3-2022

Psychophysiological Predictors of Change in Emotion Dysregulation 6 Months after Traumatic Injury

Jacklynn M. Fitzgerald
Marquette University, jacklynn.fitzgerald@marquette.edu

Sydney Clare Timmer-Murillo
Medical College of Wisconsin

Claire Sheeran
Marquette University

Hailey Begg
Marquette University

Morgan Christoph
Marquette University

See next page for additional authors

International Journal of Psychophysiology, Vol. 173 (March 2022): 29-37. DOI.

Psychophysiological Predictors of Change in Emotion Dysregulation 6 Months after Traumatic Injury

Jacklynn M. Fitzgerald
Marquette University
Sydney Timmer-Murillo
Medical College of Wisconsin
Claire Sheeran
Marquette University
Hailey Begg
Marquette University
Morgan Cristoph
Marquette University
Terri A. deRoon-Cassini
Medical College of Wisconsin
Christine L. Larson
University of Wisconsin-Milwaukee

Abstract

Emotion dysregulation that occurs after trauma conveys risk for multiple disorders, including posttraumatic stress disorder, depression, and anxiety. Psychophysiological data (e.g., skin conductance level [SCL]) may be a useful biomarker for quantifying emotion dysregulation given that autonomic nervous system (ANS)-mediated arousal may underlie this feature. In this longitudinal study, we tested whether SCL collected following a single-incident traumatic injury could predict changes in emotion dysregulation over 6 months. Sixty-six adults were recruited from the emergency department; SCL was quantified during an active trauma narrative, in which participants re-told their traumatic event to a research staff member, as well as a neutral narrative for a control condition. Change in SCL (ΔSCL) was calculated using a maximum activation – minimum activation difference score. Multilevel linear modeling was used to test ΔSCL as a predictor of emotion dysregulation using the Emotion Dysregulation Scale (EDS) over time (3 timepoints over 6 months). Results showed that greater ΔSCL – indicative of increasing arousal– during both the trauma (p = 0.037) and neutral (p = 0.013) narratives was a significant predictor of greater emotion dysregulation at each subsequent timepoint. Further, we found a ΔSCL by time interaction, such that less ΔSCL during the neutral narrative predicted decreased emotion dysregulation over time (b = −1.26, SE = 0.43, t = −2.91, p = 0.004). Results validate the use of lab-based assessments of arousal to study emotion dysregulation in trauma survivors. That recovery from emotion dysregulation was predicted by less arousal during a neutral event underscores the importance of clinically targeting response to safety in trauma survivors.

Keywords

Trauma, Emotion, Emotion dysregulation, Arousal, Psychophysiology, Skin conductance

1. Introduction

While emotions are fundamental to successful navigation of our world, it is equally critical to manage or regulate emotion effectively. Indeed, whereas emotion regulation encompasses how we modulate our emotions to fit the current demands of a situation, *emotion dysregulation* involves the inability or deficiency in deciphering one's emotional state and/or the ability to regulate the intensity, duration or onset of an emotional experience (Gross, 1998). Emotion dysregulation is associated with the presence of negative life outcomes, including increased stress, negative interpersonal interactions, and the presence of psychopathology (Chesney et al., 2019; DeSteno et al., 2013; Tull et al., 2007). Specifically, emotion dysregulation has been identified as a key feature of many disorders (e.g. Major Depressive Episodes, Borderline Personality Disorder) and its presence may maintain pathology (Stepp et al., 2014). The centrality of emotion dysregulation has even led to the hypothesis that emotion dysregulation may serve as a transdiagnostic feature of psychopathology, cutting across both internalizing and externalizing disorders (Fernandez et al., 2016; Fitzgerald et al., 2019; MacNamara et al., 2017). In this way, emotion dysregulation is an important area of psychiatric research broadly-defined.

Emotion dysregulation has also been linked to the development of mental illness after trauma exposure, specifically posttraumatic stress disorder (PTSD; Fitzgerald et al., 2018; Seligowski et al., 2015), depression (Lopez et al., 2021), and substance abuse (Ghorbani et al., 2019). Specifically, self-reported emotion dysregulation prospectively predicts varied PTSD trajectories following traumatic injury based on symptom change, in that greater dysregulation predicts individuals who experience chronic symptoms over 1 year (Pencea et al., 2020). In this case, symptoms of emotion dysregulation at the time of trauma is also a more robust predictor of chronic PTSD status above and beyond severity of PTSD at the injury timepoint (Pencea et al., 2020). Critically, emotion dysregulation may be defined by the presence of multiple disturbances. In the case of PTSD,

emotion dysregulation may manifest as atypical fear responding (Hopper et al., 2007; Rauch et al., 2006), greater attentional bias to threat (Fani et al., 2012), deficiency in knowing when stimuli in the environment are innocuous, or not related to threat (Rabinak et al., 2017), and *decreased* emotion that is incongruent to environmental circumstance (Lanius et al., 2010; Nawijn et al., 2015). In addition, individuals with PTSD exhibit negative emotions that are considered hallmarks of the disorder, including anger, guilt and shame (McLean and Foa, 2017). Emotion dysregulation is therefore related to more than one emotion, symptom or symptom cluster of PTSD (American Psychiatric Association, 2013). In contrast, changes in emotion dysregulation over the administration of prolonged exposure (PE) therapy is associated with a reduction in depressive – but not PTSD – symptoms in women veterans (Lopez et al., 2021). This suggests that while emotion dysregulation is related to multiple symptoms of PTSD, it is also not narrowly linked to this disorder; rather, it is associated with multiple trauma-related outcomes.

Nevertheless, given its influence in PTSD, evidence-based treatments of PTSD, such as PE therapy, target emotion dysregulation for treatment. Treating emotion dysregulation is a critical component to PE, often taking the form of targeted physiological regulation (M. B. Powers et al., 2010), whereby an over-active fear response and hyperarousal is targeted and those with PTSD seek to engage physiological regulation of their emotions (Hembree et al., 2003). Conversely, emotion regulation difficulties, defined by self-report, is associated with greater dropout during PE (Gilmore et al., 2020). Prior research has also linked PE efficacy to reductions in emotion dysregulation (Jerud et al., 2016) and, more specifically, elevated arousal response as a marker of this symptom (Maples-Keller et al., 2019). This suggests that emotion dysregulation may manifest as increased arousal in some cases. Indeed, physiological markers of arousal such as increased heart rate, respiration rate and skin conductance (SC) are evident post-trauma (Hinrichs et al., 2019; Seligowski et al., 2019), while heightened skin conductance level (SCL) during a trauma interview is predictive of PTSD symptoms (Hinrichs et al., 2019). Notably, this work demonstrates that SCRs measured during the trauma interview can reliability predict individuals who go onto develop non-remitting PTSD vs. individuals who recover as well as those who were deemed resilient over 1 year following traumatic injury (Hinrichs et al., 2019). Other cross-sectional work reports a positive correlation between SC responses to safety signals (e.g., shapes that were never paired with threat in a fear conditioning paradigm) with symptoms of PTSD (Seligowski et al., 2019). Further, this effect exists in individuals who have experienced trauma irrespective of PTSD diagnosis, while non-differential SCR to threat and safety signals was evident in trauma survivors but absent in a control group (Kreutzmann et al., 2021). Together, this suggests a link between physiological arousal, emotion dysregulation, and PTSD, but indicates that arousal responses may be non-specific in those who have experienced trauma as evidenced by greater SCRs to threatening – as well as neutral – events.

Accordingly, emotion dysregulation is instrumental to the etiology and treatment of trauma-induced psychopathology. Yet, research often fails to acknowledge that emotion dysregulation is labile, meaning that it is not a fixed trait. Fluctuations in emotion – as well as its regulation and dysregulation – occur in response to daily life and are often reflective of the context and demands of a situation (Cole et al., 2019). As has been theorized, emotion regulation may operate like a thermostat, a mechanism that is constantly fluctuating or adjusting to account for the moment-to-moment demands of the environment (Cole et al., 2019). While developmental research often acknowledges the dynamic aspects of emotion regulation (Martin and Ochsner, 2016), this work is lacking in adult populations and particularly in populations where emotion regulation capacity is expected to change in response environmental factors, such as a recent trauma. This is despite literature showing the importance of flexibility in harnessing emotion regulation in order to overcome the demands of life (Chesney and Gordon, 2017). As such, there is a greater need to recognize and account for the changes that may occur in emotion dysregulation following trauma. A central question is how to predict changes in emotion dysregulation over time, particularly after a traumatic event.

Given the centrality of emotion dysregulation to the study of post-trauma outcomes, the current study was designed to investigate predictors of emotion dysregulation after trauma. Self-reported emotion dysregulation correlates with elevated SCL in healthy individuals (Matejka et al., 2013) and in other disorders, such as borderline personality disorder (Kaufman et al., 2021); yet – to our knowledge – there has been no work exploring SCL as a predictor of emotion dysregulation in trauma survivors. Therefore, the current study tested whether physiological arousal experienced during the re-telling of a personal trauma in the acute aftermath of this event was related to changes emotion dysregulation in the 6 months that followed. Given prior work showing that heightened arousal in trauma survivors may occur outside of the context of threat (Kreutzmann et al., 2021; Seligowski et al., 2019), we tested whether physiological arousal experienced during the re-telling of a neutral event unrelated to the trauma incident was also related to changes in emotion dysregulation. We hypothesized that greater rise in SCL during the recounting of these events, as a marker of physiological arousal, would be prospectively and positively associated to greater severity of emotion dysregulation in trauma survivors. We further hypothesized that greater rise in SCL would be indicative of changes in emotion dysregulation, such that it would be linked to increased severity of dysregulation over time.

2. Material and methods

2.1. Participants

Participants included in this analysis were drawn from a larger study of traumatically injured adults admitted to a Level 1 Trauma Center Emergency Department (ED) for the investigation of neurobiological predictors of PTSD. Thus, all participants in this study were at elevated risk for PTSD and were recruited based on the following inclusion criteria: a) were between the ages of 18 and 60, b) were English speaking, c) met the Diagnostic and Statistical Manual-5th edition (DSM-5) Criterion A for trauma exposure that puts them at-risk for a PTSD diagnosis, and d) exhibited a greater risk of developing PTSD based on a minimum score of 3 (out of 5) on the Predicting PTSD Questionnaire (Rothbaum et al., 2014). Participants were excluded if they: a) experienced a moderate or severe head injury as the result of their trauma based on a score of >13 on the Glasgow Coma Scale (Sternbach, 2000; Teasdale et al., 2014), b) suffered a spinal cord injury with neurological deficit, c) were admitted to the ED as the result of intentional self-inflicted injury, d) exhibited severe vision or hearing impairments, e) had a history of psychotic or manic symptoms or were currently taking antipsychotic medications, f) a history of clear substance abuse, g) on police hold following their traumatic injury, or h) were MRI incompatible based on the following and due to the use of MRI for other components of the study: presence of ferromagnetic material in the body, claustrophobia, inability to lie still for 2 h, or either currently pregnant or trying to become pregnant. All participants provided written consent and all study procedures were approved by the local Institutional Review Board. Participants were compensated for their time and all procedures complied with the Helsinki Declaration.

2.2. Procedure

Participants completed a post-injury in-person appointment within 1 month of their traumatic injury. At this visit, participants were asked to engage in a trauma narrative, whereby they told the story of the traumatic event that led them to the ED to a research staff member (e.g., “trauma narrative”). In addition, participants completed a second narrative in which they told the story of a recent innocuous event, such as a recent trip to the grocery store (e.g., “neutral narrative”). This second, neutral narrative, was used as a comparison in analysis and followed immediately the trauma narrative in all cases. In both narratives, participants were allowed to freely describe what happened to them and follow-up questions were asked when participants stopped talking in order to maintain focus on the events as they occurred. Participants were encouraged to talk for a minimum of 1 min but were not given a time limit. All sessions were audio recorded so that details of the event could be transcribed by a trained research staff member to create a condensed 30-second voice recording, narrated by a

research staff member describing the incident. These audio recordings were then played to the participants during a subsequent functional magnetic resonance imaging (fMRI) scan to isolate brain functioning related to recollection of personalized traumatic events (vs. personalized neutral events). Results of the fMRI study as it pertains to the development of PTSD, including analysis of SCL during the narratives as predictors of PTSD, are the focus of a subsequent publication and are therefore not included in this analysis.During the procedure, participants were seated while continuous SCL was recorded using the eSense SC system (Mindfield Biosystems, Inc., Berlin, Germany) using the eSense software (version 4.5.9) connected to an iPhone (iOS version 12) and two electrodes attached to the middle and index fingers. eSense SC is highly correlated with SC collected from the gold-standard Biopac system for psychophysiological data collection (Hinrichs et al., 2017) and this set-up has been used previously in individuals admitted to the ER for traumatic injury. Prior research has found that eSense SCL is positively correlated with severity of PTSD symptoms (Hinrichs et al., 2017). SCL was collected continuously during trauma and neutral narratives at a sampling rate of 5 Hz.

In addition to completing the trauma and neutral narratives, participants completed the Emotion Dysregulation Scale (EDS). The EDS is comprised of 12 items that assess self-reported emotional experiences, cognitions, and behaviors (A. Powers et al., 2015). Participants are prompted to rate how true items are from “not at all” to “very true” using a 7-point Likert scale. A sample item is “It's often hard for me to calm down when I'm upset” and total scores range from 12 to 84. The EDS has good construct validity in that it is highly correlated with other measures of emotion dysregulation (e.g., Difficulties in Emotion Regulation Scale) and related to psychopathology (e.g., depression, posttraumatic stress, substance abuse) (A. Powers et al., 2015). Participants returned for in-person follow-ups at 3-months and 6-months, at which time they again completed the EDS. EDS total scores at each timepoint were calculated based on measure-specific scoring guidelines; greater EDS scores reflect greater severity of emotion dysregulation.

2.3. Data analysis

Severity of emotion dysregulation, measured by the EDS, was used as the primary dependent variable in two separate multilevel linear models (MLM) to examine the slope of emotion dysregulation across time within individuals. In each MLM, change in SCL (ΔSCL in microsiemens [μS]) was calculated using a maximum – minimum difference score (greater values = greater increase in SCL during narrative) and used as the independent predictor of-interest, similar to other methods (Hinrichs et al., 2017, Hinrichs et al., 2019). We examined ΔSCL during the trauma and neutral narratives in separate models given high concordance between these variables ($r(64) = 0.70$, $p < 0.001$).

In each model, the impact of time was examined in the first level to determine whether emotion dysregulation changed over time. Subsequently, ΔSCL during each narrative was examined in the second level as a predictor of changes in emotion dysregulation over time. Covariates in both models included gender and age, as prior research has demonstrated age (Nolen-Hoeksema and Aldao, 2011) and gender (McRae et al., 2008) differences in the use of emotion regulation. Duration (in seconds) of each narrative was added as an additional covariate given substantial variability across individuals.

All continuous predictors were grand-mean centered and gender was effects coded (−1 = male; 1 = female). Time was coded as a three level variable (0 = post-injury; 3 = 3-months; 6 = 6-months) and significant interactions with time were followed-up using a standard simple slopes approach (Holmbeck, 2002): ±1 standard deviation of ΔSCL was calculated and the MLM models were re-run evaluating the effect of time at high and low levels of ΔSCL.

3. Results

3.1. Participants

A total of *N* = 123 participants completed post-injury testing appointments including the collection of SCL during the trauma narrative. Of these, 36 participants did not have usable SCL data either because of recording issues resulting in data loss (*n* = 2) or data was poor in quality (*n* = 34). Unusable data included nonwavering SCL values across the recording (e.g., a static value) or instances of spiking in which values of >100 μS were recorded sporadically throughout the session, suggesting ill-fitting electrodes and non-plausible data collection. Of the remaining *n* = 87 individuals who had usable SCL data during the trauma narrative, *n* = 16 did not have EDS scores available at all three timepoints (post-injury, 3 months, 6 months) and *n* = 5 did not complete the neutral narrative and were thus excluded from analysis. Thus, *N* = 66 individuals were retained for analysis. There were no differences in gender ($p > 0.812$), age ($p > 0.425$), or EDS at any timepoint (p's > 0.134) between individuals with retained versus discarded SCL data due to poor quality. In addition, individuals retained for analysis versus lost to follow-up for EDS collection did not differ in post-injury EDS scores ($p > 0.222$); however, individuals lost to follow-up were significantly younger ($t(85) = 1.80$, $p = 0.038$).

Participants in the analysis completed their post-injury appointment between 3 and 30 days after injury (M = 15.60 days, SD = 5.43 days) and their follow-up appointments between 2 and 4 months (M = 3.15 months, SD = 12.75 days) for the 3-month timepoint and 4–7 months (M = 6.07 months; SD = 10.90 days) for the 6-month timepoint. Mechanism of injury varied across the sample but consisted primarily of survivors of motor vehicle crashes (64%). The remaining injuries were classified as: assault (11%), pedestrian injuries (6%), falls (<5%), motor cycle crash (<5%), stab wounds (<5%), and domestic violence (<5%) (exact percentage is not included to ensure participant confidentiality). Complete participant demographics are reported in Table 1.

Table 1. Sample demographics (*N* = 66).

	***M* (*SD*)**		
	Post-injury	**3 Months**	**6 Months**
Age	32.93 (10.57)	–	–
EDS	35.22 (18.40)	35.50 (19.32)	31.98 (19.09)
PCL-5	26.16 (18.34)	22.36 (16.72)	19.77 (17.87
DASS-Depression	8.62 (8.72)	10.28 (10.97)	7.54 (10.69)
DASS-Anxiety	8.55 (7.77)	8.19 (8.50)	7.82 (9.52)
DASS-Stress	11.66 (9/52)	12.69 (9.90)	10.62 (9.48)
		n (%)	
Gender (female)	35 (53%)	–	–
Ethnicity (Hispanic or Latino)	5 (8%)	–	–
Race			
Asian	<5 (<5%)	–	–
Black or African American	37 (56%)	–	–
White	22 (33%)	–	–
More than one race	<5 (<5%)	–	–
Unknown or not reported	<5 (<5%)	–	–
Mechanism of injury			
Motor vehicle crash	42 (64%)		
Assault	7 (11%)		
Pedestrian injury	4 (6%)		
Fall	<5 (<5%)		
Motor cycle crash	<5 (<5%)		
Stab wound	<5 (<5%)		

Domestic violence	<5 (<5%)		

Note. EDS = Emotion Dysregulation Scale; PCL-5 = PTSD Checklist for DSM-5; DASS = Depression, Anxiety, and Stress Scale. Small sample sizes for select racial groups are reported as <5% to avoid participant identification; thus, cumulative percentage surpasses 100% as reported here.

3.2. EDS

At the post-injury timepoint, EDS scores ranged from 12 to 82 (M = 35.22, SD = 18.40), 12–81 at 3 months (M = 35.50, SD = 19.32), and 12–83 at 6 months (M = 31.98, SD = 19.09), indicating considerable range in emotion dysregulation at all three timepoints.

3.3. ΔSCL during trauma narrative

The length of participant's trauma narratives ranged between 1 and 20 min (M = 10.78, SD = 4.01). On average, the minimum SCL occurred at 2.41 min (SD = 3.91) and the maximum SCL occurred at 6.24 min (SD = 4.60). Accounting for individual variability in narrative length, minimum SCL occurred on average 22.70% of the way through the narrative and maximum SCL occurred on average 57.34% of the way through the narrative. The points at which the minimum and maximum SCLs occurred differed significantly ($t(64) = 4.68$, $p < 0.001$). Additional descriptive statistics for SCL and ΔSCL during the trauma narrative are reported in Table 2; the time at which participants achieved their minimum and maximum SCLs is depicted in Supplemental Fig. 1.

Results of the MLM testing ΔSCL during the trauma narrative as a predictor of EDS are presented in Table 3. In the first model examining predictors of EDS scores over time, there was a significant effect of ΔSCL, such that greater ΔSCL – reflecting greater rise in SCL during the trauma narrative – predicted greater EDS scores across time ($b = 5.80$, SE = 2.26, $t = 2.57$, $p = 0.013$). In the second model examining interactions with time, we did not find a significant interaction between ΔSCL and time ($p > 0.156$). That is, ΔSCL was related to greater emotion dysregulation at each timepoint, but it was not associated with changes in emotion dysregulation as a function of time. Fig. 1 (Panels A-C) depict the cross-sectional relationship between ΔSCL during the trauma narrative and EDS scores at each timepoint.

Table 2. Skin conductance level (SCL) values.

	SCL		***ΔSCL***	
	Trauma narrative	**Neutral narrative**	**Trauma narrative**	**Neutral narrative**
Minimum	0.43	0.47	0.10	0.04
Maximum	9.39	8.05	4.17	3.61
Mean	2.09	2.41	1.16	0.94
Standard deviation	1.36	1.47	0.96	0.73

Note. ΔSCL calculated using a maximum - minimum difference score. All values are reported in microSiemems (μS).

Table 3. Mixed growth models examining impact of SCL during trauma narrative on EDS over 6 months.

Variable	**EDS**				Empty Cell
	b	***SE***	***t***	***p*-Value**	
Step 1					
Intercept	36.09	3.05	11.85	<0.001	***
Time	−0.49	0.32	−1.52	0.130	
Gender	−0.83	4.01	−0.21	0.837	
Age	−0.20	0.20	−1.03	0.309	
Duration of narrative (seconds)	0.00	0.01	−0.31	0.759	
ΔSCL	5.80	2.26	2.57	0.013	*
Step 2					

Intercept	35.29	2.12	16.66	<0.001	
Gender × Time	−0.61	0.41	−1.46	0.146	
Age × Time	−0.05	0.03	−1.81	0.072	
Duration of narrative (seconds) × Time	0.00	0.00	−2.04	0.043	
ΔSCL × Time	0.48	0.34	1.42	0.157	

*** $p < 0.001$. ** $p < 0.01$. * $p < 0.05$.

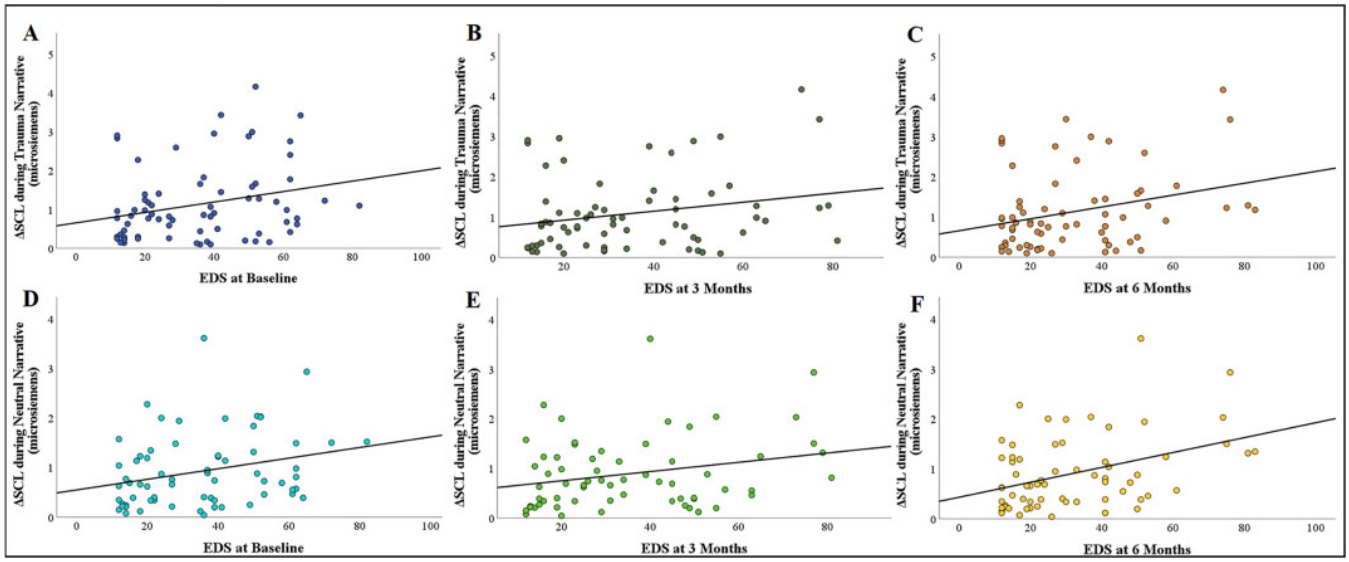

Fig. 1. Scatterplots depicting relationship between ΔSCL during trauma narrative and EDS scores at (A) post-injury, (B) 3 months, and (C) 6 months as well as ΔSCL during neutral narrative and EDS scores at (D) post-injury, (E) 3 months, and (F) 6 months. Plots indicate that that greater rise in skin conductance response during the narratives was associated with greater emotion dysregulation severity at each timepoint. Note: EDS = Emotion Dysregulation Scale.

3.4. ΔSCL during neutral narrative

The length of participant's neutral narratives ranged between 1.5 and 15 min (M = 6.94, SD = 2.10). On average, the minimum SCL occurred at 3.25 min (SD = 2.82) and the maximum SCL occurred at 3.48 min (SD = 2.48). Accounting for individual variability in narrative length, minimum SCL occurred on average 49.49% of the way through the narrative and maximum SCL occurred on average 49.88% of the way through the narrative. The points at which the minimum and maximum SCLs occurred did not differ ($p > 0.340$). Descriptive statistics for SCL and ΔSCL during the neutral narrative are reported in Table 2; the time at which participants achieved their minimum and maximum SCLs is depicted in Supplemental Fig. 1.

Results of the MLM testing ΔSCL during the neutral narrative as a predictor of EDS scores are presented in Table 4. Identical to results from the trauma narrative, we found a significant effect of ΔSCL in the first model, such that greater ΔSCL – reflecting greater rise in SCL during the neutral narrative – predicted greater EDS scores across time ($b = 6.88$, SE = 2.84, $t = 2.42$, $p = 0.019$). Fig. 1 (Panels D–F) depict the cross-sectional relationship between ΔSCL during the neutral narrative and EDS scores at each timepoint.

Table 4. Mixed growth models examining impact of SCL during neutral narrative on EDS over 6-months.

Variable	**EDS**				
	b	***SE***	***t***	***p*-Value**	
Step 1					
Intercept	36.45	3.08	11.84	<0.001	***

Time	−0.50	0.33	−1.52	0.132	
Gender	−0.98	3.99	−0.25	0.807	
Age	−0.23	0.20	−1.19	0.241	
Duration of narrative (seconds)	0.00	0.02	0.14	0.888	
ΔSCL	6.88	2.84	2.42	0.019	*
Step 2					
Intercept	35.84	2.09	17.15	<0.001	***
Gender × Time	−0.74	0.42	−1.77	0.079	
Age × Time	−0.06	0.03	−1.95	0.053	
Duration of narrative (seconds) × Time	0.00	0.00	0.13	0.894	
ΔSCL × Time	0.93	0.42	2.21	0.029	*

Note. SCL = Skin Conductance Level; ΔSCL = change in SCL during narrative; EDS = Emotion Dysregulation Scale; b = beta estimate; SE = standard error. ** $p < 0.01$. ∗∗∗$p < 0.001$. ∗$p < 0.05$.

In the second model exploring the interaction with time, there was a significant interaction between ΔSCL and time ($b = 0.934$, SE = 0.42, $t = 2.21$, $p = 0.029$). Follow-up simple slopes analyses revealed that smaller ΔSCL – reflecting smaller rise in SCL during the neutral narrative – was related to a decline in emotion dysregulation over time ($b = -1.26$, SE = 0.43, $t = -2.91$, $p = 0.004$). By contrast, there was no change in emotion dysregulation over time for relatively larger ΔSCL ($p > 0.531$), meaning that emotion dysregulation did not decline for individuals that experienced increased SCL during the neutral narrative (Fig. 2).

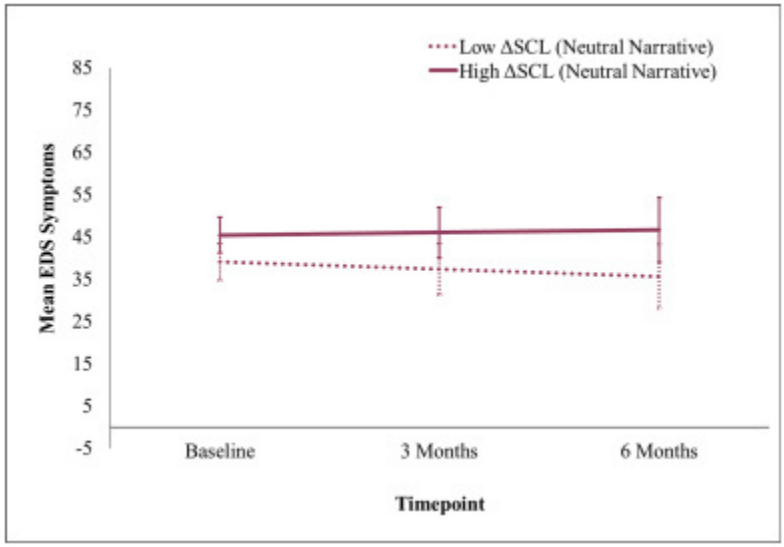

Fig. 2. Change in EDS over time for individuals with relatively larger versus smaller ΔSCL during the neutral narrative. Compared to individuals with relatively larger, indicating greater rise in SCL during the neutral narrative, individuals with relatively smaller ΔSCL declined in EDS scores over time. Note: Relative smaller versus larger ΔSCL defined by ±1 standard deviation; age and gender were included as covariates in obtaining relative ΔSCL values; SCL = skin conductance level; EDS = Emotion Dysregulation Scale.

4. Discussion

In this study we tested the utility of skin conductance, a physiological marker of arousal, as a predictor of emotion dysregulation and their fluctuation over 6 months following a traumatic injury. Three important

findings emerged from this investigation: first, we found that emotion dysregulation declined over 6 months following injury, indicating that – at the group level – emotion dysregulation is not static. Second, greater rise in physiological arousal to *both* the trauma and neutral narratives was a significant predictor of greater emotion dysregulation across the 6 months. Finally, smaller rise in skin conductance during the neutral narrative (i.e., the re-telling of an innocuous event) foretold decline in emotion dysregulation over 6 months. Comparatively, relatively larger rise in arousal during the neutral event was related to no change in emotion dysregulation across 6 months. Combined, these findings demonstrate the importance in considering natural changes in emotion dysregulation in the months that follow a traumatic event, as well as the utility in lab-based measures of physiological arousal as a predictor of such changes.

Emotion dysregulation is a cardinal feature of stress-related psychopathology (Fitzgerald et al., 2018; Forbes et al., 2020a; Weis et al., 2021) and self-reported emotion dysregulation predicts future PTSD severity in the days (Orcutt et al., 2014) and months after trauma exposure (Bardeen et al., 2013; Forbes et al., 2020a; Jenness et al., 2016; Miron et al., 2014) as well as after completion of common therapeutics, such as PE (Cloitre et al., 2016). Other research demonstrates that increased difficulty in regulating emotion coincides with increased depression severity in individuals who have experienced military sexual trauma (Lopez et al., 2021) and that *adaptive* vs. maladaptive emotion regulation skills after trauma is significantly associated with a lower probability of developing PTSD (Weiss et al., 2020). Further, emotion dysregulation mediates (Choi and Oh, 2014; Forkus et al., 2020; Janiri et al., 2021; Michopoulos et al., 2015, Michopoulos et al., 2015) the relationship between trauma and emergence of stress-related psychopathology, while emotion dysregulation may also be linked to experiencing additional trauma among sexual assault survivors (Villalta et al., 2020). However, despite its influence on trauma-related outcomes, relatively few studies have centered on emotion dysregulation as the target of inquiry and/or investigated clinical, behavioral, or biological predictors of changes in emotion dysregulation. Thus, the findings presented herein are relatively novel in this regard and provide evidence in support of emotion dysregulation as an important area of research (Aldao, 2013).

A significant relationship between a self-report measure of emotion dysregulation and a biological-based marker of emotion dysregulation is consistent with prior research. Specifically, our findings are congruent with those involving other known biomarkers of emotion dysregulation, such as low heart rate variability (HRV) (A. Powers et al., 2021). HRV is a commonly used marker of non-linear adaptation of the heart to threat and has been used as an individual-differences marker in the body's adaptability to stress, with lower HRV associated with emotion dysregulation (Fantini-Hauwel et al., 2020). Thus, we extend findings showing utility in measuring self-reported emotion dysregulation in that it is linked to higher physiological arousal via skin conductance as well. Notably, PTSD severity does not correlate with HRV in all instances (A. Powers et al., 2021); thus, self-reported emotion dysregulation may be a more useful clinic-based measurement of underlying biological changes associated with adaptive stress responding (A. Powers et al., 2021) which may or may not transpire into full-blown illness.

The current study yields important implications regarding emotion dysregulation in the context of trauma and intervention. To date, a majority of the literature examines the role that emotion dysregulation and SCL play when assessing for PTSD risk. However, understanding emotion dysregulation post-trauma is critical as it may be tied to treatment outcomes. Indeed, predicting heightened dysregulation may allow for targeting novel intervention towards those most dysregulated to reduced dropout rates and to improve engagement in treatment (Gilmore et al., 2020). Likewise, the literature is increasingly acknowledging variability in PTSD presentations, underscoring a potential need for tailored individualized intervention (Galatzer-Levy and Bryant, 2013). Therefore, recognition that while emotion dysregulation is a feature of PTSD broadly but may also be more closely tied to treatment outcomes as a feature itself is critical. As such, current findings emphasize the utility of using SCL as a screening tool for subsequent dysregulation and support targeted intervention for those who may fall into the more at-risk group of persistent problems in regulating emotional states.

The utility of measuring emotion dysregulation separately is further supported by the fact that emotion dysregulation is often a main target of treatments for stress-related disorders, specifically, the most common "gold-standard" intervention for treating PTSD: PE. The foundational pieces of PE consist of (a) exposing patients to in vivo exposures of their personal trauma-related situations and (b) re-imagining the trauma (Foa and Kozak, 1986). The theorized mechanism of neurobiological change in PE centers on the activation of fear and subsequent forming of new associations between in vivo exposures and/or re-imaginings with safety, a process that intrinsically regulates negative affect (Foa and Kozak, 1986). Such treatments and their mechanisms maintain a focus on emotion dysregulation. Indeed, trauma recollections lead to the experience of negative affect that, when unchecked, leads to multitudinous negative health outcomes. Active management of negative affect triggered by trauma recollections – by contrast – is adaptive. All told, emotion dysregulation is therefore a worthwhile target of study, bolstered by its centrality in common therapeutics for stress-related disorders. Thus, the relationship between SCL and emotion dysregulation in those who have experienced trauma provides more impetus for the study of emotion dysregulation for potential treatment outcomes, particularly for therapies that focus on physiological regulation, such as PE (Forbes et al., 2020b).

The fact that changes in arousal to the neutral narrative – and not in response to the trauma narrative – predicted changes in emotion dysregulation over time is particularly noteworthy. Of note, participants first engaged in the trauma narrative, followed by the neutral narrative in all cases. This sequence was chosen for clinical and ethical reasons: its intent was to give participants time to regulate negative affect that was intentionally spurred by recounting their trauma narrative. Results therefore suggest that individuals who were unable to regulate physiological arousal during the neutral narrative, which followed the trauma narrative, and instead showed paradoxical *increased* arousal response during this time, are those most at risk for persistent emotion dysregulation. This sequence of events may indicate that participants with persistent emotional dysregulation may have been delayed in their ability to recover from the trauma narrative. Indeed, on average, participant's achieved their maximum SCL later during the trauma narrative compared to their minimum SCL, suggesting an increase in SCL during the re-telling of the traumatic event. Thus, on average, participants began their neutral narrative with comparatively higher SCL. During the subsequent neutral narrative, individuals who declined in their SCL at a slower rate, unable to achieve a lower SCL, may therefore experience a failure to recover. However, we note that any such pattern of uniform decline was absent in the neutral narrative amidst greater individual variability in the timing of the maximum and minimum SCL values during this event (see supplemental material). Some individuals continued to experience an increase in SCL during this event. This suggests that the ΔSCL metric during the neutral narrative may also reflect an inappropriate increased arousal to neutral events. Difficulty in threat vs. safety discrimination is a well-known feature of PTSD (Norrholm and Jovanovic, 2018), while greater SCRs in response to safety signals is reported in trauma-exposed individuals (Kreutzmann et al., 2021). Future research that randomizes the administration of these narratives (i.e., neutral prior to trauma recall) are needed to better isolate these effects.

Indeed, as outlined above, emotion dysregulation is both the occurrence of hyper-responding to threat (e.g., during the re-telling of a traumatic event) as well as instances of incorrect responding to situations that are safe (e.g., during the re-telling of an innocuous event). For example, individuals with PTSD exhibit misplaced elevated arousal during conditions that are safe (Rabinak et al., 2017) and the inability to differentiate arousal response between threat and safety is a hallmark symptom of the disorder (Rabinak et al., 2017; van der Kolk, 1997). Other work shows that individuals with PTSD exhibit elevated response in brain regions that are the precursor of the physiological arousal response (e.g., the amygdala) in response to neutral imagery (Brunetti et al., 2015; Sambuco et al., 2020). Thus, mismatched arousal response to safety – exhibited by the neutral narrative in this study – seems most impactful for predicting changes in emotion dysregulation over time. Stated another way, if individuals do not show elevated arousal response to safety, they then decline in emotion dysregulation over time and may therefore be most protected against the development of psychopathology (e.g., PTSD). This

finding therefore means that the study of arousal response to neutral and safety cues is equally important for studying post-trauma outcomes in addition to arousal response to threat.

The present study is not without limitations. First, the eSense system is a validated method for accessible psychophysiology recording, but results should be confirmed using other standard systems, such as Biopac. Second, emotion dysregulation was calculated from one self-report measure, the EDS, which provided a short assessment of emotion dysregulation. This scale is positively correlated with other, longer measures of emotion dysregulation, such as the DERS. It has been found to measure the presence of emotion dysregulation behaviors and strategies but may be less equipped to measure emotional awareness (A. Powers et al., 2015). Thus, these results may be specific to the way in which arousal response predicts changes in some – but not all – aspects of emotion dysregulation. Related, the decline in emotion dysregulation over time in this study was minimal. Thus, despite the significant association between lower arousal response during neutral narrative and decline in emotion dysregulation over time, it is unclear how findings may translate to observable changes in emotion dysregulation that can be measured or tracked by behavior. Future research should also investigate ways in which changes in emotion dysregulation predicted by arousal response also relate to development of stress-related psychopathology, such as symptoms of PTSD. This is particularly true as average severity of PTSD symptoms in this sample remained low throughout the 6 months; thus, it is unclear how findings will replicate in a sample with diagnosed PTSD. Finally, as we did not obtain EDS scores on participants prior to their traumatic event, it is unclear how many of these participants were experiencing pre-existing emotion dysregulation and how this may have altered their trajectory of emotion dysregulation post-injury. Future research that can disentangle such effects (i.e., pre- vs. post-trauma onset) is needed.

5. Conclusion

To our knowledge, this is the first study to use a physiological marker of arousal to predict changes in emotion dysregulation over time in trauma survivors. Findings again establish the importance of studying emotion dysregulation in trauma survivors, in that we confirm a relationship between biological marker of arousal and self-reported emotion dysregulation. However, results may be most impactful in when considering the relationship between increased arousal during a neutral event and changes to emotion dysregulation in the months that follow trauma. This finding suggests that post-trauma outcomes related to the inability to regulate emotional states are shaped both by physiological response to threat as well as to safety.

The following is the supplementary data related to this article.Download : **Download Word document (109KB)**

Supplemental Fig. 1. Stacked histogram showing the time during the (A) trauma and (B) neutral narratives when the minimum and maximum SCL was achieved across participants. Note: SCL = skin conductance level.

Disclosures

This research was supported by a National Institute of Mental Health grant (R01 MH106574; PIs: Larson, deRoon-Cassini).

References

Aldao, A., 2013. The future of emotion regulation research: capturing context. Perspect. Psychol. Sci. 8 (2), 155–172. https://doi.org/10.1177/1745691612459518.

American Psychiatric Association, 2013. Diagnostic and Statistical Manual of Mental Disorders, 5th ed.

Bardeen, J.R., Kumpula, M.J., Orcutt, H.K., 2013. Emotion regulation difficulties as a prospective predictor of posttraumatic stress symptoms following a mass shooting. J. Anxiety Disord. 27 (2), 188–196. https://doi.org/10.1016/j.janxdis.2013.01.003.

Brunetti, M., Sepede, G., Ferretti, A., Mingoia, G., Romani, G.L., Babiloni, C., 2015. Response inhibition failure to visual stimuli paired with a “single-type” stressor in PTSD patients: an fMRI pilot study. Brain Res. Bull. 114, 20–30. https://doi.org/ 10.1016/j.brainresbull.2015.03.001.

Chesney, S.A., Gordon, N.S., 2017. Profiles of emotion regulation: understanding regulatory patterns and the implications for posttraumatic stress. Cognit. Emot. 31 (3), 598–606. https://doi.org/10.1080/02699931.2015.1126555.

Chesney, S.A., Timmer-Murillo, S.C., Gordon, N.S., 2019. Establishment and replication of emotion regulation profiles: implications for psychological health. Anxiety Stress Coping 32 (3), 329–345. https://doi.org/10.1080/10615806.2019.1573990.

Choi, J.Y., Oh, K.J., 2014. Cumulative childhood trauma and psychological maladjustment of sexually abused children in Korea: mediating effects of emotion regulation. Child Abuse Negl. 38 (2), 296–303. https://doi.org/10.1016/j. chiabu.2013.09.009.

Cloitre, M., Petkova, E., Su, Z., Weiss, B., 2016. Patient characteristics as a moderator of post-traumatic stress disorder treatment outcome: combining symptom burden and strengths. Br.J. Psychiatry Open 2 (2), 101–106. https://doi.org/10.1192/bjpo.bp.115.000745.

Cole, P.M., Ashana Ramsook, K., Ram, N., 2019. Emotion dysregulation as a dynamic process. Dev. Psychopathol. 31 (3), 1191–1201. https://doi.org/10.1017/ S0954579419000695.

DeSteno, D., Gross, J.J., Kubzansky, L., 2013. Affective science and health: the importance of emotion and emotion regulation. Health Psychol. 32 (5), 474–486. https://doi.org/10.1037/a0030259.

Fani, N., Jovanovic, T., Ely, T.D., Bradley, B., Gutman, D., Tone, E.B., Ressler, K.J., 2012. Neural correlates of attention bias to threat in post-traumatic stress disorder. Biol. Psychol. 90 (2), 134–142. https://doi.org/10.1016/j.biopsycho.2012.03.001.

Fantini-Hauwel, C., Batsel´e, E., Gois, C., Noel, X., 2020. Emotion regulation difficulties are not always associated with negative outcomes on women: the buffer effect of HRV. Front. Psychol. 11, 697. https://doi.org/10.3389/fpsyg.2020.00697.

Fernandez, K.C., Jazaieri, H., Gross, J.J., 2016. Emotion regulation: a transdiagnostic perspective on a new RDoC domain. Cogn. Ther. Res. 40 (3), 426–440. https://doi. org/10.1007/s10608-016-9772-2.

Fitzgerald, J.M., DiGangi, J.A., Phan, K.L., 2018. Functional neuroanatomy of emotion and its regulation in PTSD. Harv. Rev. Psychiatry 26 (3), 116–128.

Fitzgerald, J.M., Klumpp, H., Langenecker, S., Phan, K.L., 2019. Transdiagnostic neural correlates of volitional emotion regulation in anxiety and depression. Depress. Anxiety 36 (5), 453–464. https://doi.org/10.1002/da.22859.

Foa, E.B., Kozak, M.J., 1986. Emotional Processing of Fear: Exposure to Corrective Information, 16.

Forbes, C.N., Tull, M.T., Rapport, D., Xie, H., Kaminski, B., Wang, X., 2020. Emotion dysregulation prospectively predicts posttraumatic stress disorder symptom severity 3 months after trauma exposure. J. Trauma. Stress. 33 (6), 1007–1016. https://doi. org/10.1002/jts.22551.

Forbes, D., Bisson, J., Monson, C., Berliner, L., 2020. Effective Treatments for PTSD, Third edition. Guilford Press.

Forkus, S.R., Rosellini, A.J., Monteith, L.L., Contractor, A.A., Weiss, N.H., 2020. Military sexual trauma and alcohol misuse among military veterans: the roles of negative and positive emotion dysregulation. Psychol. Trauma Theory Res. Pract. Policy 12 (7), 716–724. https://doi.org/10.1037/tra0000604.

Galatzer-Levy, I.R., Bryant, R.A., 2013. 636,120 ways to have posttraumatic stress disorder. Perspect. Psychol. Sci. 8 (6), 651–662.

Ghorbani, F., Khosravani, V., Mohammadzadeh, A., Shadnia, S., 2019. The role of emotion dysregulation in the relation of childhood trauma to heroin craving in individuals with heroin dependence. Drug Alcohol Depend. 195, 132–139. https://doi.org/10.1016/j.drugalcdep.2018.12.008.

Gilmore, A.K., Lopez, C., Muzzy, W., Brown, W.J., Grubaugh, A., Oesterle, D.W., Acierno, R., 2020. Emotion dysregulation predicts dropout from prolonged exposure treatment among women veterans with military sexual trauma-related posttraumatic stress disorder. Womens Health Issues 30 (6), 462–469. https://doi.org/10.1016/j.whi.2020.07.004.

Gross, J.J., 1998. The emerging field of emotion regulation: an integrative review. Rev. Gen. Psychol. 2 (3), 271–299.

Hembree, E.A., Rauch, S.A.M., Foa, E.B., 2003. Beyond the manual: the insider's guide to prolonged exposure therapy for PTSD. Cogn. Behav. Pract. 10 (1), 22–30. https:// doi.org/10.1016/S1077-7229(03)80005-6.

Hinrichs, R., Michopoulos, V., Winters, S., Rothbaum, A.O., Rothbaum, B.O., Ressler, K. J., Jovanovic, T., 2017. Mobile assessment of heightened skin conductance in posttraumatic stress disorder: H inrichs et al. Depression and Anxiety 34 (6), 502–507. https://doi.org/10.1002/da.22610.

Hinrichs, R., van Rooij, S.J.H., Michopoulos, V., Schultebraucks, K., Winters, S., Maples- Keller, J., Rothbaum, A.O., Stevens, J.S., Galatzer-Levy, I., Rothbaum, B.O., Ressler, K.J., Jovanovic, T., 2019. Increased skin conductance response in the immediate aftermath of trauma predicts PTSD risk. Chronic Stress 3, 247054701984444. https://doi.org/10.1177/2470547019844441.

Holmbeck, G.N., 2002. Post-hoc probing of significant moderational and mediational effects in studies of pediatric populations. J. Pediatr. Psychol. 27 (1), 87–96.

Hopper, J.W., Frewen, P.A., van der Kolk, B.A., Lanius, R.A., 2007. Neural correlates of reexperiencing, avoidance, and dissociation in PTSD: symptom dimensions and emotion dysregulation in responses to script-driven trauma imagery. J. Trauma. Stress. 20 (5), 713–725. https://doi.org/10.1002/jts.20284.

Janiri, D., Moccia, L., Dattoli, L., Pepe, M., Molinaro, M., De Martin, V., Chieffo, D., Di Nicola, M., Fiorillo, A., Janiri, L., Sani, G., 2021. Emotional dysregulation mediates the impact of childhood trauma on psychological distress: First Italian data during the early phase of COVID-19 outbreak. Australian & New Zealand Journal of Psychiatry, 000486742199880. https://doi.org/10.1177/0004867421998802.

Jenness, J.L., Jager-Hyman, S., Heleniak, C., Beck, A.T., Sheridan, M.A., McLaughlin, K. A., 2016. Catastrophizing, rumination, and reappraisal prospectively predict adolescent PTSD symptom onset following a terrorist attack: Jenness et al. Depression and Anxiety 33 (11), 1039–1047. https://doi.org/10.1002/da.22548.

Jerud, A.B., Pruitt, L.D., Zoellner, L.A., Feeny, N.C., 2016. The effects of prolonged exposure and sertraline on emotion regulation in individuals with posttraumatic stress disorder. Behav. Res. Ther. 77, 62–67. https://doi.org/10.1016/j.brat.2015.12.002.

Kaufman, E.A., Graves, J.L., Wallace, M.L., Lazarus, S.A., Stepp, S.D., Pedersen, S.L., 2021. Associations between physiological and self-reported indices of emotion dysregulation across varying levels of alcohol use among individuals with and without borderline personality disorder. Biol. Psychol. 160, 108044 https://doi.org/ 10.1016/j.biopsycho.2021.108044.

van der Kolk, B.A., 1997. The psychobiology of posttraumatic stress disorder. J. Clin. Psychiatry 58 (Suppl. 9), 16–24.

Kreutzmann, J.C., Marin, M.-F., Fendt, M., Milad, M.R., Ressler, K., Jovanovic, T., 2021. Unconditioned response to an aversive stimulus as predictor of response to conditioned fear and safety: a cross-species study. Behav. Brain Res. 402, 113105 https://doi.org/10.1016/j.bbr.2020.113105.

Lanius, R.A., Vermetten, E., Loewenstein, R.J., Brand, B., Schmahl, C., Bremner, J.D., Spiegel, D., 2010. Emotion modulation in PTSD: clinical and neurobiological subtype. Am. J. Psychiatr. 167 (6), 640–647. https://doi./appi.ajp.2009.09081168.