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Facets of emotion dysregulation differentially predict depression and PTSD symptom severity following traumatic injury

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ABSTRACT

Background: Emotion dysregulation is a hallmark characteristic of psychopathology following trauma. Yet, emotion dysregulation is multifaceted, and little is known about which aspects of emotion dysregulation predict depression and posttraumatic stress disorder (PTSD) symptom severity following traumatic injury.

Objective: The aim of this longitudinal study was to evaluate how facets of dysregulation differentially predicted the severity of PTSD symptom clusters and depressive symptoms six months after a traumatic injury requiring medical treatment.

Methods: Traumatically injured adults ($N = 99$) presenting to a Level 1 trauma centre completed a measure of emotion dysregulation 2 weeks post-injury, and PTSD and depression were assessed at 2-weeks and 6 months later.

Results: Using stepwise regressions controlling for baseline symptoms, age, gender, race, and injury severity, results showed baseline emotion dysregulation significantly predicted the four symptom clusters of PTSD 6 months post-injury. Notably, hyperarousal symptoms and negative alterations in mood and cognition were predicted by a lack of clarity. On the other hand, depressive symptoms were significantly predicted by difficulty accessing emotion regulation strategies.

Conclusion: Results highlight that specific facets of emotion dysregulation predict PTSD and depression symptom severity differentially after injury. Indeed, lack of emotional clarity appears to predict PTSD symptomatology, suggesting a potential mechanism driving worsening symptoms. Lack of clarity could also be detrimental to engagement in PTSD treatment. Conversely, lack of regulation strategies may represent a sense of helplessness in managing depression after trauma. As such, future research should elucidate whether interventions targeting aspects of emotion dysregulation based on symptom presentations are useful in treating PTSD and depression following injury.

Facetas de la desregulación emocional predicen gravedad de los síntomas de depresión y TEPT luego de lesión traumática

Antecedentes: La desregulación emocional es una característica distintiva de la psicopatología luego de trauma. Es más, la desregulación emocional es multifacética, y poco se conoce sobre cuales aspectos de la desregulación emocional predicen la gravedad de los síntomas de depresión y del trastorno de estrés postraumático (TEPT) luego de una lesión traumática.

Objetivo: El objetivo de este estudio longitudinal fue evaluar como las facetas de la desregulación predicen diferencialmente la gravedad de los grupos de síntomas del TEPT y los síntomas depresivos seis meses después de una lesión traumática que requiere tratamiento médico.

Método: Adultos lesionados traumáticamente ($N = 99$) que se presentaron a un centro de trauma de Nivel 1 completaron una medida de desregulación emocional dos semanas luego de la lesión, y el TEPT y la depresión fueron medidos a las dos semanas y a los 6 meses después.

Resultados: Usando regresiones paso a paso controlando los síntomas de la línea base, la edad, el género, la raza, y la severidad de la lesión, los resultados mostraron que la desregulación emocional de la línea base predijo significativamente los cuatro grupos de síntomas del TEPT 6 meses después de la lesión. Notablemente, los síntomas de hiperactivación y las alteraciones negativas en el estado de ánimo y la cognición fueron predichos por la carencia de claridad. Por otro lado, los síntomas depresivos fueron predichos de manera significativa por la dificultad en acceder a estrategias de regulación emocional.

Discusión: Los resultados enfatizan que facetas específicas de la desregulación emocional predicen la gravedad de los síntomas de TEPT y depresión de diferente forma luego de la lesión. Por supuesto, la carencia de claridad emocional aparece prediciendo la sintomatología del TEPT, sugiriendo un mecanismo potencial que puede llevar a un

HIGHLIGHTS
- Specific facets of emotion dysregulation are differentially associated with PTSD symptom clusters and depression symptom severity after injury.
- Findings suggest that targeting lack of emotional clarity may be critical for patients with prominent hyperarousal and trauma-related changes in mood and cognitions (i.e. two PTSD symptom clusters).
- Patients with depressive symptoms following trauma may especially benefit from the development of emotion regulation strategies.

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empeoramiento de los síntomas. La carencia de claridad podría ser también perjudicial para el compromiso en el tratamiento del TEPT. Por otro lado, la carencia de estrategias de regulación podría representar un sentido de desesperanza en manejar la depresión luego del trauma. De manera tal, las investigaciones futuras deberían dilucidar si las intervenciones orientadas a los aspectos de la desregulación emocional basadas en las presentaciones de los síntomas son útiles en el tratamiento del TEPT y la depresión luego de una lesión.

情绪失调的各个方面不同地预测创伤性受伤后抑郁和 PTSD 症状严重程度

背景：情绪失调是创伤后精神病的一个标志性特征。 然而，情绪失调是多方面的，关于情绪失调的哪些方面可以预测创伤性受伤后抑郁和创伤后应激障碍 (PTSD) 症状的严重程度，我们知之甚少。
目的：本纵向研究旨在评估在需要医疗的创伤性受伤后六个月，失调的各个方面如何不同地预测 PTSD 症状簇和抑郁症状的严重程度。
方法：到一级创伤中心就诊的创伤性受伤成人 (N = 99) 在受伤后 2 周完成情绪失调测量，并在 2 周和 6 个月后评估 PTSD 和抑郁。
结果：使用逐步回归控制基线症状、年龄、性别、种族和受伤严重程度，结果表明基线情绪失调显著预测了受伤后 6 个月 PTSD 的四个症状簇。值得注意的是，高唤起症状以及情绪和认知的负性改变是由缺乏清晰度预测。 另一方面，难以获得情绪调节策略可显著预测抑郁症状。
结论：结果强调，情绪失调的特定方面可以在受伤后以不同方式预测 PTSD 和抑郁症状的严重程度。 事实上，情绪明确性的缺乏似乎可以预测 PTSD 症状，表明这可能是导致症状恶化的机制。 缺乏明确性也可能不利于参与 PTSD 治疗。 相反，缺乏调节策略可能代表一种在处理创伤后抑郁方面的无助感。 因此，未来的研究应该阐明基于症状表现针对情绪失调方面的干预措施是否有助于治疗受伤后的创伤后应激障碍和抑郁。

Emotion dysregulation encompasses the inability to or deficits in regulating the intensity, duration, or onset of one's emotional experience (Gross, 1998). Successful emotion regulation is comprised of (1) understanding and awareness of one's emotions, (2) acceptance of emotions, (3) ability to manage impulse and control behaviour to support one's goals, and (4) use emotion regulation strategies for a given context in a successful manner (Kaufman et al., 2016). The field of emotion regulation has explored a host of contexts in which dysregulation impacts functioning, with severe forms of dysregulation often associated with the development of psychopathology (e.g. Major Depressive Episodes, Borderline Personality Disorder; Stepp et al., 2014). Indeed, traits related to emotion expressional (e.g. alexithymia, or the inability to identify and describe emotion) and, in turn, regulation can meaningfully impact the process of experiencing emotion, shaping a person's understanding of their emotion and what might be necessary in order to modulate affect (Boden et al., 2012; Feldman Barret & Gross, 2001; Moormann et al., 2012; Lane et al., 2022). Further, deficits in emotion regulation are proposed to maintain psychopathology transdiagnostically (Fernandez et al., 2016). Following trauma, one's trait abilities related to emotion expression, awareness, and dysregulation appear to be a critical component of posttraumatic stress disorder (PTSD; Frewen et al., 2008; Moormann et al., 2012). Neuroimaging research implicates areas critical to emotion processing, awareness, and regulation in PTSD development (Fitzgerald et al., 2018; Frewen et al., 2008). Emotion dysregulation is also present across the four symptom clusters of PTSD: Re-experiencing, Avoidance, Negative alterations in mood and cognition, and Hyperarousal symptoms. PTSD is characterized by heightened emotionality in response to stimuli associated with the traumatic event, attempts to avoid these stimuli and associated emotions, and overall increases in negative emotions and diminished positive emotions making dysregulation central to many components of each cluster (American Psychiatric Association, 2013). As such, understanding the role of emotion dysregulation in the acute aftermath of trauma is a vital mechanism for informing how dysregulation might contribute to overall recovery as well as the development and treatment of disorders, such as PTSD (Lane et al., 2022).

Given the interplay between emotion dysregulation and psychopathology following trauma, the consideration of emotion dysregulation is vital for accurate assessment and treatment of dysregulation. Across a host of trauma-exposed populations, difficulty regulating both negative and positive emotions is evident as predictors of PTSD (Goldsmith et al., 2013). In community samples with trauma histories, generally maladaptive emotion regulation strategies such as rumination, suppression, and avoidance of emotion predict PTSD (Pugach et al., 2020; Seligowski et al., 2016). Similarly, for those recently exposed to (e.g. witnessing, learning of) firearm violence, individuals with greater emotion dysregulation were more likely to have PTSD symptoms eight months after the shooting (Bardeen et al., 2013). Congruent with the literature on PTSD, individuals with histories of trauma

use fewer adaptive regulation strategies and more maladaptive strategies, which relate to depressive symptoms (Berking et al., 2014). Individuals who experienced childhood trauma were also more likely to have greater depression if they showed broad deficits in emotion regulation skills, including in acceptance (Cloitre et al., 2019; Ulman et al., 2014; Hopfinger et al., 2016).

Yet, it is vital to examine the process and skills needed to adaptively use regulation strategies in the context of stress and trauma. Conceptually, it is proposed that beyond simply using 'better' emotion regulation strategies, it is the manner of understanding one's emotions, evaluating context, and flexibly responding that is associated with better outcomes (Aldao et al., 2015). Indeed, facets such as emotional clarity or ability to accept emotions can carry significant implications in how one chooses to ultimately regulate emotions that arise (Tull et al., 2007). Therefore, to better understand the interplay between emotion dysregulation and mental health, research has examined specific facets of dysregulation in the context of trauma. For instance, in a cross-sectional study, lack of access to regulation strategies, non-acceptance of emotion, and lack of emotional clarity accounted for 27% of the variance of overall PTSD symptom severity (Christ et al., 2021). This was congruent with the work of Weiss and colleagues (2012) showing that these facets of emotion dysregulation as well as impulse control difficulties were greater in patients with PTSD than a control group. Further, in another study exploring dysregulation as a mediator of PTSD symptoms and physical health following acute trauma exposure, results showed that limited access to strategies, lack of emotional clarity, and impulse control difficulties were mediators of the relationship between PTSD on poor health (Carter et al., 2020). In individuals who experienced early or chronic trauma (e.g. childhood sexual/physical abuse), current lack of emotional clarity accounted for variance of current distress (Ehring & Quack, 2010). As such, there is variability across studies on what aspects of dysregulation relate to PTSD symptomatology. Further, this previous work is with a wide array of trauma exposures and does not consistently assess or control for when the trauma occurred. Additionally, there is limited understanding on what aspects of dysregulation predict the specific symptom clusters of PTSD, which has the potential to inform personalized, targeted treatment (Rothbaum et al., 2012). To date, limited work has explored the relationship between emotion dysregulation and acute trauma recovery, as looking at this acute timeframe can influence interventions to better target dysfunctional regulation processes that may inhibit treatment of PTSD. Further, none of the prior studies involved a traumatically injured sample nor investigated specific PTSD symptom clusters in relation to facets of emotion dysregulation.

This lack of knowledge about the long-term effects of emotion dysregulation in the acute window of trauma is a major knowledge gap since over 3 million individuals in the United States sustain a traumatic injury requiring medical attention each year (National Institute of Trauma, 2014). Injuries occur in a host of traumatic events, such as motor vehicle collisions, falls, or assaults. Individuals experiencing injury in the context of trauma exposure are at heightened risk of poor quality of life (e.g. Brasel et al., 2010; Trevino et al., 2014), highlighting the importance of understanding the early emotional processes underlying this risk. To date, two studies have examined emotion dysregulation in an injury sample presenting to emergency departments and found that emotion dysregulation predicted overall greater PTSD symptom severity as well as a greater likelihood of having chronic PTSD (Forbes et al., 2020; Pencea et al., 2020). Specifically, Forbes and colleagues used the Difficulty in Emotion Regulation Scale (DERS), and while controlling for baseline PTSD symptoms, emotion dysregulation predicted PTSD symptom severity at 3 months. Similarly, Pencea and colleagues used the standard trauma interview to control for symptoms and found that emotion dysregulation as assessed by the Emotion Dysregulation scale predicted PTSD symptom severity over the course of 12 months. However, within these studies, facets of dysregulation were not significant predictors of PTSD nor were they examined in relation to PTSD symptom clusters (Forbes et al., 2020; Pencea et al., 2020). Similarly, only one study has examined traumatic injury survivors' emotion regulation and depression, though baseline emotion dysregulation was not evaluated; Van Loey and colleagues (2014) found in burn injury survivors, greater rumination, as assessed using the Cognitive Emotion Regulation Questionnaire at 9 months after injury, predicted depression two years post-injury. This suggests regulation can play an important role in PTSD and depression development in the acute phase of recovery. With earlier treatment of mental health having greater effects, emotion dysregulation may be an important target for early intervention after trauma. Research should expand these findings to explore specific skills of emotion regulation to understand how to inform intervention.

To this end, we sought to fill this knowledge gap by investigating how aspects of emotion dysregulation predict PTSD and depression symptom severity after acute traumatic injury, while controlling for covariates including baseline symptoms and demographic factors. The authors assessed whether the components of emotion dysregulation differentially predicted greater PTSD and depression symptom severity after traumatic injury. PTSD was broken into the specific

symptom clusters (i.e. Re-experiencing, Avoidance, Hyperarousal, and Negative Alterations in Mood and Cognition) to determine if facets of emotion dysregulation predicted greater symptom severity by the clusters of PTSD. Given the initial literature, we hypothesized that difficulty accessing strategies, non-acceptance of emotion, lack of emotional clarity, and impulse control would predict higher PTSD symptom clusters severity. We also hypothesized that difficulty accessing strategies and non-acceptance of emotion (indicative of greater rumination and difficulty regulating) would predict higher depression symptom severity.

1. Methods

1.1. Participants

The sample was from a longitudinal study aimed at identifying risk factors for the posttraumatic development of psychopathology using biospecimens, genetics, and self-report measures. Enrolled participants were either treated in the emergency department or admitted to the Level I Trauma centre for a single-incident traumatic injury (e.g. falls, motor vehicle collisions). Inclusion criteria included the following: (1) 18 years of age or older; (2) Glasgow Coma Scale > 13 on arrival; (3) did not meet criteria for moderate to severe TBI; (4) injury was not self-inflicted; and (5) adequate English language proficiency. A total of 7947 patients were assessed for eligibility, and 7,748 were excluded due to exclusion criteria. A total of 199 participants consented to the study, though only 193 completed relevant baseline measures. Of those participants, 96 completed relevant 6-month follow-up measures (49.7% retention rate, congruent with retention rates of studies with similar injury populations). Difference testing was completed to identify significant differences between completers and non-completers by gender, racial/ethnic minority status, interpersonal mechanism of injury, injury severity, age, baseline emotion dysregulation, depression, and PTSD symptom severity. Specifically, chi-square test of independence was utilized for categorical variables and Mann–Whitney U test was utilized for skewed variables. Completers and those lost to follow-up did not significantly differ across any of these variables ($p > .05$). See Table 1 for demographic characteristics and descriptive statistics.

1.2. Procedure

Potential participants were screened consecutively via a daily emergency department and trauma census and based on the inclusion criteria. Potential participants were approached with information about the study and, if interested, written informed consent was

Table 1. Sample demographic characteristics & descriptive statistics.

	M (SD)
Age	38.80 (15.36)
ISS	13.43 (9.57)
Baseline PTSD severity	24.08 (21.42)
Baseline Depression severity	11.72 (8.25)
Six-month PTSD severity	
Total	15.79 (15.14)
Re-experiencing	4.22 (4.55)
Avoidance	1.95 (2.06)
Negative alteration in mood and cognition	5.10 (5.60)
Hyperarousal	4.52 (4.20)
Six month Depression severity	11.25 (7.64)
DERS-SF Subscales	
Clarity	5.62 (3.13)
Goals	7.90 (4.02)
Impulse	5.66 (3.48)
Non-acceptance	6.05 (3.27)
Strategies	6.12 (3.37)
Awareness	5.94 (2.97)
	N (%)
Female	36 (36.4%)
Race/Ethnicity Minority	
African American/Black	55 (55.6%)
White	19 (19.2%)
Hispanic/Latinx	21 (21.2%)
Asian	2 (2.0%)
Native American/Alaska Native/Native Hawaiian	2 (2.0%)
Mechanism of Injury	
Motor vehicle crash	38 (38.4%)
Gun Shot Wound	20 (20.2%)
Fall	11 (11.1%)
Stab Wound	5 (5.1%)
Pedestrian struck by vehicle	7 (7.1%)
Motorcycle crash	8 (8.1%)
Industrial Accident	1 (1.0%)
Other	9 (9.1%)
N = 99	

obtained. Participants were asked to complete study visits at baseline (in hospital or within two weeks post-injury or hospital discharge), and 6 months post-injury. During each visit (approximately 90-120 min at baseline and 180 min at 6-month), participants completed a battery of self-report measures and provided biospecimens. For the current study, only self-report data were used. Participants received compensation at each study visit. The study's procedure was approved by the institutional review board (Medical College of Wisconsin IRB, PRO#22827).

1.3. Measures

1.3.1. Emotion dysregulation

The Difficulties in Emotion Regulation Scale – Short Form (DERS-SF; Kaufman et al., 2016) was developed using Gratz and colleagues' model to assess the deficits that one has in successful emotion regulation. The DERS-SF is an 18-item measure that assesses six facets of emotion dysregulation, along with a total score of general emotion regulation difficulty. Subscales include difficulty accessing strategies (Strategies), difficulty with emotional awareness (Awareness), non-acceptance of emotion (Non-acceptance), difficulty with impulse (Impulse), difficulty with goals (Goals), and difficulty with emotional clarity

(Clarity). Respondents report the extent to which they believe each item applies to them using a scale ranging from 1 = *almost never, 0-10%* to 5 = *almost always, 91-100%*. Items are summed for a total score and six sub-scale scores with higher scores indicating greater self-reported difficulties in regulating emotion.

1.3.2. PTSD symptoms

Baseline symptoms of PTSD were assessed using the PTSD Checklist (PCL-5; Blevins et al., 2015), which is a 20-item self-report measure that assesses symptoms of PTSD. Participants endorse symptom severity on a five-point Likert scale from 0 ('not at all') to 4 ('extremely') of PTSD symptoms within the past month. Participants were asked to refer to the injury event which brought them into the ED prior to enrolment when responding to the measure. Scores are totalled across all items for a range of 0–80, with higher scores indicative of greater symptom severity. The PCL-5 demonstrates good psychometric properties, including internal consistency, test-retest reliability, and convergent and discriminant validity (Blevins et al., 2015).

At six months, PTSD symptoms were assessed using the Clinician Administered PTSD Scale for DSM-5 (CAPS-5; Weathers et al., 2013), including sections evaluating each symptom cluster of the DSM-5 PTSD diagnosis. The CAPS-5 shows good interrater reliability for frequency and intensity of symptoms (Pearson's $r = .83$–1.00; Forbes et al., 2015). For the current study, sum scores were calculated for each of the four symptom clusters: Hyperarousal, Re-experiencing, Avoidance, and Negative Alterations in Mood and Cognition.

1.3.3. Depressive symptoms

The Center for Epidemiologic Studies Depression Scale – Revised (CESD-R; Eaton et al., 2004) is a 20-item measure designed to assess for depressive symptoms. Respondents report the extent to which they have experienced different symptoms on a Likert scale of 0 (Not at all) to 4 (Nearly every day) for the past 2-week period. Items are summed for a total symptom severity score (0–60) with higher numbers indicative of greater depressive symptoms.

1.4. Data analytic plan

Initial bivariate correlation analyses were conducted to determine significant relationships between DERS-SF subscales, PTSD symptom clusters, and depressive symptoms. Variables with significant correlations were included in stepwise regression analyses. Specifically, six stepwise regression analyses were used to determine if the DERS-SF subscales, collected at baseline, predicted variance in total PTSD symptom severity, the four PTSD symptom clusters, and depressive symptoms 6-months post-injury. Step 1 of each regression included baseline covariates: gender, age, race/ethnicity, injury severity score (ISS; an objective measure of the severity of physical injury) and baseline depression or PTSD, given support for the inclusion of these as covariates in prior research (Brasel et al., 2010). Step 2 of each regression included DERS-SF subscales which were significant at the bivariate level. We checked for potential violations of the assumptions of inferential statistics we utilized using best practices (Cohen et al., 2003; Field, 2013). Results of assumption testing indicated that all key assumptions were adequately met.

2. Results

2.1. Total depression & PTSD symptom severity

Bivariate analyses demonstrated that the Awareness subscale was not related to PTSD clusters or depression; it was subsequently not included in hierarchical regression analyses (see Table 2). To start, two regression analyses were conducted to assess DERS-SF subscales as a predictor of the overall PTSD symptom severity and depression severity, separately (see Table 3). When examining depression, results demonstrated that Step 1 of the regression (ISS, age, gender, and baseline depression) was significant ($F(4,75) = 14.92$, $p < .001$) and predicted 45.7% of variance in depression symptoms. Step 2, including the DERS-SF subscales was also significant ($F(9,75) = 15.28$, $p < .001$, $\Delta F(5,66) = 8.92$, $p < .001$, $R^2_{change} = .22$), suggesting that DERS-SF subscales predicted 21.9% additive variance of depression symptoms, even when controlling for baseline depression. Results showed both baseline depression ($\beta = .37$, $p < .001$) and Strategies ($\beta = .35$, $p < .05$) were significant predictors of depression symptoms at 6 months. When examining total PTSD symptoms severity, results demonstrated that Step 1 of the regression (ISS, age, gender, and baseline PTSD) was significant ($F(4,78) = 19.89$, $p < .001$) and predicted 51.8% of variance in PTSD symptoms at 6 months. Step 2 was also significant ($F(9,78) = 12.74$, $p < .001$, $\Delta F(5,69) = 3.91$, $p < .05$, $R^2_{change} = .11$), suggesting DERS-SF subscales predicted 10.6% additive variance of PTSD symptoms even when controlling for baseline PTSD symptoms. Results showed both baseline PTSD ($\beta = .37$, $p < .05$) and Clarity ($\beta = .25$, $p < .05$) were significant predictors of PTSD symptoms at 6 months.

2.2. Emotion dysregulation & PTSD symptom clusters

To explore how DERS-SF subscales might differentially predict PTSD symptomatology, four stepwise regressions were conducted examining the four

Table 2. Correlations of primary independent and dependent variables.

	1	2	3	4	5	6	7	8	10	11	12	13	14	15
1. DERS Clarity	1	-	-	-	-	-	-	-	-	-	-	-	-	-
2. DERS Goals	.47**	1	-	-	-	-	-	-	-	-	-	-	-	-
3. DERS Impulse	.47**	.60**	1	-	-	-	-	-	-	-	-	-	-	-
4. DERS Nonacceptance	.67**	.60**	.55**	1	-	-	-	-	-	-	-	-	-	-
5. DERS Strategies	.73**	.75*	.66**	.78**	1	-	-	-	-	-	-	-	-	-
6. DERS Awareness	.03	-.23*	-.12	-.19	-.14	1	-	-	-	-	-	-	-	-
7. Baseline Depression	.54**	.56**	.47**	.45**	.59**	.31	1	-	-	-	-	-	-	-
8. Baseline PTSD	.51**	.49**	.42**	.44**	.54**	.10	.70**	1	-	-	-	-	-	-
9. 6 mo Depression	.65**	.63**	.47**	.61**	.73**	-.12	.69**	.60**	1	-	-	-	-	-
10. 6 mo PTSD Total	.53**	.55**	.51**	.50**	.61**	-.13	.55**	.67**	.73**	1	-	-	-	-
11. 6 mo Avoidance	.47**	.50*	.50**	.46**	.56**	.01	.47**	.58**	.63**	.83**	1	-	-	-
12. 6 mo Hyperarousal	.48**	.48**	.44**	.40**	.52**	-.11	.47**	.57**	.67**	.92**	.70**	1	-	-
13. 6 mo NAMC*	.52**	.49**	.47**	.48**	.55**	-.17	.51**	.62**	.68**	.95**	.77**	.81**	1	-
14. 6 mo Re-experiencing	.48**	.57**	.49**	.48**	.62**	-.11	.53**	.68**	.70**	.93**	.73**	.83**	.82**	1

*Negative alterations in mood and cognition (NAMC).

symptom clusters of PTSD separately (see Table 4). When examining avoidance symptoms, results showed that while both steps of regression were significant, the DERS-SF subscales did not significantly predict variance of avoidance symptoms beyond Step 1 ($\Delta F(5,69) = 1.68$, p = ns, R^2_{change} = .07). When examining Negative Alterations in Mood and Cognition symptoms, results demonstrated that Step 1 of the regression was significant ($F(4,78) = 16.67$, $p < .001$) and predicted 47.4% of variance in PTSD symptoms at 6 months. Step 2 was also significant ($F(9,78) = 9.85$, $p < .001$, $\Delta F(5,69) = 2.79$, $p < .05$, R^2_{change} = .09), suggesting DERS-SF subscales predicted 8.8% additive variance of Negative Alterations in Mood and Cognition symptoms even when controlling for baseline PTSD symptoms. Results showed both baseline PTSD ($\beta = .35$, $p < .05$) and Clarity ($\beta = .27$, $p < .05$) were significant predictors of Negative Alterations in Mood and Cognition symptoms at 6 months.

When examining hyperarousal symptoms, results demonstrated that Step 1 of the regression including ISS, age, gender, and baseline PTSD symptoms was significant ($F(4,78) = 11.85$, $p < .001$), and predicted 35.7% of variance in PTSD symptoms at 6 months. Step 2, including the DERS-SF subscales was also

significant, ($F(9,78) = 7.86$, $p < .05$, $\Delta F(5,69) = 3.23$, $p < .05$, R^2_{change} = .12), suggesting that DERS-SF subscales predicted 11.6% additive variance of hyperarousal symptoms even when controlling for baseline PTSD symptoms. Both baseline PTSD ($\beta = .32$, $p < .05$) and Clarity ($\beta = 33$, $p < .05$) were significant predictors of hyperarousal symptoms at 6 months.

Lastly, when examining re-experiencing symptoms, results demonstrated that Step 1 of the regression including ISS, age, gender, and baseline PTSD symptoms was significant ($F(4,78) = 19.51$, $p < .001$), and predicted 51.3% of variance in PTSD symptoms at 6 months. Step 2, including the DERS-SF subscales was also significant, ($F(9,78) = 12.86$, $p < .05$, $\Delta F(5,69) = 4.12$, $p < .05$, R^2_{change} = .11), suggesting that DERS-SF subscales predicted 11.3% additive variance of re-experiencing symptoms even when controlling for baseline PTSD symptoms. However, results showed that baseline PTSD ($\beta = .36$, $p < .05$) was the driving predictor of re-experiencing symptoms at 6 months.

3. Discussion

The purpose of this study was to investigate how different facets of emotion dysregulation after

Table 3. Depression and PTSD symptom severity stepwise regression variable coefficients.

Predictor	Depression symptom severity			PTSD symptom severity		
	b (SE)	β	p	b (SE)	β	p
Step 1						
Constant	4.54 (2.55)	–	.08	.79 (4.91)	–	.87
Age	−.002(.05)	−.005	.96	.03 (.09)	.02	.78
Gender	1.66 (1.54)	.10	.28	4.11 (2.77)	.12	.14
Injury Severity	−.08 (.07)	−.11	.24	.11 (.14)	.07	.41
Baseline Symptoms	.61(.10)	.64	<.001	.53 (.07)	.69	<.001
Step 2						
Constant	−2.12 (2.44)	–	.39	−10.88 (6.53)	–	.10
Age	.04 (.04)	.08	.31	.07 (.09)	.07	.36
Gender	.54 (1.29)	.03	.68	1.06 (2.82)	.03	.71
Injury Severity	−.09 (.06)	−.11	.13	.12 (13)	.07	.36
Baseline Symptoms	.35 (.09)	.37	<.001	.28 (.09)	.37	.002
Clarity	.35 (.30)	.14	.25	1.36 (.68)	.25	.05
Goals	.06 (.22)	.03	.78	.56 (.51)	1.09	.28
Strategies	.72 (.34)	.35	.04	.49 (.79)	.11	.54
Non-acceptance	.17 (.24)	.08	.48	−.31 (.56)	−.07	.59
Impulse	.08 (.19)	.04	.66	.56 (.44)	.14	.20

Note. Gender coded 0 = Male, 1 = Female.

Table 4. PTSD Symptom Cluster Severity Stepwise Regression Variable Coefficients.

Predictor	Re-experiencing			Avoidance			Negative Alterations in Mood & Cognition			Hyperarousal		
	b (SE)	β	p	b (SE)	β	p	b (SE)	β	p	b (SE)	β	p
Step 1												
Constant	−1.19 (1.46)	–	.42	.73 (.75)	–	.34	1.04 (.05)	–	.59	.20 (1.52)	–	.90
Age	.02 (.03)	.08	.39	−.01 (.01)	−.06	.55	−.02 (.04)	−.04	.63	.03 (.03)	.10	.33
Gender	1.16 (.82)	.12	.16	.28 (.43)	.06	.52	1.15 (1.07)	.09	.29	1.53 (.86)	.17	.08
Injury Severity	.05 (.04)	.11	.19	.01 (.02)	.06	.69	.05 (.05)	.08	.36	−.003 (.04)	−.01	.95
Baseline Symptoms	.16 (.02)	.69	<.001	.06 (.01)	.58	<.001	.18 (.03)	.64	<.001	.13 (.02)	.61	<.001
Step 2												
Constant	−4.31 (1.56)	–	.007	−.45 (.87)	–	.60	−2.67 (2.11)	–	.21	−3.16 (1.67)	–	.06
Age	.03 (.03)	.11	.19	−.003 (.01)	−.02	.81	−.01 (.05)	−.01	.88	.05 (.03)	.16	.09
Gender	.03 (.82)	.003	.97	−.02 (.46)	−.004	.97	.15 (1.12)	.01	.89	.90 (.89)	.10	.31
Injury Severity	.06 (.04)	.12	.12	.01 (.02)	.06	.52	.05 (.05)	.08	.35	.001 (.04)	.002	.99
Baseline Symptoms	.08 (.03)	.36	.002	.04 (.01)	.33	.02	.10 (.03)	.35	.01	.07 (.03)	.32	.01
Clarity	.22 (.19)	.14	.25	.11 (.11)	.15	.31	.54 (.26)	.27	.05	.50 (.21)	.33	.02
Goals	.23 (.14)	.20	.12	.05 (.08)	.09	.54	.11 (.20)	.08	.58	.16 (.16)	.15	.31
Strategies	.30 (.23)	.24	.19	.06 (.13)	.10	.65	.05 (.31)	.03	.87	.08 (.25)	.07	.74
Non-acceptance	−.11 (.16)	−.09	.50	−.02 (.09)	−.04	.81	.06 (.22)	.03	.80	−.24 (.17)	−.20	.18
Impulse	.09 (.13)	.08	.47	.09 (.07)	.15	.24	.17 (.17)	.11	.32	.21 (.14)	.18	.13

Note: Gender coded 0 = Male, 1 = Female.

traumatic injury incur risk for total symptom severity of PTSD and its 4 clusters along with depression, while controlling for covariates. Our results show that the role of emotion dysregulation in predicting PTSD and depression symptom severity six months after traumatic injury varies meaningfully across subtypes of emotion dysregulation and specific PTSD symptom clusters and depression. Findings highlight the prominent role of two subtypes of emotion dysregulation in the development of PTSD and depression symptom severity after traumatic injury: difficulty with emotional clarity and difficulty with accessing emotion regulation strategies. First, difficulty with emotional clarity was associated with Hyperarousal and Negative Alterations in Mood and Cognition symptom clusters as well as overall PTSD symptom severity. This relationship was still evidenced even when controlling for important covariates, including baseline acute stress symptoms. Of note, no other facet of emotion dysregulation predicted PTSD symptom severity, suggesting partial support of our hypothesis that access to regulation strategies, non-acceptance of emotion, lack of emotional clarity, and difficulty with impulse would predict PTSD symptom clusters. Regarding depression, difficulties accessing emotion regulation strategies was the only component of emotion dysregulation that predicted depression symptom severity. This again showed partial support for our hypothesis that both difficulty accessing strategies and non-acceptance of emotion would predict depressive symptom severity.

These results are consistent with several prior studies examining how aspects of emotion dysregulation relate differentially to PTSD and depression symptom severity. Emotional clarity is a foundational aspect of understanding one's emotions and involves the ability to differentiate one's emotional experiences and have awareness of one's own emotions (Frewen et al., 2012). It is notable that deficits in clarity are related to overall PTSD symptom severity as well as Hyperarousal and Negative Alterations in Mood and Cognition symptoms, suggesting heightened emotionality related to the trauma is difficult to understand when one exhibits low clarity. This could be due to the relationship between low emotional clarity and alexithymia, an experience defined by difficulty in both identifying and describing one's emotions (Frewen et al., 2012), which is also associated with greater PTSD symptom severity (Frewen et al., 2008). Similarly, individuals with low emotional clarity exhibit poor differentiation of emotions, and they may be more likely to label their emotional state in ways that perpetuate hyperarousal symptoms and negative cognitions about trauma or the world (e.g. feeling threatened, fear; Pond et al., 2012). Further, exacerbating this is an association between emotional clarity and anxiety sensitivity, suggesting not only confusion about emotions but also limited tolerance for fear or anxiety (Viana et al., 2018).

The contribution of emotional clarity in perpetuating PTSD symptoms and its association with both anxiety sensitivity and alexithymia may also influence response to trauma-focused treatment as well as patient experience in the hospital setting (Frewen et al., 2008). Indeed, although research suggests intervention for PTSD is most effective earlier after injury (Giummarra et al., 2018), lack of emotional clarity may deleteriously impact response to treatment. Engaging with trauma-related stimuli may be more challenging with deficits in emotional clarity, suggesting a need to improve the ability to understand emotions (e.g. physiological reactions of emotion, interpretation of emotion) prior to engagement of exposure to fear-inducing stimuli (Tull et al., 2007). While hospitalized, healthcare professionals can use trauma-informed care practices to reduce re-traumatization and foster more supportive environments of patients' emotional recovery (Steinhausen et al., 2014). Within mental

health intervention, addressing anxiety sensitivity and improving emotional clarity may promote greater tolerance of trauma-related stimuli prior to beginning exposure work (Viana et al., 2017).

Yet, it is notable the current study did not find that specific deficits in dysregulation predicted avoidance symptoms, given the literature proposing the connection between dysregulation and specific maladaptive strategies (e.g. suppression and avoidance) as a mechanism for PTSD development (Pugach et al., 2020; Seligowski et al., 2016; Tull et al., 2007). This may be explained by limited findings on emotional awareness (which may serve as a tool of avoidance) and marked deficits in clarity limiting one's understanding of avoidance as a tool (Frewen et al., 2012). Ongoing work might explore whether facets of regulation, such as clarity or awareness, indirectly shape avoidance symptoms through other mechanisms (e.g. use an avoidant coping style, suppression) to better understand how dysregulation influences avoidance. From an intervention perspective, mindfulness-based treatments show promise in improving emotional clarity (Butler et al., 2018; Cooper et al., 2018), and further trait mindfulness has mediated the relationship between emotion dysregulation and trauma (Reffi et al., 2019). Continued research should evaluate whether early utilization of mindfulness when emotional clarity is limited might improve one's emotional response to trauma cues and subsequent improvement in PTSD symptom severity generally.

The current study also extended the understanding of depression and emotion dysregulation following traumatic injury. Emotion dysregulation maintains depressive symptomatology, as individuals struggle to regulate negative affective states which reinforce negative appraisals of themselves and their experiences (Berking et al., 2014). Further, deficits in regulation perpetuate the perception that emotions are outside of one's control and their inability to be regulated using skills (Berking et al., 2014). As such, a deficit in accessing strategies is critical to self-efficacy in managing low mood and depressive symptoms after trauma. Although limited work has examined depression symptom severity and emotion dysregulation immediately following trauma, research on comorbid depression and anxiety highlights how dysregulation can perpetuate low mood and fear responses. This may explain, in part, the relationship observed in the current study between depression severity and skills deficits in the aftermath of trauma. For example, experiencing greater hopelessness and dysphoria can reinforce lack of confidence in managing fear, inhibiting overall recovery (Alloy et al., 1990). Conversely, emotion regulation skills training has been effective at improving both depression and anxiety symptoms (Wirtz et al., 2014). Additionally, when combining emotion regulation skills training

with cognitive behavioural therapy for veterans with PTSD, skills training reduced negative appraisals compared to those without skills training (Bryant et al., 2013). Therefore, goals for intervention might focus on emotion regulation skills training to not only build a repertoire of coping strategies but to also reduce hopelessness and negative appraisals that perpetuate depression.

Of interest, our hypotheses regarding other aspects of emotion dysregulation were not supported, despite past research implicating other aspects of emotion dysregulation in predicting both PTSD and depression symptom severity. For instance, lack of access to strategies was predictive of depressive symptom severity but not PTSD symptom severity; wherein previous studies, lack of access to strategies, was associated with PTSD symptoms (Forbes et al., 2020; Pugach et al., 2020; Tull et al., 2007). This could be due to the acuity of trauma exposure. For example, while the current study and previous work typically explore trait levels of dysregulation skills, individuals often demonstrate variability in how they regulate emotions at the state level (e.g. Chesney et al., 2019). Individual's ability to pull on specific regulation strategies acutely may be less related to their PTSD symptoms and how they cope generally.

Similarly, both non-acceptance of emotion and impulse control difficulty were not predictive of PTSD symptom severity, despite previous findings. Again, this could be due to the acuity of trauma exposure, and that assessment two weeks post-trauma exposure may not accurately capture how potential deficits go on to impact mental health recovery. However, this may also be due to the evidenced importance of emotional clarity in predicting symptomatology. Given the foundational importance of understanding one's emotions to adaptively regulate, it is possible that deficits in emotional clarity diminish one's ability to understand other facets of dysregulation (Boden et al., 2012; Feldman Barret & Gross, 2001; Lane et al., 2022). Similarly, this could be connected to the finding that the DERS-SF subscale of emotional awareness deficits was unrelated to other aspects of regulation, apart from difficulties with goals, and outcome variables. With items assessing an individuals' ability and desire to attend to their emotions, deficits in emotional awareness might influence their understanding of regulation, influence their experience following trauma, as well as their understanding of recovery (Frewen et al., 2012; Lane et al., 2022). Indeed, improved emotional clarity is proposed to reduce PTSD symptom severity and relate to attention to emotions, again suggesting that it may be challenging to be aware of emotions as well as evaluate the effects of other components of dysregulation when clarity is impaired (Boden et al., 2012; Vine & Aldao, 2014). Future work may address this by either exploring state regulation skills or by evaluating emotion

regulation deficits across time after trauma (e.g. ecological momentary assessment).

The current study is not without limitations. Although data were collected longitudinally to draw conclusions regarding baseline emotion dysregulation and subsequent psychopathology, we were unable to assess emotion dysregulation prior to injury, and emotion dysregulation was evaluated shortly after injury, which is a period in which emotion dysregulation is prominent. Further, emotion dysregulation was assessed via self-report, which is common practice in the literature, though it may be susceptible to demand characteristics or could be impacted by dysregulation itself (e.g. alexithymia). Similarly, the focus of the current study was self-reported symptom severity, which limits our understanding regarding dysregulation predicting diagnostic rates. Additionally, while the current study aim was to explore a traumatically injured sample, this limits generalizability to trauma populations exposed to other forms of trauma as well as our understanding of recovery outside of this acute phase. Lastly, post-hoc power analyses suggest fair power related to depression and PTSD. Yet, we were underpowered when examining symptoms clusters (power = .53–.73), suggesting that while we found current relationships between symptoms and emotional clarity, there may be additional relationships between emotion dysregulation and PTSD symptom clusters which should continue to be explored.

Overall, the current study highlights that after traumatic injury, emotion dysregulation broadly and, in particular, lack of emotional clarity and limited access to strategies, are critical aspects of emotion dysregulation that predict subsequent PTSD and depression symptom severity. Healthcare professionals in Trauma Centres can support patients in validating and attending to their emotions as they navigate the impact of physical and emotional trauma. Further, this work demonstrates how emotion dysregulation may shape mental health recovery and intervention after injury. Promoting emotion regulation post-trauma, such as with mindfulness-based interventions, may be especially impactful and provide treatment goals for early intervention targeting emotional clarity and the development of emotion regulation skills to address specific symptom presentations.

Disclosure statement

No potential conflict of interest was reported by the author(s).

Data availability statement

The data that support the findings of this study have been submitted and stored with NIMH in the RDoCdb repository as a requirement of receiving funding and are available from the senior author, TAD, upon reasonable request.

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