Davis et al., 2014; Gratz & Roemer, 2008; Gross & Jazaieri, 2014). In this way, emotional reactivity and emotion dysregulation are distinct constructs that capture separate aspects of the emotional response process via the elicitation of and response to emotions.

A considerable literature base indicates greater PTSD symptoms are associated with greater emotion dysregulation, including meta-analytic findings (Seligowski et al., 2015). Emotion dysregulation is

thought to be exacerbated among SV survivors because of the uncontrollable and unexpected nature of SV, as well as the increased demands and obstacles associated with regulating chronic PTSD symptoms (Walsh et al., 2012). Moreover, recent work examining in vivo emotion regulation strategies found that undergraduate trauma survivors (40% of whom reported SV) with greater PTSD symptoms are more likely to employ maladaptive emotion regulation strategies in response to negative stimuli (Hannan & Orcutt, 2020).

In addition to known associations with PTSD symptoms, emotion dysregulation has been linked to deliberate self-harm (Chapman et al., 2006; Gratz, 2003; Gratz & Roemer, 2008; Taylor et al., 2018). Women who frequently engage in deliberate self-harm report greater trait emotion dysregulation than those without prior self-harm (Andover & Morris, 2014; Gratz & Roemer, 2008). Among community men and women, deliberate self-harm is also related to greater difficulties executing putatively adaptive emotion regulation strategies (i.e., reappraisal) and less success regulating negative emotions in the moment (Davis et al., 2014).

The Present Study

Although this prior literature suggests greater emotional reactivity and emotion dysregulation arising from PTSD symptoms may increase risk for deliberate self-harm among SV survivors, the ability to test this model has been hampered by methodological factors common across most of the research in this area. First, most studies have used dispositional measures that ask participants to report on how they typically experience and respond to emotions. However, stressors may vary greatly from person-to-person and emotional responses may be context-driven. Further, dispositional self-report measures of emotional responding may be particularly problematic in the context of PTSD, where affective processes are likely to be state- and context-dependent (e.g., SV-related reminders and situations) and symptoms such as avoidance and impaired memory may make it difficult for survivors to accurately recall and report on past affective experiences. To overcome these limitations, this study used a validated laboratory stressor (Paced Auditory Serial Addition Task [PASAT-C]; Lejuez et al., 2003) found to induce anger and anxiety spectrum emotions to assess state emotional reactivity and regulation difficulties. In addition, most prior work has used cross-sectional designs, which inform our knowledge of general associations between these constructs but do not permit conclusions about temporal relations. To address this limitation, this study used a prospective design and autoregressive controls to isolate the extent to which PTSD symptoms and emotional responding relate to engagement in future deliberate self-harm. Lastly, despite findings supporting independent linkages among these constructs, prior work has not yet tested an integrated model wherein greater emotional reactivity and emotion dysregulation are examined as mechanisms for the relation between greater PTSD symptoms and engagement in future deliberate self-harm among SV survivors. The present study tests this model, predicting:

1. Among SV survivors, greater PTSD symptoms at baseline will have an indirect association with deliberate self-harm 4 months later through greater state emotional reactivity in response to a laboratory stressor.
2. Among SV survivors, greater PTSD symptoms at baseline will have an indirect association with deliberate self-harm 4 months later through greater state emotion dysregulation following a laboratory stressor.

Method

Participants

Participants were 140 community women recruited at four sites in the U.S. (22.1% from Jackson, Mississippi; 34.3% from Lincoln, Nebraska; 18.6% from Omaha, Nebraska; and 25% from Oxford, Ohio) to participate in a longitudinal study on women's sexual revictimization (Watkins et al., 2015). Although prior studies from this larger data set have examined similar constructs (e.g., SV-related PTSD, Jaffe et al., 2017; McConnell et al., 2020; emotion regulation, Dixon-Gordon et al., 2015), the present study is unique in its focus on how SV-related PTSD and state emotion processes are related to deliberate self-harm. The current sample was drawn from the full sample of 491 women who completed Wave 1 (Watkins et al., 2015). Women who were between the ages of 18–25 and who lived in one of the four site locations were eligible to participate in the larger study. To be included in the current analyses, participants had to report unwanted sexual activity (i.e., rape or other unwanted sexual experience) on the life events checklist (LEC; Gray et al., 2004) and identify it as their "most traumatic" event. Participants' ages at baseline ranged from 18 to 25 ($M = 21.7$, $SD = 2.2$). The racial/ethnic composition of the sample was 69.2% White, 27.1% Black/African American, 6.4% Latina/Hispanic, 6.4% Asian, and 3.6% American Indian or Alaskan Native (percentages exceed 100% because participants could select multiple categories). Participants primarily identified as heterosexual (78.6%), with the remaining participants identifying as bisexual (17.1%), lesbian (2.9%), or questioning (0.7%). In the current sample, 57.9% of participants were full or part-time students. The most common income bracket was under $10,000 (48.6%), and most participants (87.9%) reported earning less than $49,999 per year.

Procedure

Institutional review boards (IRB) at all four sites approved study procedures. Participants were recruited through community advertisements for a study on "women's life experiences" at each of the sites, as well as recruitment letters that were sent to randomly selected women between the ages of 18 and 25 who lived in one of the study site locations and were identified through Survey Sampling International (SSI). Recruitment methods were identical across sites. All participants provided written informed consent. Participants were compensated $75 for the baseline assessment and $25 for the Wave 2 assessment 4 months later.

After providing written informed consent, participants completed the baseline session, including online questionnaires and laboratory tasks. Following completion of online questionnaires in Qualtrics on a laboratory computer, participants received standardized instructions for completing the laboratory stressor (described below). To ensure participant engagement with the task, participants were told that their performance on the task would determine the amount of compensation they received. Once they confirmed their understanding of these instructions, they completed the stressor (providing ratings of

their current negative affect immediately before the stressor began and following the third [most difficult] level of the task). Following completion of the laboratory stressor, participants immediately completed a measure of state emotion dysregulation. After completing this measure, participants were informed that their performance on the laboratory stressor qualified them for full compensation and all participants received the full $75 for completion of the baseline assessment. To ensure that participants did not leave the session in a state of elevated distress, participants reported their current level of distress on a scale from 1 (*no distress*) to 7 (*extremely distressed*) at the beginning and end of the baseline session. If participants reported at least a two-point increase in distress, a research assistant discussed distress tolerance strategies with the participant and stayed with them until their distress returned to prestudy levels. At the Wave 2 assessment (which occurred approximately 4 months after the initial assessment), participants completed online self-report questionnaires in Qualtrics.

Measures

Life Events Checklist (Gray et al., 2004)

The LEC is a 16-item measure that assesses the lifetime occurrence of potentially traumatic events, including SV. Participants responded either "*yes*" or "*no*" to indicate whether they had experienced each event. Participants were also allowed to identify and describe any other potentially traumatic event that was not listed. Participants were then asked to indicate which of the events they identified was "most traumatic" and whether they felt fear, helplessness, or horror at the time of that event. They were asked to complete the measure of PTSD symptoms in reference to that event. Prior work supports the adequate test-retest reliability of the LEC over a 1–2-week period (range = 5–14 days) at the scale and item level, as well as convergence with PTSD symptom measures (Gray et al., 2004). All 140 participants endorsed either "rape (forced oral, anal, or vaginal penetration)" or "other unwanted or uncomfortable sexual experience" as their "most traumatic" event. Both items were included because many women do not label unwanted sexual experiences as rape (Wilson & Miller, 2016).

PTSD Checklist—Civilian Version (Weathers et al., 1993)

The PTSD Checklist—Civilian Version (PCL-C) is a 17-item measure that assesses the degree to which participants experienced Diagnostic and Statistical Manual of Mental Disorders, fourth edition (DSM-IV), PTSD symptoms in the past month. The PCL-C was used in the present study because data were collected prior to the development of the PTSD Checklist for DSM-5 (PCL-5; Blevins et al., 2015). Participants completed the PCL-C in relation to the Criterion A SV event they identified on the LEC. Participants rated the extent to which each PTSD symptom bothered them in the past month on a 5-point scale ranging from 1 (*not at all*) to 5 (*extremely*). A total score representing PTSD symptom severity was calculated by summing all 17 items. Prior work provides support for the test-retest reliability of the PCL-C over 1–2 weeks, as well as its convergent validity with other trauma symptom measures and structured PTSD interviews (Conybeare et al., 2012; Weathers et al., 1993; Wilkins et al., 2011). Internal consistency in prior work (αs > .75; Wilkins et al., 2011) and in the current sample was good (α = .95).

Laboratory Assessment of State Emotional Reactivity and Emotion Dysregulation

To examine state emotional responding, participants first completed the computerized version of the PASAT-C (Lejuez et al., 2003), a laboratory stressor. In support of the construct validity of the PASAT-C as a laboratory stressor, this task has been shown to induce emotional distress in the form of anxiety, anger, frustration, and irritability among clinical and nonclinical samples (Bornovalova et al., 2008; Lejuez et al., 2003). During this task, numbers are flashed sequentially on a computer screen and participants are instructed to sum the most recent number with the previous number (using the computer mouse to click on the answer). Participants must then ignore the sum and add the next number to the most recently presented number. One point is earned for each correct answer. If an incorrect answer is provided (or participants fail to provide an answer before the next number is presented), an explosion sound is played and no points are earned. The version used here consisted of four levels, the first three of which had increasingly shorter latencies between number presentations. Because the correct answer must be provided prior to the presentation of the next number in order to obtain a point, difficulty increases as latencies decrease. The third and fourth levels have the same 1-s latency between number presentations and are designed to make it virtually impossible for participants to provide a correct answer prior to the presentation of the next number, thereby inducing distress.

To assess state emotional reactivity in response to the laboratory stressor, participants completed the negative affect (NA) scale of the Positive and Negative Affect Schedule (PANAS-NA; Watson et al., 1988) immediately before the stressor and following completion of the third (most difficult) level of the task. Participants were asked to rate the extent to which they were experiencing each of 10 forms of NA (e.g., "distressed") right now. Responses were provided on a 5-point scale ranging from 1 (*very slightly/not at all*) to 5 (*extremely*). As in prior work (Winward et al., 2014), emotional reactivity in response to the task was calculated by subtracting the pretask NA sum score from the peak-task NA sum score. Pretask and peak-task NA scores were correlated but not collinear (r = .66, p < .001). Furthermore, and providing support for the use of this laboratory stressor to elicit emotional reactivity, participants' peak-task NA (M = 21.42, SD = 8.18) was significantly higher than their pretask NA (M = 14.47, SD = 4.74), $t(138) = 13.29$, $p < .001$. The PANAS-NA scale, including with momentary rating instructions, has been found to be sensitive to change (Watson et al., 1988), and change scores have been widely used as a measure of emotional reactivity, both in response to the laboratory stressor used in this study (Winward et al., 2014) and among samples of trauma survivors (Wisco et al., 2015). The PANAS-NA scale demonstrated good internal consistency in this sample at both assessments (αs ≥ .83).

Next, to assess state emotion dysregulation in response to the laboratory stressor, participants completed the State Difficulties in Emotion Regulation Scale (S-DERS; Lavender et al., 2017) immediately following the stressor. The S-DERS is a 21-item measure of state emotion regulation difficulties (e.g., "My emotions feel out of control"). Participants are asked to rate the extent to which each item applies to them "right now" using a 5-point scale ranging from 1 (*not at all*) to 5 (*completely*). The S-DERS includes four subscales: awareness (5 items), clarity (2 items), modulate (7 items), and nonacceptance (7 items); items are summed to create an overall

index of state emotion dysregulation. Evidence supporting the construct and predictive validity of the S-DERS in relation to both trait- and state-based measures of emotional responding and related constructs (e.g., substance use) has been provided (Lavender et al., 2017). Internal consistency in prior work ($\alpha = .86$; Lavender et al., 2017) and in this sample ($\alpha = .85$) was good.

Deliberate Self-Harm Inventory (Gratz, 2001)

The Deliberate Self-Harm Inventory (DSHI) is a 17-item measure assessing the frequency, severity, duration, and type of deliberate self-harm across specific timeframes using behaviorally specific items. Participants are asked to indicate whether they intentionally engaged in 16 deliberate self-harm behaviors without intending to kill themselves (e.g., "burned yourself with a cigarette") by responding "*yes*" or "*no*." The last item assesses whether participants have done anything else to harm themselves physically that was not previously assessed; responses to this item are then coded for whether they meet the definition of deliberate self-harm. Participants were asked to report on lifetime deliberate self-harm at baseline and past 4-month deliberate self-harm at Wave 2. For the purposes of this study, two dichotomous self-harm variables were computed, reflecting the presence versus absence of deliberate self-harm during one's lifetime (for the baseline assessment) or in the past 4 months (for the Wave 2 assessment), with "0" indicating the absence of this behavior and "1" indicating at least one instance of deliberate self-harm. The DSHI has demonstrated high internal consistency ($\alpha =$ .75–.82 in prior validation studies), adequate test-retest reliability over periods ranging from 2 weeks to 5 months, and adequate construct, discriminant, and convergent validity among undergraduate student, community adult, and patient samples (Fliege et al., 2006; Gratz, 2001; Gratz et al., 2014). Internal consistency was adequate in the current sample at baseline ($\alpha = .74$) and 4-month follow-up ($\alpha = .69$).

Data Analytic Plan

Longitudinal data were analyzed using Statistical Package for the Social Sciences (SPSS) Version 27 for descriptive statistics and Mplus Version 8.4 to test our primary mediation model (Muthén & Muthén, 1998–2017). Due to attrition across repeated measurements ($n = 21$, or 15% of participants, did not complete Wave 2), there were missing data for scores of deliberate self-harm at the Wave 2 assessment. The covariance coverage for the primary mediation model ranged from 84% to 100%. Missing data were addressed using maximum likelihood (ML) estimation (Enders, 2010). Thus, all 140 participants were included in analyses. Given our use of a categorical binary outcome variable (i.e., presence or absence of deliberate self-harm) and ML, a logit link function generalized linear model (GLiM) and Monte Carlo numerical integration were applied to address violations of assumptions and better capture nonnormality. When using a logit link, the coefficient represents the log-odds.

First, descriptive statistics were conducted in SPSS. Next, to test the role of state emotion dysregulation and emotional reactivity in accounting for the relation between SV-related PTSD symptoms and later deliberate self-harm, a parallel mediation model was tested and the hypothesized indirect effects were estimated. Given our use of prospective data, baseline deliberate self-harm was included as an autoregressive control when predicting Wave 2 self-harm. Further, to isolate the impact of prior SV on future deliberate self-harm, the presence or absence of any new SV that occurred between the Wave 1 and Wave 2 assessments was included as a covariate when predicting Wave 2 self-harm. All exogenous variables (i.e., baseline PTSD symptoms and deliberate self-harm, Wave 2 SV) were correlated in the model. Further, baseline state emotion dysregulation, emotional reactivity, and deliberate self-harm were correlated. Bias-corrected bootstrapping with 5,000 samples was used to obtain 95% confidence intervals (CI) for the indirect effects. If the CI does not include zero, an indirect effect is supported.

Power necessary for testing our hypotheses was calculated in G*Power 3 (Faul et al., 2007) and indicated a sample of 43 participants was needed for adequate power (0.80, $p < .05$) to detect the effects of five independent variables (PTSD symptoms, emotional reactivity, emotion dysregulation, new SV, and baseline deliberate self-harm), estimating a medium effect size ($f^2 = 0.15$) based on prior literature (see Badour & Feldner, 2013; Glenn et al., 2011; Gratz & Roemer, 2008; Nock & Mendes, 2008; Seligowski et al., 2015). For a more conservative test, we also tested power to detect smaller effects ($f^2 = 0.05$) with five independent variables, which indicated a necessary sample of 126 participants. Thus, our sample of 140 participants should be sufficient to detect small to moderate effects, which are consistent with prior identified effect sizes.

Results

Descriptive Statistics

Descriptive statistics for the primary variables are reported in Table 1. All variables were within acceptable ranges for skewness and kurtosis (Kline, 2015). The mean PCL-C score was 35.5 ($SD =$ 16.5), suggesting the average participant was experiencing clinically significant PTSD symptoms above the cut point for a probable PTSD diagnosis (i.e., score of 33; Weathers et al., 1993). Indeed, 47.1% of participants reported a score of 33 or greater on the PCL-C. Participants reported a mean NA score of 14.47 ($SD = 4.74$) before the laboratory stressor and 21.42 ($SD = 8.18$) following the most difficult level of the task. Consistent with this, mean scores of state emotional reactivity ($M = 7.0$, $SD = 6.2$) and state emotion dysregulation ($M = 38.4$, $SD = 11.9$) in response to the laboratory stressor suggest that the task effectively induced emotional distress (e.g., participants reported an average NA increase of 7 points in response to the task) and difficulties regulating emotions in the moment (with the mean score of 38.4 on the S-DERS suggesting mild difficulties in emotion regulation). Finally, 50% of participants reported lifetime deliberate self-harm at Wave 1, 16% reported deliberate self-harm in the past 4 months at Wave 2, and 23.8% reported new SV at Wave 2.

Bivariate correlations are reported in Table 1. Greater baseline PTSD symptoms were significantly associated with greater state emotion dysregulation but not state emotional reactivity, Wave 1 or Wave 2 deliberate self-harm, or Wave 2 SV. Greater state emotion dysregulation was significantly related to greater state emotional reactivity and Wave 2 deliberate self-harm, but not Wave 1 deliberate self-harm or Wave 2 SV. State emotional reactivity was not significantly associated with Wave 1 or Wave 2 deliberate self-harm

Table 1
Descriptive Statistics and Bivariate Correlations

Variable	Wave	*n*	*M*	*SD*	Range	1	2	3	4	5	6
1. PTSD symptoms	1	140	35.5	16.5	17–84	—	.16	.53***	.10	.14	.01
2. State emotional reactivity	1	139	7.0	6.2	−3–32		—	.36***	.09	.01	−.10
3. State emotion dysregulation	1	139	38.4	11.9	21–81			—	.06	.38***	−.13
			n endorsed	%							
4. Deliberate self-harm	1	140	70	50%	0–1				—	.21*	.05
5. Deliberate self-harm	2	119	19	16%	0–1					—	.03
6. Sexual victimization	2	118	28	23.7%	0–1						—

Note. PTSD = posttraumatic stress disorder.
* $p < 05$. ** $p < .01$. *** $p < .001$.

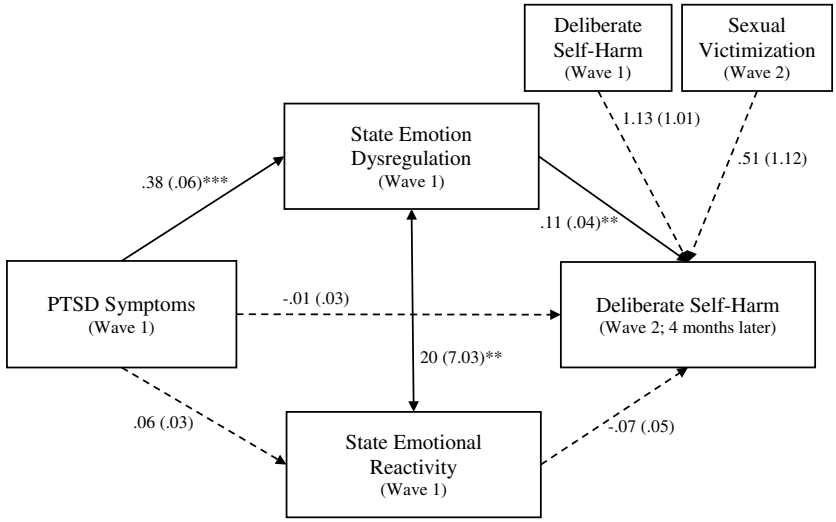
or Wave 2 SV. Finally, Wave 1 and Wave 2 deliberate self-harm were positively associated.

Given our use of prospective data, we conducted attrition analyses to determine whether participants who completed the Wave 2 assessment ($n = 119$) differed significantly from those who did not ($n = 21$). Participants who did not complete the Wave 2 assessment reported greater PTSD symptoms $t(138) = 4.13$, $p < .001$ and greater state emotion dysregulation $t(137) = 2.50$, $p < .02$ at Wave 1 than those who did complete Wave 2. However, emotional reactivity $t(137) = -0.96$, $p = .34$ and deliberate self-harm $t(138) = -0.24$, $p = .82$ did not differ across participants who did and did not complete Wave 2. Because only deliberate self-harm was assessed at Wave 2 and these data were missing completely at random, we retained all 140 participants who completed Wave 1 in analyses using ML estimation.

Parallel Mediation Model

Because fit indices are not available for ML with Monte Carlo integration, we were unable to examine global model fit. As shown in Figure 1, greater PTSD symptoms were not significantly associated with greater state emotional reactivity, $b = 0.06$ ($SE = 0.03$), $p = .09$, $\beta = .15$, contrary to expectations. Further, greater state emotional reactivity did not predict the log-odds of deliberate self-harm at Wave 2 when all other variables were in the model, $b = -0.07$ ($SE = 0.05$), $p = .19$, $\beta = -.20$. However, as expected,

Figure 1
Path Analyses for Parallel Mediation Model



Note. PTSD = posttraumatic stress disorder; SV = sexual violence. Unstandardized coefficients (*SE*) are reported for each path. Wave 1 deliberate self-harm and any new SV during the 4-month period were controlled for when predicting Wave 2 deliberate self-harm. The indirect relation from Wave 1 PTSD symptoms to Wave 2 deliberate self-harm via state emotion dysregulation was significant when accounting for covariates, $b = .04$, 95% CI [.017, .067], $\beta = .30$; the indirect relation through state emotional reactivity when accounting for covariates was not significant, 95% CI [−.017, 0.001]. The model explained 28% of the variance in state emotion dysregulation and 34.4% of the variance in Wave 2 deliberate self-harm.
** $p < .01$. *** $p < .001$.

greater PTSD symptoms were significantly associated with greater state emotion dysregulation, $b = 0.38$ ($SE = 0.06$), $p < .001$, $\beta = .53$, and greater state emotion dysregulation predicted higher odds of deliberate self-harm at Wave 2 when all other variables were in the model, $b = 0.11$ ($SE = 0.04$), $p < .002$, $\beta = .56$. More specifically, for a 1-point increase in state emotion dysregulation, the log-odds of any deliberate self-harm (vs. none) at Wave 2 increased by .11. There was no significant direct relation between PTSD symptoms and Wave 2 deliberate self-harm when accounting for mediators and covariates, $b = -0.01$ ($SE = 0.03$), $p = .64$, $\beta = -.09$. Finally, contrary to our first hypothesis, state emotional reactivity did not account for significant variance in the association between baseline PTSD symptoms and later deliberate self-harm when accounting for state emotion dysregulation and covariates, 95% CI [−.017, 0.001]; however, supporting our second hypothesis, state emotion dysregulation did account for significant variance in the association between baseline PTSD symptoms and the odds of Wave 2 deliberate self-harm when controlling for state emotional reactivity, baseline self-harm, and new Wave 2 SV, $b = 0.04$, 95% CI [.017, .067], $\beta = .30$. The overall model accounted for 28% of the variance in state emotion dysregulation and 34.4% of the variance in the odds of Wave 2 deliberate self-harm.

Discussion

The present study examined whether state emotional reactivity and state emotion dysregulation account for significant variance in the association between baseline PTSD symptoms and later deliberate self-harm among women with a history of SV. State emotion dysregulation, but not state emotional reactivity, accounted for the prospective relation between PTSD symptoms and deliberate self-harm. Specifically, women who reported greater baseline PTSD symptoms endorsed greater state emotion dysregulation after a laboratory stressor and, in turn, were more likely to report deliberate self-harm 4 months later. Findings support the role of in-the-moment difficulties regulating emotions in response to stressors in risk for later deliberate self-harm among women with SV-related PTSD symptoms.

Contrary to expectations, PTSD symptoms were not significantly associated with the presence of deliberate self-harm at baseline or 4-months later. Given that most of our sample of SV survivors (87.9%) endorsed some PTSD symptoms, it is possible that it is the presence of any PTSD symptoms, rather than symptom severity as examined here, that is associated with greater risk for deliberate self-harm. Alternatively, the relations found in prior work (Holliday et al., 2018; Nada-Raja & Skegg, 2011) may have been driven by the shared associations of PTSD symptoms and deliberate self-harm with third variables (e.g., emotion dysregulation).

PTSD symptom severity was not significantly related to state emotional reactivity among our sample of SV survivors. This finding contradicts prior work showing heightened emotional reactivity among trauma survivors with greater PTSD symptoms following trauma-related (Badour & Feldner, 2013) and general (Brown et al., 2018) laboratory stressors. Given similarities in research design between the current and past studies (including the dimensional assessment of PTSD symptom severity and operationalization of emotional reactivity as change in NA in response to a laboratory task), this difference in findings is surprising. Although it is unclear what accounts for this difference, Brown et al. (2018) included male military veterans whereas we examined civilian women with a history of SV. Further, whereas Badour and Feldner (2013) used a trauma-specific task with personal relevance to the individual, we used a generalized stressor that is not directly related to the source of PTSD symptoms.

Findings also did not support the hypothesized association between state emotional reactivity and greater likelihood of deliberate self-harm 4 months later. Although theory suggests that individuals may turn to deliberate self-harm to cope with more reactive emotions (Chapman et al., 2006), the results of our study align with those of prior studies using both self-report and physiological measures of emotional reactivity that failed to find an association between emotional reactivity and deliberate self-harm across multiple populations (Bresin, 2014; Davis et al., 2014; Glenn et al., 2011; Gratz, 2006). Together, these studies suggest that the tendency to respond more strongly to emotional stimuli may not increase risk for deliberate self-harm on its own. Instead, emotional reactivity may increase risk for deliberate self-harm because of its association with emotion dysregulation. That is, because more reactive emotions are more difficult to regulate (Gross & Jazaieri, 2014; Lynch et al., 2001), it may be the association of greater emotional reactivity with greater emotion dysregulation in some cases that explains past findings of an association between emotional reactivity and self-harm. In the absence of emotion regulation difficulties, emotional reactivity may not in and of itself increase risk for maladaptive behaviors such as deliberate self-harm. Alternatively, the relation of emotional reactivity to deliberate self-harm may be driven by reactivity to certain types of stressors (e.g., trauma-related cues) but not others.

As expected, more severe PTSD symptoms were associated with greater state emotion dysregulation following a laboratory stressor. These findings add to the literature documenting higher levels of emotion dysregulation among trauma survivors with greater PTSD symptoms and may reflect the increased regulatory demands associated with PTSD-related distress and arousal (Hannan & Orcutt, 2020; Seligowski et al., 2015). Applied to the context of survivors' daily lives, SV survivors with greater PTSD symptoms may experience greater difficulties regulating emotions in response to daily stressors. However, given that maladaptive responses to emotions may also increase vulnerability to both traumatic experiences and their emotional sequelae (Bardeen et al., 2013), the association between PTSD symptoms and emotion dysregulation is likely bidirectional.

Similarly, our finding that state emotion dysregulation in response to a generalized laboratory stressor predicted future deliberate self-harm supports the central role of emotion dysregulation in the development and maintenance of self-harm (Andover & Morris, 2014; Gratz & Roemer, 2008). Because deliberate self-harm is often used to avoid or escape unwanted emotional experiences that are deemed intolerable (Chapman et al., 2006), SV survivors may be particularly likely to turn to deliberate self-harm to manage unbearable internal experiences associated with PTSD symptoms. Indeed, supporting our second hypothesis, state emotion dysregulation accounted for the relation between SV-related PTSD symptoms and deliberate self-harm 4 months later, beyond state emotional reactivity. Given that our first hypothesis was not supported and state emotional reactivity did not contribute to the association between SV-related PTSD symptoms and later deliberate self-harm, results underscore the importance of maladaptive responses

to emotional reactions, rather than the nature or quality of one's emotional reactions per se, to future deliberate self-harm among SV survivors. Women with greater PTSD symptoms may be less able to identify, understand, and effectively regulate emotional experiences in response to stressors, thereby increasing their risk of using more harmful emotion regulatory strategies, such as deliberate self-harm. If responses to the laboratory stressor used here are characteristic of how SV survivors typically respond to stressors in daily life, results suggest that difficulties regulating emotions in response to daily stressors may contribute to the likelihood of future deliberate self-harm.

Limitations

This study has several limitations that should be considered. First, regarding our sample characteristics, participants were primarily well-educated, heterosexual, and cisgender. Further, our sample consisted of only young adult women, which precludes generalization of findings to both adolescents and adult men and nonbinary survivors of SV (who would also be at high risk for PTSD symptoms). Additionally, the use of two items to assess SV may not have adequately captured the full range of participants' victimization experiences, and there are known limitations of asking participants to label experiences as SV (Wilson & Miller, 2016). Further, given the relatively low rates of deliberate self-harm at Wave 2 (16%), this study may have been underpowered to detect small associations among constructs, which could explain the absence of significant findings pertaining to state emotional reactivity.

It is also important to note that we assessed subjective emotional reactivity and dysregulation (rather than physiological indices of these constructs) and used retrospective self-report measures to capture SV history and related PTSD symptoms. Thus, our findings could still be influenced by retrospective recall and social desirability biases, as well as limitations related to an individual's ability to accurately report on internal experiences. Further, because we assessed PTSD symptoms and our proposed intermediary variables at the same time point, we cannot draw conclusions about the temporal relations among these variables. It is possible, for instance, that PTSD symptoms and emotion dysregulation are bidirectionally related (Bardeen et al., 2013). Although our analyses focused on the presence of deliberate self-harm occurring after SV, it is possible that women were engaging in self-harm before initial victimization. Relatedly, because we examined whether greater emotional reactivity and emotion dysregulation predicted the occurrence of deliberate self-harm 4 months later, we are unable to draw conclusions about proximal associations among these constructs, such as those occurring within a single day.

Finally, our study measured, rather than manipulated, study variables. Accordingly, we are unable to draw causal conclusions about the nature of associations among SV-related PTSD symptoms, emotional responding, and deliberate self-harm. There may also be other unassessed variables that could contribute to the current pattern of findings. For example, women in our study could have used deliberate self-harm for a variety of reasons, including to communicate distress or punish themselves (Taylor et al., 2018). Measuring these motives could lead to a more complete understanding of why women with a history of SV engage in deliberate self-harm.

Future Research Directions

First, replication of the present study using larger samples is warranted, given that we did not find the anticipated effects related to state emotional reactivity. To address study limitations, future studies should use more detailed and thorough assessments of SV, particularly those that use behaviorally specific descriptions of SV acts and tactics rather than asking survivors to label their own SV (Anderson et al., 2021; Wilson & Miller, 2016). Future research would also benefit from the use of more extended prospective designs to clarify the precise nature and directions of the relations among PTSD symptoms, emotional responding, and deliberate self-harm. Specifically, studies should assess women close to initial SV exposure to examine the development and potential reciprocity of PTSD symptoms and emotional responding over time. Longitudinal research in younger adolescent samples would also help clarify the direction of the relation between SV exposure and deliberate self-harm, adding to the understanding of whether self-harm may be best considered a correlate or consequence of SV.

Likewise, future research should more closely model the momentary associations posited by prominent theories of deliberate self-harm, such as by using ecological momentary assessment (EMA) to assess whether acute elevations in PTSD symptoms and emotion dysregulation predict self-harm later that day. Although these designs are intensive and often require increased risk assessment and response (Davidson et al., 2017), EMA could be used to answer questions about proximal temporal relations with high ecological validity. Further, EMA could be used to examine whether measurements of state emotional responding from naturalistic settings mirror observations from highly controlled laboratory studies. Experimental paradigms could also be used to examine causal associations among PTSD symptoms, state emotional responding, and deliberate self-harm. Trauma-specific paradigms (e.g., trauma narrative scripts), mood induction tasks, or laboratory stressors could be paired with experimental deliberate self-harm analogues, such as algometers, cold pressor tasks, or self-administration of electric shocks (Ammerman et al., 2018). Such studies could examine whether higher levels of PTSD symptoms and emotion dysregulation following experimental manipulations influence persistence in and tolerance of deliberate self-harm analogue tasks. Finally, to complement the current focus on risk factors, future studies should examine possible mechanisms of resiliency for deliberate self-harm–a particularly important aim, given that most women in our sample (84%) did not engage in deliberate self-harm during the follow-up period. There may be natural sources of resiliency that could be leveraged to reduce risk for ongoing deliberate self-harm, such as social support.

Clinical Implications

The present study has several clinical implications for understanding and reducing high rates of deliberate self-harm among SV survivors. Given evidence for the relevance of maladaptive responses to emotions (i.e., state emotion regulation difficulties) rather than emotional reactions to a stressor, future work should examine the clinical utility of teaching SV survivors adaptive ways to regulate and respond to their emotions during daily stressors. Empirically supported treatments such as dialectical behavioral therapy incorporate skills to bolster emotion regulation

(e.g., emotional acceptance, opposite action; Neacsiu et al., 2014) and could be examined as a potential intervention for improving emotion regulation and mitigating risk for deliberate self-harm among SV survivors. Because our study focused on participants' perceptions of their own emotion regulatory abilities, it might also be beneficial to examine whether targeting survivors' perceptions of and beliefs about their emotion regulation abilities could buffer against deliberate self-harm. Such studies could examine potential barriers (e.g., lack of accessible strategies, lack of confidence) to using more adaptive regulation strategies (e.g., acceptance, seeking social support) and identify targets to help women overcome these barriers. Although more research is needed, findings suggest that helping women with elevated SV-related PTSD symptoms learn and implement effective strategies for responding to emotional distress related to stressors might help reduce their risk for later deliberate self-harm.

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