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Emergency Department Predictors of Posttraumatic Stress Reduction for Trauma-Exposed Individuals With and Without an Early Intervention

Matthew Price,

Department of Psychology, University of Vermont

Megan Kearns,

Centers for Disease Control and Prevention, and Department of Psychiatry and Behavioral Sciences, Emory University School of Medicine

Debra Houry, and

Department of Emergency Medicine, Emory University School of Medicine

Barbara O. Rothbaum

Department of Psychiatry and Behavioral Sciences, Emory University School of Medicine

Abstract

Objective—Recent data have supported the use of an early exposure intervention to promote a reduction in acute stress and posttraumatic stress disorder (PTSD) symptoms after trauma exposure. The present study explored a comprehensive predictive model that included history of trauma exposure, dissociation at the time of the trauma and early intervention, and physiological responses (cortisol and heart rate) to determine which variables were most indicative of reduced PTSD symptoms for an early intervention or treatment as usual.

Method—Participants ($n = 137$) were randomly assigned to the early intervention condition ($n = 68$) or assessment-only condition ($n = 69$) while receiving care at the emergency department of a Level 1 trauma center. Follow-up assessments occurred at 4 and 12 weeks posttrauma.

Results—Findings suggested that dissociation at the time of the 1st treatment session was associated with reduced response to the early intervention. No other predictors were associated with treatment response. For treatment as usual, cortisol levels at the time of acute care and dissociation at the time of the traumatic event were positively associated with PTSD symptoms.

Conclusions—Dissociation at the time at which treatment starts may indicate poorer response to early intervention for PTSD. Similarly, dissociation at the time of the event was positively related to PTSD symptoms in those who received treatment as usual.

Recent work has suggested exposure therapy administered shortly after a trauma can reduce subsequent posttraumatic stress disorder (PTSD) symptoms (Rothbaum et al., 2012). The

variability in outcomes, however, highlights a need to identify predictors of response in order to target treatment to those at greatest risk and those most likely to benefit (Kearns, Ressler, Zatzick, & Rothbaum, 2012). The present study evaluated several variables theorized to predict response to an early intervention due to their enhancement of fear acquisition after a trauma (Craske et al., 2008). These variables include childhood trauma exposure, heart rate, cortisol levels, and dissociation.

Childhood trauma exposure is hypothesized to influence response to an adulthood trauma. Childhood exposure may establish fear pathways that are strengthened after exposure to subsequent stressful events (Clemmons, Walsh, DiLillo, & Messman-Moore, 2007). Indeed, childhood traumas are associated with more severe PTSD symptoms after an adulthood trauma (Nishith, Mechanic, & Resick, 2000). Those with such histories may be less likely to benefit from early intervention after an adulthood trauma as a result. Prior work is inconclusive on the influence of childhood trauma on treatment outcome in chronic PTSD samples, however. Hembree, Street, Riggs, and Foa (2004) reported childhood trauma exposure was associated with reduced PTSD symptoms for prolonged exposure (PE), whereas Resick, Nishith, and Griffin (2003) did not find this association using PE or cognitive processing therapy (CPT).

The initial physiological response to trauma may also enhance fear acquisition. An elevated heart rate (HR) 1 week post-trauma was indicative of increased PTSD symptoms 4 months (Shalev et al., 1998), 6 months (Bryant, Harvey, Guthrie, & Moulds, 2000), and 2 years later (Bryant & Harvey, 2002). Similarly, posttrauma cortisol has been negatively related to increased PTSD symptoms (for a review see DiGangi et al., 2013). Three studies suggested baseline cortisol was not predictive of symptom reduction during treatment (Gerardi, Rothbaum, Astin, & Kelley, 2010; Olff, de Vries, Güzelcan, Assies, & Gersons, 2007; Yehuda et al., 2009). However, none of these studies examined the relation of physiological responses with an early intervention.

Last, increased peritraumatic dissociation, which involves a disruption of consciousness, memory, body awareness, and perception of the environment in response to intense emotional distress, is associated with increased PTSD symptoms (Lanius et al., 2010). Recent work has posited that dissociation attenuates treatment response (Lanius, Brand, Vermetten, Frewen, & Spiegel, 2012). However, empirical work has not supported this hypothesis in PE (Hagenaars, van Minnen, & Hoogduin, 2010), CPT (Resick, Suvak, Johnides, Mitchell, & Iverson, 2012), or social skills with narrative storytelling therapy (Cloitre, Petkova, Wang, & Lu Lassell, 2012). A potential explanation for the discrepancy between the theorized and empirical findings is the assessment of dissociation. The above studies assessed dissociation over the past week or month, whereas dissociation at the time of treatment is posited to reduce response (Lanius et al., 2012).

No studies have examined these factors as predictors of response to an early intervention for PTSD. Four predictors were hypothesized to be associated with increased PTSD symptoms 4 and 12 weeks posttrauma among those who received an early intervention in the emergency department and those who received treatment as usual: (1) childhood trauma

exposure, (2) increased HR, (3) reduced cortisol, and (4) increased peritraumatic dissociation and dissociation at the start of treatment.

Method

Participants

Participants were 137 individuals who presented at the emergency department (ED) after experiencing a Criterion A trauma according to the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; *DSM-IV*; American Psychiatric Association, 1994). Participants had a mean age of 31.47 years ($SD = 11.65$) and were mostly African American (78%) and female (65%), with 77% making less than \$40,000 annually.

Measures

Heart rate—Nurses obtained HR during the initial physical exam.

Salivary cortisol—Cortisol was obtained from a sample of 25–50 μ l of saliva and expressed using the high sensitivity salivary cortisol enzyme immunoassay (Salimetrics, State College, Pennsylvania).

Standardized Trauma Interview (STI; Foa & Rothbaum, 2001)—The STI is a 41-item interview on relevant aspects of trauma (interrater agreement = 0.99). The STI was used to determine if the trauma met Criterion A for a diagnosis of PTSD.

Childhood Trauma Questionnaire (CTQ; Bernstein, Fink, Handelsman, & Foote, 1994)—The CTQ is a 25-item self-report scale that assesses negative childhood experiences and has excellent psychometric properties. CTQ total scores (range = 25–125) offer a valid indicator of early trauma exposure (Scher, Stein, Asmundson, McCreary, & Forde, 2001). Internal consistency for the current study was excellent (0.93).

Immediate Stress Reaction Checklist (ISRC; Fein, Kassam-Adams, Vu, & Datner, 2001)—The peritraumatic and postevent dissociation subscales of the ISRC were used to assess reactions at the time of the trauma (range = 0–36) and at the time of the assessment (range = 0–9). Internal consistency for the current study was good (0.86).

PTSD Symptom Scale, Interview Version (PSS-I; Foa, Riggs, Dancu, & Rothbaum, 1993)—The PSS-I is a 17-item interview that corresponds to the *DSM-IV* PTSD symptoms. Symptoms were rated on a 0–3 scale, with total scores ranging from 0 to 51 (interrater agreement = 0.99).

Procedure

Assessors with a minimum of a master's degree were positioned in the ED of the largest Level One trauma center in Georgia from 7 a.m. to 7 p.m. daily. Patients who spoke English, did not have a loss of consciousness, and were alert and oriented were included in the study. After completing baseline measures (CTQ, ISRC sub-scales, HR, and Cortisol), patients were randomly assigned to receive a modified prolonged exposure intervention (INT) in the

ED ($n = 68$) or treatment as usual (TAU; $n = 69$), which involved no additional intervention. Participants were assessed for PTSD symptoms 4 and 12 weeks posttrauma. The majority of participants completed the 4-week (TAU: 81%; INT: 66%) and 12-week (TAU: 72%; INT: 59%) evaluations. The hospital and university institutional review board approved this investigation.

Treatment—Treatment included three 60-min sessions across 3 weeks (1 per week). The first session began in the ED after the patient was medically stable. The remaining sessions occurred in the ED in a private space. The protocol was a modified version of PE (Foa, Hembree, & Rothbaum, 2007), and treatment was provided by master's- or doctoral-level clinicians. The first session included a presentation of the treatment rationale, 30–45 min of imaginal exposure followed by processing, identification of in vivo exposures to be completed as homework, and deep breathing skills. The session concluded with a review of normal trauma reactions and the assignment of three tasks for homework: (1) listening to an audiotaped recording of in-session imaginal exposure exercise, (2) in vivo exposures to reduce fear, and (3) scheduling of self-care tasks such as engaging in social activities. The second and third sessions began with a review of the homework assigned during the previous sessions. These sessions included additional imaginal exposure with processing, the continued assignment of imaginal and in vivo exposure, and self-care tasks for homework. Patients were encouraged to continue in vivo exposure and self-care tasks after the third session.

Data Analytic Plan

Separate mixed effect models that included fixed effects for time, baseline cortisol, HR, peritraumatic dissociation, postevent dissociation, sexual assault, and child trauma history were used (Hox, 2010). PTSD symptoms served as the outcome. Time corresponded to change from Week 4 to Week 12 and was coded as -1 and 0 , respectively. Continuous predictors were group-mean-centered. A random intercept was included but not a random slope. Missing data were handled with full information maximum-likelihood estimation. An intent-to-treat sample was used in that all participants who were enrolled in the study were included in the analyses and a Bonferroni correction of $\alpha = .025$ was used. Separate receiver operating characteristic (ROC) curves for the assessment and intervention conditions were used to identify cutoff points on significant predictors of Week 12 PTSD. PTSD status was derived from the PSS-I such that a diagnosis corresponded to a response of 2 or greater on one reexperiencing item, three avoidance items, and two hyperarousal items.

Results

Descriptive information and a correlation matrix are presented in Table 1 and Table 2, respectively. Overall dropout rate was higher for INT ($n = 27$, 59%) than for TAU ($n = 19$, 41%), but the difference was not significant, $\chi^2(1) = 1.92$, $p = .167$. Dropouts did not differ on demographic variables of age, ethnicity, gender, or income ($ps = .07$ – $.79$). None of the predictor variables were associated with slope for both conditions. Removal of these variables did not significantly alter fit according to the change in the deviance statistic for the intervention, $\chi^2(6) = 2.37$, $p = .887$, or the assessment, $\chi^2(6) = 2.78$, $p = .835$, conditions.

For INT, PTSD significantly changed from Week 4 to Week 12 ($\gamma_{10} = -3.58, p = .015$). Dissociation at the start of treatment was positively associated with PTSD at Week 12 ($\gamma_{02} = 5.08, p < .001$). Baseline cortisol, child trauma history, peritraumatic dissociation, and HR were not related to Week 12 PTSD for INT (see Table 3). The total model accounted for 51% of the variance in Week 12 PTSD. For TAU, PTSD symptoms significantly changed from Week 4 to Week 12 ($\gamma_{10} = -4.89, p = .010$). Baseline cortisol was negatively related to PTSD at Week 12 ($\gamma_{04} = -30.16, p = .002$), but child trauma history was not ($\gamma_{03} = 0.14, p = .030$). Peritraumatic dissociation was positively related to PTSD ($\gamma_{01} = 0.95, p = .003$), yet postevent dissociation ($\gamma_{02} = -0.07, p = .958$) and HR were not (see Table 3). The model accounted for 68% of the variance in Week 12 PTSD.

Dissociation at the start of treatment for INT and peritraumatic dissociation for TAU were used to identify a cutoff point to predict PTSD status at Week 12 (see Figure 1 for ROC curves for dissociation as predictor of PTSD status). At Week 12, 26% ($n = 11$) of INT and 47% ($n = 23$) of TAU were classified as having PTSD. For INT, dissociation at the start of treatment accounted for 73% of the area under the curve ($p = .025$), which was classified as fair (Metz, 1978). The cutoff with the optimal balance of sensitivity (0.91) and specificity (0.57) was 2. This score correctly identified 56% of the sample who received the intervention who did not have PTSD at Week 12. For TAU, peritraumatic dissociation accounted for 66% of the area under the curve ($p = .058$), which was classified as poor.

Discussion

The high prevalence of trauma exposure coupled with challenges in delivering universal prevention demonstrates a clear need for methods to identify those at greatest risk for PTSD and those most likely to benefit from specific interventions. The present study suggested sexual assault victims and those with reduced dissociation at the start of treatment benefited most from early treatment. Increased dissociation, childhood trauma exposure, and low salivary cortisol were associated with more severe PTSD symptoms in those who did not receive treatment.

High levels of dissociation at the start of treatment may interfere with treatment response through reduced acquisition of new inhibitory learning (Lanius et al., 2012). Inhibitory learning is the acquisition of nonfearful learning during exposure and is the presumed mechanism by which exposure therapy is effective (Craske et al., 2008). Dissociation at the start of an early intervention may serve as a clinically useful indicator of treatment response. The current study suggested that a score of 2 or greater on the ISRC used in the current study could identify those who are less likely to respond. This finding should be considered preliminary, however, pending replication with other early interventions and traditional exposure therapy approaches.

The present study did not assess dissociation at subsequent treatment sessions, however. It is unclear if variation in dissociation across treatment influences response or if dissociation at the start of treatment is predictive of outcomes. This distinction is important given that related research has shown that fluctuations in symptoms across sessions interfere with response to exposure therapy (Price & Anderson, 2011). Such findings would inform

clinical practice in that elevated dissociation at the start of a given session would help the therapist plan a given session. The optimal course of treatment must also be explored to determine if a higher dose of exposure is needed or if use of alternative treatments (e.g., coping strategies) would be more beneficial.

Findings in the TAU condition corroborate prior research showing that a reduced cortisol reaction at the time of trauma exposure, indicative of a chronic stress response, and increased peritraumatic dissociation represent possible mechanisms of PTSD development (Ozer, Best, Lipsey, & Weiss, 2003). However, the measure of peritraumatic dissociation did not identify those at greatest risk for PTSD according to the ROC analysis. It is unclear if this was due to the measure used in the current study, the sample size, or the predictive utility of self-reported dissociation. Additional research on this topic is needed.

A limitation of the study was the assessment of PTSD only at 4 and 12 weeks posttrauma. Additional follow-up periods may have provided further insight into the relationship between the proposed risk factors and PTSD outcome over time. In addition, limited information is available about the participants' experiences in the intervening hours between trauma exposure and enrollment in the study. Although this is a brief time frame (median = 6.92; $M = 11.79$ hr, $SD = 12.90$) relative to other research on predictors of PTSD and early intervention, we believe it is a critical period for the formation of fear memories. Furthermore, the sample size was not sufficiently powered to detect small to medium effects in such models. Work with larger, noninferiority samples is needed to conclusively determine if these variables have clinically useful associations with early intervention response and PTSD risk. Last, the dropout rates and exclusion criteria may limit the generalizability of the findings to all trauma-exposed individuals.

Despite these limitations, the findings of this study have important implications for future research on PTSD and early intervention. Screening for peritraumatic dissociation and low cortisol levels in recent trauma survivors could identify those at greatest risk and those who need further intervention. For those who receive early intervention and treatment, the present findings highlight the potentially interfering role of dissociation at the start of treatment. Additional work is needed to develop clinically useful screening tools from these measures.

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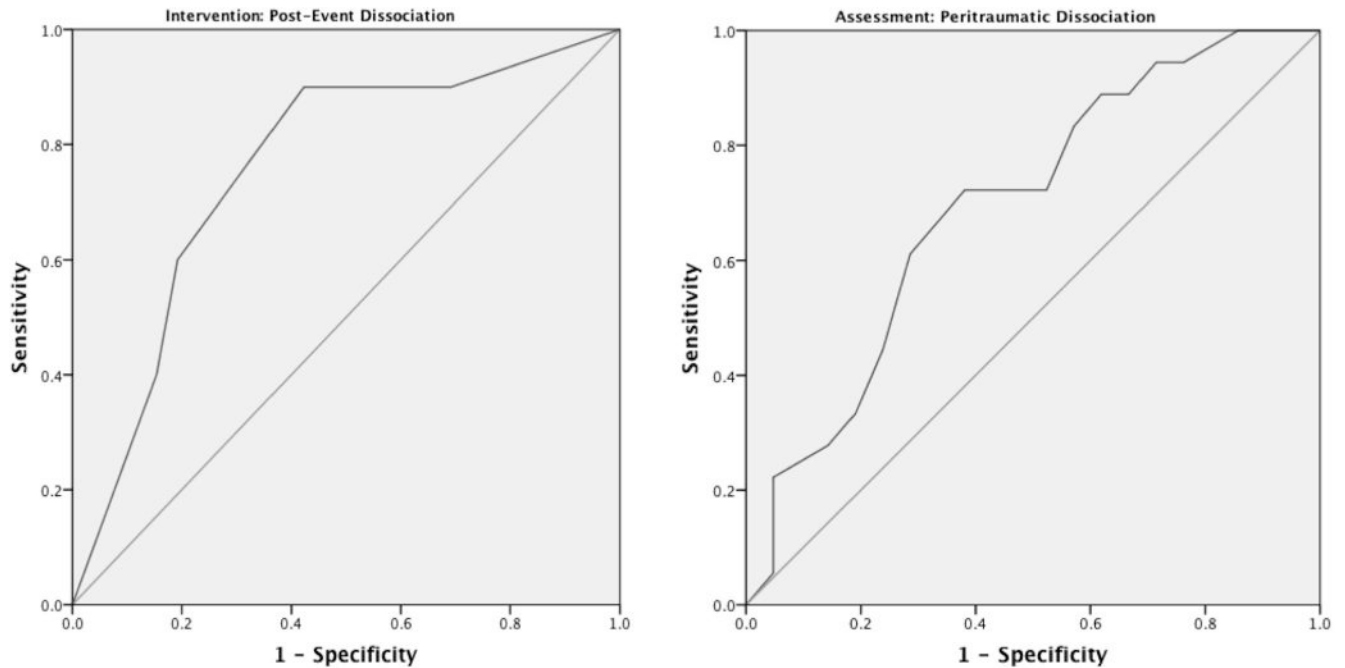


Figure 1.

Receiver operating characteristic (ROC) curves for intervention and assessment (treatment as usual) conditions using the respective dissociation variables as predictors of posttraumatic stress disorder diagnostic status at Week 12. The ROC curve for the intervention condition used postevent dissociation. The ROC curve for the assessment condition used peritraumatic dissociation.

Table 1
Descriptive Statistics for Intervention and Treatment as Usual Conditions at Week 4 and Week 12

	Intervention		Treatment as usual	
	Week 4 (<i>n</i> = 47)	Week 12 (<i>n</i> = 42)	Week 4 (<i>n</i> = 55)	Week 12 (<i>n</i> = 49)
PTSD symptoms	19.19 (12.32)	15.97 (11.68)	23.03 (11.24)	19.03 (12.17)
	Baseline variables		Baseline variables	
Peritraumatic dissociation	11.98 (5.29)		10.65 (4.74)	
Postevent dissociation	2.31 (1.83)		1.81 (1.67)	
Child trauma exposure	49.60 (20.98)		50.27 (20.79)	
Cortisol	0.15 (0.21)		0.15 (0.14)	
Heart rate	84.42 (16.41)		84.73 (19.20)	
Sexual assault (%)	41		30	

Note. Values in parenthesis are standard deviations. Posttraumatic stress disorder (PTSD) was measured with the Posttraumatic Symptom Scale–Interview Version. Peritraumatic and postevent dissociation were measured with the Immediate Stress Reaction Checklist. Child trauma exposure was measured with the Child Trauma Questionnaire.

Table 2
Correlations for Intervention and Treatment as Usual Conditions at Week 4 and Week 12

Condition	1	2	3	4	5	6
Treatment as usual (Week 4)						
1. PTSD symptoms	1.00	0.18	0.43**	0.29*	-0.10	0.03
2. Child trauma exposure		1.00	0.03	0.11	-0.29*	-0.28*
3. Peritraumatic dissociation			1.00	0.56**	0.02	-0.07
4. Postevent dissociation				1.00	0.14	0.03
5. Cortisol					1.00	0.28*
6. Heart rate						1.00
Treatment as usual (Week 12)						
1. PTSD symptoms	1.00	0.19	0.38**	0.25	-0.09	0.12
2. Child trauma exposure		1.00	0.03	0.11	-0.29*	-0.28*
3. Peritraumatic dissociation			1.00	0.56**	0.02	-0.07
4. Postevent dissociation				1.00	0.14	0.03
5. Cortisol					1.00	0.28*
6. Heart rate						1.00
Intervention (Week 4)						
1. PTSD symptoms	1.00	0.28	0.37*	0.43**	0.07	0.01
2. Child trauma exposure		1.00	0.08	0.13	-0.11	-0.13
3. Peritraumatic dissociation			1.00	0.63**	-0.03	0.04
4. Postevent dissociation				1.00	-0.09	-0.17
5. Cortisol					1.00	0.14
6. Heart rate						1.00
Intervention (Week 12)						
1. PTSD symptoms	1.00	0.29	0.27	0.43**	0.09	0.00
2. Child trauma exposure		1.00	0.08	0.13	-0.11	-0.13
3. Peritraumatic dissociation			1.00	0.63**	-0.03	0.04
4. Postevent dissociation				1.00	-0.09	-0.17

Condition	1	2	3	4	5	6
5. Cortisol					1.00	0.14
6. Heart rate						1.00

Note. Posttraumatic stress disorder (PTSD) was measured with the Posttraumatic Symptom Scale-Interview Version. Child trauma exposure was measured with the Child Trauma Questionnaire. Peritraumatic and postevent dissociation were measured with the Immediate Stress Reaction Checklist.

* $p < .05$.

** $p < .01$.

Table 3
Parameters for Models of Predictors of PTSD Symptoms at Week 4 and Week 12 for
Those in the Intervention Condition and Those in the Treatment as Usual Condition

Variable	Parameter (SE)	<i>p</i>	95% CI	Standardized effect ^a
Intervention				
Fixed effects				
Week 12 (intercept)	20.06 (2.53)	<.001	[15.11, 25.02]	—
Peritraumatic dissociation	−0.37 (0.42)	.373	[−1.18, 0.44]	0.16
Postevent dissociation	5.08 (1.18)	<.001	[2.76, 7.39]	0.74
Child trauma exposure	0.17 (0.09)	.057	[−0.01, 0.34]	0.29
Cortisol	14.69 (15.09)	.33	[−14.89, 44.28]	0.25
Heart rate	0.04 (0.08)	.611	[−0.12, 0.20]	0.06
Sexual assault	−11.32 (3.89)	.004	[−18.95, −3.68]	0.46
Week 4 to Week 12 change	−3.58 (1.48)	.015	[0.68, 6.47]	0.15
Random effect				
Intercept	55.98 (24.13)	.020	[8.68, 103.27]	
Treatment as usual				
Fixed effects				
Week 12 (intercept)	17.80 (1.89)	<.001	[14.10, 21.50]	0.21
Peritraumatic dissociation	0.95 (0.32)	.003	[0.62, 1.57]	0.37
Postevent dissociation	−0.07 (1.25)	.958	[−2.52, 2.39]	0.01
Child trauma exposure	0.14 (0.06)	.030	[0.01, 0.26]	0.24
Cortisol	−30.16 (9.51)	.002	[−48.81, −11.52]	6.41
Heart rate	0.14 (0.08)	.095	[−0.02, 0.29]	0.20
Sexual assault	3.15 (3.31)	.340	[−3.33, 9.63]	0.12
Week 4 to Week 12 change	−4.89 (1.89)	.010	[1.19, 8.59]	0.21
Random effect				
Intercept	30.62 (17.70)	.084	[−4.08, 65.32]	

Note. Posttraumatic stress disorder (PTSD) was measured with the Posttraumatic Symptom Scale-Interview Version. Child trauma exposure was measured with the Child Trauma Questionnaire. Peritraumatic and postevent dissociation were measured with the Immediate Stress Reaction Checklist. CI = confidence interval.

^aStandardized effects were calculated according to the recommendations of Hox (2010) and correspond to standardized unit changes in PTSD symptoms per 1 standardized unit change in the predictor. Absolute values are presented to facilitate interpretation.