Neural Correlates and Predictive Power of Trait Resilience in an Acutely Traumatized Sample: A Pilot Investigation

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ABSTRACT

Objective: Resilience refers to the ability to thrive despite adversity and is defined as a multidimensional phenomenon, spanning internal locus of control, sense of meaning, social problem-solving skills, and self-esteem. We aimed to investigate the predictive value of resilience for the development of posttraumatic stress disorder (PTSD) and to examine the neural correlates mediating the relationship between resilience and recovery from a traumatic event in acutely traumatized subjects. We hypothesized that resilience would mediate the relationship between childhood trauma and posttraumatic recovery.

Method: We conducted a prospective study with 70 acutely traumatized subjects with *DSM-IV* PTSD recruited at the emergency department, assessing PTSD symptom severity at 3 time points within the first 3 months posttrauma. Scores for childhood trauma as assessed with the Childhood Trauma Questionnaire and trait resilience as assessed with the Connor-Davidson Resilience Scale were used as predictors of symptom severity. A subsample of 12 subjects additionally underwent a functional 4 Tesla magnetic resonance imaging scan 2 to 4 months posttrauma. We employed the traumatic script-driven imagery paradigm to assess the correlations between trait resilience and blood oxygen level–dependent (BOLD) response. The study was conducted from 2003 to 2007.

Results: Resilience predicted PTSD symptom severity at 5 to 6 weeks ($\beta = -0.326$, $P = .01$) as well as at 3 months ($\beta = -0.423$, $P = .003$) posttrauma better than childhood trauma. Resilience essentially mediated the relationship between childhood trauma and posttraumatic adjustment. Resilience scores were positively correlated with BOLD signal strength in the right thalamus as well as the inferior and middle frontal gyri (Brodmann area 47).

Conclusions: This pilot investigation revealed a significant relationship between resilience and emotion regulation areas during trauma recall in an acutely traumatized sample. Resilience was established as a significant predictor of PTSD symptom severity and mediated the influence of childhood trauma on posttraumatic adjustment.

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Resilience refers to the ability to thrive despite adversity and has been defined as a multidimensional phenomenon, spanning internal locus of control, sense of meaning, social problem-solving skills, and self-esteem.[1,2] The concept of hardiness integrates many of these characteristics and describes individuals who are committed to finding meaningful purpose in life, believe that one can influence one's surroundings and the outcome of events, and trust that one can learn and grow from both positive and negative life experiences.[3] High levels of this personality trait have been shown to enhance posttraumatic adjustment,[4–6] possibly by means of a faster physiological recovery from stress.[7] However, there is no widely accepted definition of resilience. Instead, different researchers have focused on specific protective attributes like specific personality traits, supportive social relationships, emotion regulation, and coping skills (for comprehensive reviews, see Layne et al[8] and Reich et al[9]). The Connor-Davidson Resilience Scale (CD-RISC) has been put forward as a new instrument that combines both hardiness as well as what the authors conceptualize to be the underpinnings of resilience (ie, secure and stable attachment and prior experiences with successful coping).[10] Resilience as assessed with this instrument has been shown to be significantly correlated with 3 factors from the 5-factor model of personality—neuroticism, extraversion, and conscientiousness.[11]

Childhood Trauma and Resilience

While some individuals show no signs of psychopathology, even after exposure to severe childhood trauma,[12–14] 2 meta-analyses identified prior traumatization as a major risk factor for the development of posttraumatic stress disorder (PTSD) after a traumatic event during adulthood[15,16] and childhood abuse as a risk factor that was uniformly identified across different study types.[16] In the National Comorbidity Survey, physical and sexual abuse were uniquely related to PTSD after controlling for demographic variables, depression, and other anxiety disorders.[17] Childhood trauma has been associated with lower self-esteem, demoralization, loss of a sense of meaning, and affect dysregulation[18–21]—factors thought to be directly linked to resilience. In 2 other investigations,[11,22] measures of trait resilience moderated the relationship between childhood trauma and current psychiatric symptoms in university students (also see Luthar[23]).

In the acute aftermath of a traumatic event, it is imperative to identify risk factors for pathogenesis early in order to determine who should be offered close monitoring or early interventions. As childhood abuse has been associated with lower levels of trait resilience, and lower level of trait resilience in turn has been linked to higher PTSD rates in trauma-exposed samples,[4–6] it seems plausible that a standardized assessment of resilience might predict posttraumatic recovery better than an account of childhood abuse. However, to date no empirical evidence exists for the predictive value of trait resilience on PSTD development in acutely traumatized samples.

Neural Correlates of Resilience

The underlying neurocognitive characteristics mediating the relationship between trait resilience and recovery from a traumatic event have

Clinical Points

- Resilience can help predict recovery from an acute traumatic event.
- Resilience mediates the relationship between childhood trauma and posttraumatic recovery.
- Resilience is associated with activation of emotion regulation areas in the brain.

also not yet been identified. Charney[24] proposed a complex model of allostasis involving various brain structures. While many studies have included healthy, trauma-exposed control groups, to our knowledge only 1 functional magnetic resonance imaging (fMRI) study has directly analyzed the relationship between trait resilience scores and blood oxygen level–dependent (BOLD) signal strength. Waugh et al[25] compared brain activation of low- and high-trait resilient individuals during recovery from threat. Aversive and neutral stimuli were preceded by a threat cue with equal probabilities. During stimulus exposure following the threat cue, individuals scoring low on the resilience measure exhibited an extended insula activation independent of stimulus valence, while highly resilient subjects showed sustained insula activation only during exposure to aversive stimuli. These results indicate that resilience is associated with flexible and timely down-regulation of arousal and are thus consistent with the allostasis model suggested by Charney.[24]

Hypotheses

In order to test the predictive value of resilience for PTSD development and investigate the neural correlates mediating the relationship between resilience and recovery from a traumatic event in acutely traumatized subjects, we conducted a prospective questionnaire study in conjunction with an fMRI investigation.

We hypothesized that resilience would mediate the relationship between childhood trauma and posttraumatic recovery. We applied the well-established traumatic script-driven imagery paradigm to investigate neural activations during trauma recall.

METHOD

Subjects

Assessments were carried out within the scope of a larger prospective study in an acutely traumatized sample. This pilot investigation included 70 subjects (mean ± SD age = 36.24 ± 12.60 years; women, n = 41) who presented to the emergency department at the London Health Sciences Center in London, Ontario, or to the Department of Emergency Medicine at the University of Alberta in Edmonton after they had been involved in a motor vehicle accident (n = 53), workplace accident (n = 8), or physical assault (n = 5) or another traumatic event (n = 4). All subjects met *DSM-IV* criterion A for PTSD. Exclusion criteria were (1) significant head injury, (2) history of neurologic disorders, (3) current Axis I disorders at the time of the accident, (4) lifetime history of bipolar disorder or schizophrenia, and (5) psychotropic or steroid medication (for the fMRI sample, described below). The study was approved by the research ethics board at the London Health Sciences Centre and University of Alberta; informed written consent was obtained from all patients. At the time of the accident, 32.9% of the participants were single, 41.1% were married, 15.7% lived in common-law marriage, and 8.6% were separated or divorced. Concerning alcohol use, 21.4% of the participants stated that they never use alcohol, 72.9% reported that they occasionally use alcohol, and 5.7% described that they consume alcoholic beverages on a daily basis. Subjects were not enrolled in psychotherapeutic interventions during study participation. The study was conducted from 2003 to 2007.

In addition, a convenience subsample of 12 subjects underwent a 4 Tesla fMRI scan at 2 to 4 months posttrauma.

Measures

The Acute Stress Disorder Scale[26] (α = .951) is a 19-item questionnaire that measures the severity of acute stress disorder symptoms on a 5-point Likert scale (1 = "not at all," 5 = "very much") and was assessed 1 to 2 weeks posttrauma. We applied a cutoff score of 56 to establish diagnostic status. The Clinician-Administered PTSD Scale (CAPS)[27] (α = .950) is a semistructured clinical interview that assesses posttraumatic symptoms as defined in the current *DSM-IV*. It was assessed both at 5 to 6 weeks and at 3 months posttrauma. The CAPS uses standardized questions to identify both symptom frequency and intensity. Severity scores were calculated as the sum of frequency and intensity ratings across all *DSM-IV* symptoms of PTSD, and diagnostic status was coded as CAPS score < 50 indicating subclinical and CAPS score > 50 indicating clinical symptomatology. The CD-RISC (α = .938) is a 25-item scale that measures the ability to cope with adversity with a 5-point Likert scale (0 = "not true at all" to 4 = "true nearly all the time"), which was assessed either 1 to 2 weeks (n = 40, 57.1%) or 5 to 6 weeks (n = 30, 42.9%) posttrauma. The Childhood Trauma Questionnaire Short Form[28] (α = .949) consists of 25 clinical items assessing emotional abuse, emotional neglect, physical abuse, physical neglect, and sexual abuse during childhood on a 5-point Likert scale (1 = "never true" to 5 = "very often true"). In addition, the scale contains 3 validity items assessing denial and minimization. These items were excluded from all further analyses.

Statistics

Group statistics were calculated as Pearson correlations and 2-sample *t* tests. Linear regression analyses were employed to analyze predictive power and test for mediation. In all statistics, we considered *P* values < .05 to be significant. Statistical analyses were carried out using Statistical Package for Social Sciences (SPSS Inc, Chicago, Illinois), Version 15.0.

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Table 1. Descriptive Statistics

	Total Sample		fMRI Subsample		
Scale	n	Mean ± SD	n	Mean ± SD	*P*
ASD score	55	47.73 ± 19.28	10	46.60 ± 22.04	.840
CAPS score at 5–6 weeks	64	28.56 ± 24.92	12	23.25 ± 22.55	.417
CAPS score at 3 months	44	18.52 ± 20.31	9	17.11 ± 21.21	.818
CD-RISC score	70	68.91 ± 15.34	12	68.00 ± 14.47	.823
CTQ score	70	42.03 ± 17.47	12	50.42 ± 28.16	.248

Abbreviations: ASD = Acute Stress Disorder Scale, CAPS = Clinician-Administered PTSD Scale, CD-RISC = Connor-Davidson Resilience Scale, CTQ = Childhood Trauma Questionnaire, fMRI = functional magnetic resonance imaging.

Table 2. Zero-Order Correlations

	2	3	4	CAPS at 3 Months
1 CD-RISC	−0.243*	−0.307*	−0.376**	−0.485**
2 CTQ		0.187	0.285*	0.354*
3 ASD			0.308**	0.748**
4 CAPS at 5–6 weeks				0.451**

*P = .05. **P = .001.

Abbreviations: ASD = Acute Stress Disorder Scale, CAPS = Clinician-Administered PTSD Scale, CD-RISC = Connor-Davidson Resilience Scale, CTQ = Childhood Trauma Questionnaire.

Functional Imaging Paradigm

We employed a well-established script-driven symptom provocation paradigm (neutral and trauma scripts) adapted for fMRI according to previously published methods.[29] Magnetic resonance scans were performed on a 4 Tesla whole-body Varian/Siemens imaging system with a 90-cm diameter horizontal bore and a whole-body 68-cm diameter gradient set with a maximum strength of 40 mT/m and a slew rate of 120 mT/m per second. Each functional brain volume was acquired by using a navigator echo-corrected, interleaved multishot (4 shots), echo-planar imaging pulse sequence with a 128 × 128 matrix size and a total volume acquisition time of 5 seconds (echo time = 15 milliseconds, flip angle = 45°, field of view = 24.0 cm). The volume acquired covered the whole brain and consisted of 12 transverse slices, 6 mm thick (voxel size = 1.87 × 1.87 × 6 mm).

Image processing was performed with SPM 2 (Wellcome Department of Imaging Neuroscience, London, England). Volumes were realigned to the first volume of the series and parameters for normalization were determined from the mean functional image. Realigned functional images were spatially normalized to an echo-planar imaging template and spatially smoothed with an 8-mm full width at half maximum isotropic Gaussian kernel.

Statistical Mapping

Images were analyzed using a 2-stage random effects analysis. At the first level, each subject's functional data were analyzed separately by modeling the evoked BOLD responses for each task epoch of interest as basis functions (ie, a boxcar function convolved with a hemodynamic response function). For each subject a contrast of trauma script greater than neutral script was created. These contrasts were entered into a second-level analysis and correlations between subjects' CD-RISC score and the BOLD response were examined. We conducted a region-of-interest analysis thresholded at cluster size = 10 and P = .001, with small volume correction in 10-mm spheres. We covaried out time since the traumatic event to account for variability in the time point of the scan.

Consistent with the allostasis model,[24] we hypothesized that resilience would be negatively related to activation in brain regions previously implicated in emotional arousal and known to show increased responsiveness in PTSD. We therefore hypothesized that resilience would be negatively related to activation in the amygdala and insula. In addition, we expected resilience to be positively correlated with BOLD signal strength in brain regions recruited during emotion regulation that have been shown to be hypoactivated in PTSD (for a meta-analysis, see Etkin and Wagner[30]), ie, the ventromedial and ventrolateral prefrontal cortex and the thalamus.

RESULTS

Descriptive Statistics and Zero-Order Correlations

Descriptive statistics for all scales are listed in Table 1. Nineteen of 55 subjects (34.5%) assessed with the Acute Stress Disorder Scale 1 to 2 weeks posttrauma met the diagnostic cutoff for acute stress disorder. At 5 to 6 weeks posttrauma, 12 of 64 subjects (18.7%) who underwent the CAPS interview were diagnosed with PTSD. At 3 months posttrauma, 44 subjects were available for interviewing with the CAPS, 5 (11.4%) of which met diagnostic criteria for PTSD. The subgroup undergoing fMRI scanning did not differ significantly from the remaining sample in terms of psychopathology (see Table 1).

The amount of time elapsed between the traumatic event and the assessment of the resilience scale did not significantly influence CD-RISC scores (M_1 = 70.8, M_2 = 66.4, t = 1.191, P = .238). Connor-Davidson Resilience Scale scores were significantly related to PTSD symptom severity at 1 to 2 weeks (r = –0.307), 5 to 6 weeks (r = –0.376), and 3 months (r = –0.485) posttrauma (Table 2). These correlations indicate that higher resilience was related to less severe PTSD symptomatology throughout the first 3 months after the traumatic event. Resilience was also significantly correlated with exposure to childhood trauma (r = –0.243, P = .043), indicating that childhood trauma is associated with lower level of trait resilience. In turn, childhood trauma was significantly correlated with PTSD symptom severity at 5 to 6 weeks (r = 0.285, P = .022) and at 3 months (r = 0.354, P = .018) posttrauma, but not at 1 to 2 weeks posttrauma (r = 0.187, P = .172).

Regression Analysis

The Childhood Trauma Questionnaire Short Form yielded a skewed distribution that significantly deviated from a normal distribution (Kolmogorov-Smirnov Z test = 1.784, P = .003). We therefore computed 5 dummy variables. Subjects reporting no childhood maltreatment at all were grouped together. In order to ensure comparable cell sizes, the remaining subjects were divided into quartiles. Following unsuccessful efforts at redressing skewness

Table 3. Linear Regression Analyses

Regressand	*F*	Correlation, R^2	Predictor Variables	Nonstandardized Coefficient β	Standardized Coefficient β	*t*	*P*
CAPS scores at 5 to 6 weeks (n = 64)							
Model 1	5.963*	0.073	CTQ dummy[a]	17.290	0.296	2.442	.017
Model 2	10.222*	0.128	CD-RISC	−0.607	−0.376	−3.197	.002
Model 3[b]	7.482**	0.171	CTQ dummy[a]	**13.950**	**0.239**	**2.052**	**.044**
			CD-RISC	**−0.525**	**−0.326**	**−2.666**	**.010**
CAPS scores at 3 months (n = 44)							
Model 1	6.199*	0.108	CTQ dummy[a]	17.182	0.359	2.490	.017
Model 2	12.934**	0.217	CD-RISC	−0.695	−0.485	−3.596	.001
Model 3[b]	8.677**	0.263	CTQ dummy[a]	12.287	0.256	1.901	.064
			CD-RISC	**−0.606**	**−0.423**	**−3.138**	**.003**

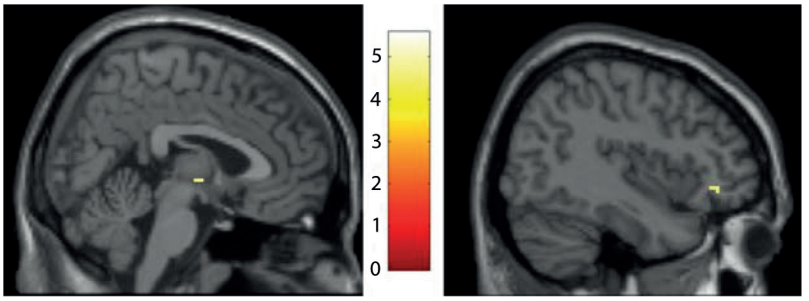
[a]Dummy variable coding inclusion in the CTQ quartile reporting the most severe maltreatment.
[b]Boldface numbers indicate significant results in the final regression model.
*P = .05. **P = .001.
Abbreviations: CAPS = Clinician-Administered PTSD Scale, CD-RISC = Connor-Davidson Resilience Scale, CTQ = Childhood Trauma Questionnaire.

Table 4. Positive Correlations Between CD-RISC Scores and Blood Oxygen Level–Dependent Signal Intensity After Covariation of Time Since Trauma (n = 12)

MNI Coordinates	*z* Score	Cluster Size	Brain Region
2, −8, −2	3.57	17	Thalamus
40, 38, −8	3.22	10	Middle frontal gyrus, BA 47
44, 32, −4	3.14		Inferior frontal gyrus, BA 47

Abbreviations: BA = Brodmann area, BOLD = blood oxygen level–dependent, CD-RISC = Connor-Davidson Resilience Scale, MNI = Montreal Neurological Institute.

Figure 1. Correlation Between Connor-Davidson Resilience Scale Score and Blood Oxygen Level–Dependent Signal Strength in the Thalamus and the Right Ventrolateral Prefrontal Cortex



through other means, including data transformations, this approach was deemed optimal. All 5 groups were then entered as predictors in a stepwise linear regression analyses as dichotomous dummy variables. Of all 5 Childhood Trauma Questionnaire Short Form dummy variables, only the variable coding inclusion in the quartile reporting the most severe maltreatment was found to be a significant predictor for CAPS at 5 to 6 weeks (β = 0.296, t = 2.442, P = .017) and 3 months (β = 0.359, t = 2.490, P = .017) posttrauma (Table 3). Connor-Davidson Resilience Scale scores significantly predicted CAPS at 5 to 6 weeks (β = −0.376, t = −3.197, P = .002) and 3 months (β = −0.485, t = −3.596, P = .001) posttrauma. In a stepwise regression with Childhood Trauma Questionnaire Short Form dummy variables as block 1 and CD-RISC scores as block 2, CD-RISC predicted CAPS over and above the Childhood Trauma Questionnaire Short Form dummy variable coding inclusion in the quartile reporting the most severe maltreatment at both time points (Table 3).

fMRI Results

During script-driven imagery of the traumatic versus the neutral event, CD-RISC scores correlated positively with BOLD signal intensity in the right thalamus and the right inferior and middle frontal gyri (Brodmann area 47) after covariation of time since trauma (Table 4 and Figure 1). No negative correlations could be established.

DISCUSSION

To our knowledge, this is the first study investigating the predictive power of resilience as defined by the CD-RISC as well the relationship between resilience and brain activation during trauma recall in an acutely traumatized sample. The prospective analyses presented here suggest that trait resilience mediates the relationship between childhood trauma and posttraumatic adjustment. Resilience predicted PTSD symptom severity at 5 to 6 weeks as well as 3 months posttrauma better than childhood trauma. The neuroimaging results suggest that resilience is associated with greater activation of the right thalamus as well as the inferior and middle frontal gyri—regions previously implicated in emotion regulation.

Questionnaire Study

As expected, trait resilience was negatively correlated with symptom severity at all 3 time points, with correlation coefficients being higher for later assessments. As many individuals develop subclinical symptom levels initially, yet recover within the first few weeks after a traumatic event,[31] trait resilience is more closely correlated with later symptom assessments. Resilience was also negatively correlated with childhood trauma, which is consistent with previous reports.[11,22,32] Childhood trauma in turn was significantly correlated with symptom

severity at 5 to 6 weeks and 3 months posttrauma; however, it was not significantly correlated with the initial symptoms assessed at 1 to 2 weeks posttrauma.

Resilience scores significantly predicted PTSD symptom severity at 5 to 6 weeks and at 3 months posttrauma. These results indicate that the inclusion of resilience measures could inform preventative treatments for PTSD in the acute aftermath of a traumatic event and might also prove useful in the study of general recovery processes following stressful life events.

When childhood trauma was entered into a stepwise regression analysis in the form of 5 dummy variables coding the extent of the maltreatment, the most severe forms of maltreatment were identified as a significant predictor of CAPS scores at 5 to 6 weeks and at 3 months posttrauma. Thus, both variables, resilience and childhood trauma, were established as significant predictors of symptom severity when entered separately. When entered into a stepwise regression analysis in consecutive blocks, resilience predicted PTSD symptom severity over and above severe childhood maltreatment. The results of this pilot investigation thus indicate that both variables are valuable predictors of future symptom development and therefore can guide the identification of individuals at risk. However, trait resilience is a more proximal variable and carried greater predictive power in the current sample.

fMRI Study

The script-driven neuroimaging investigation identified the right thalamus as well as a ventrolateral prefrontal region spanning the right inferior and middle frontal gyri as neural correlates of trait resilience. Both areas have previously been implicated in emotion regulation[33–36] and are known to show differential activations in PTSD.[37–39]

Phan et al[36] showed that the right inferior frontal gyrus is implicated in suppression of emotional arousal in healthy participants as activation differences in this area between suppression and maintenance of an emotion were significantly correlated to the concurrent amygdala activation. Hopper et al[38] showed that activation of the inferior frontal gyrus was associated with less reexperiencing and less state dissociation during traumatic script-driven imagery. Convergently, trauma-exposed individuals who did not develop any psychopathology were found to activate this area significantly more during traumatic script-driven imagery.[37]

Equally, the thalamus region showed greater activation in healthy, trauma-exposed controls than in PTSD subjects during script-driven imagery in the studies by Lanius et al[29] and Shin et al.[39] The thalamus is thought to be connected with the prefrontal cortex by complex thalamo-cortico-thalamic circuits and has been proposed to be a key regulatory region for arousal modulation. In a study by Herwig et al,[33] the expectation of unpleasant stimuli alone lead to activations in this region, indicating a cognitive emotion regulation effort. According to a recent model,[35] emotion regulation through reappraisal is facilitated by connections between the right ventrolateral prefrontal cortex and the basal ganglia modulating emotional arousal. In conjunction, the areas identified here as neural correlates of resilience therefore point toward emotion regulation as the mediator between trait resilience and posttraumatic adjustment.

Limitations

There are several limitations to this study. First, the mean CD-RISC score for this sample (68.91 [SD = 15.34]) is considerably lower[10] than the normative score for the general population (80.4 [SD = 12.8]) and more consistent with normative scores for psychiatric outpatients (68.0 [SD = 15.3]) and primary care patients (71.8 [SD = 18.4]). This might be due to the screening criteria employed in the recruitment for this study. As all participants were required to meet criterion A of the *DSM-IV* PTSD diagnosis, they were characterized by reactions to the trauma that are deemed pathogenic. As such, the investigated sample is characterized by a higher risk for the development of psychiatric disorders and might therefore exhibit the lower CD-RISC scores. This hypothetical explanation should be tested in future studies. Future studies should also seek to examine the relationship between past psychiatric illness and current levels of resilience. Second, this pilot investigation relies on comparatively small sample sizes. This is particularly true for the assessment of symptom severity at 3 months posttrauma and the neuroimaging study. The small sample size might have prevented us from detecting additional brain regions associated with trait resilience due to limited power. Third, no repeated assessments of resilience are available, a limitation that precludes us from assessing the test-retest reliability over the course of posttraumatic adjustment. Average resilience levels in the subgroup assessed 5 to 6 weeks posttrauma were slightly lower than in the subgroup assessed 1 to 2 weeks posttrauma, but this difference failed to reach significance and time of assessment could not be established as a significant predictor in the regression analyses. As resilience was assessed after the traumatic event, it is also impossible to determine whether the traumatic event itself or the early symptoms it brought on might have influenced the resilience measurement. The concept of resilience is still in its early stages and possibly hasn't been fully refined yet. For example, it has been suggested that the context in which resilience appears might need to be taken into account.[8] In this view, resilience would be conceptualized as a domain-specific variable, ie, a subject could show resilience in the realm of parenting, but not in a work context. Fourth, trials suggest that psychopharmacologic treatment can elevate resilience levels in PTSD,[40,41] indicating that, after a traumatic event, resilience might not be a stable trait but might be influenced by symptom severity. Therefore, longitudinal studies assessing resilience repeatedly as a process variable pretrauma and posttrauma as well as a predictor of recovery from acute stress disorder are needed to reliably quantify its impact prospectively (for a review, see Connor[42]).

CONCLUSION

This pilot investigation revealed a significant relationship between trait resiliency and emotion regulation areas during

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trauma recall in an acutely traumatized sample. Resilience was established as a significant predictor of future PTSD symptom severity and fully mediated the influence of childhood trauma on posttraumatic adjustment statistically.

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