

# Neural and Behavioral Correlates of Peritraumatic Dissociation in an Acutely Traumatized Sample

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## ABSTRACT

**Objective:** Peritraumatic dissociative responses have been identified as strong predictors of subsequent posttraumatic stress disorder (PTSD) development. We aimed to clarify the mechanism by which peritraumatic dissociation is related to PTSD development by exploring the neural correlates of peritraumatic dissociation during posttraumatic adjustment.

**Method:** We combined a prospective questionnaire study with a neuroimaging paradigm in an acutely traumatized sample recruited from the emergency department from 2004 until 2009. 121 acutely traumatized subjects were assessed for acute stress disorder, PTSD, and dissociative symptoms at 3 time points within the first 3 months post trauma. A subsample of 21 subjects underwent a script-driven 4-Tesla functional magnetic resonance imaging scan 2 to 4 months post trauma.

**Results:** Peritraumatic dissociation predicted PTSD diagnostic status at 5–6 weeks and 3 months over and above childhood trauma (Wald = 4.035,  $P = .045$ ; Wald = 4.793,  $P = .029$ , respectively). Peritraumatic dissociation scores were positively correlated with activation in the right occipital lobe, ie, the lingual (Brodmann area [BA] 18,  $z = 3.37$ ), fusiform (BA 19,  $z = 3.64$ ), and parahippocampal (BA 19,  $z = 3.25$ ) gyri. After covariation of dissociation at the time of the scan, peritraumatic dissociation remained positively correlated with activation in the right lingual (BA 18,  $z = 3.21$ ) and fusiform (BA 19,  $z = 3.55$ ) gyri.

**Conclusions:** The neuroimaging findings indicate that peritraumatic dissociation is associated with greater activation of the right occipital lobe (BAs 18 and 19), a region previously implicated in vivid autobiographical memory recall of highly emotional events. These results suggest that peritraumatic dissociation directly leads to the formation of intrusive memories. Peritraumatic dissociation and childhood trauma emerged as valuable predictors of PTSD development and therefore can guide the identification of individuals at risk.

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Although many people experience traumatic events, only some subsequently develop posttraumatic stress disorder (PTSD). Previously, dissociative responses during the traumatic incident have been identified as a strong predictor of PTSD.<sup>1–3</sup> This so-called peritraumatic dissociation typically comprises perceptual alterations, emotional detachment from one's surroundings, and a loss of agency. Two complementary trajectories of the adverse effects of peritraumatic dissociation have been discussed (for a review, see Waelde et al<sup>4</sup>). It has been hypothesized that peritraumatic dissociation causes (1) alterations in memory formation during the traumatic event, which lead to intrusions,<sup>1,2</sup> and (2) the persistence of dissociative reactions upon exposure to trauma cues after the traumatic event, which predicts PTSD development over and above peritraumatic dissociation.<sup>5</sup>

## Peritraumatic Dissociation and Memory Formation

Etiologic theories of PTSD state that peritraumatic dissociation leads to alterations in memory encoding and storage, which in turn cause intrusions as well as partial amnesias typical for PTSD.<sup>1,2,6</sup> Intrusions are considered a recall of memory fragments that lack temporal and spatial contextualization and carry a high load of sensory associations. Posttraumatic stress disorder severity is strongly correlated with distress associated with intrusions, lack of context, and the sense that the memories are being relived.<sup>7</sup> These characteristics predict the severity of PTSD 6 months later over and above initial diagnostic status and appear to be related to the presence of PTSD rather than to be general characteristics of trauma memories. Different theorists have proposed dual-representation memory models that might be helpful in elucidating the underlying neurobiology of intrusive memory recall.<sup>1,8</sup> These models assume that an event will be encoded both in a sensory format lacking a contextual framework and as autobiographical memories embedded in the continuous stream of personal memories. Conway<sup>8</sup> proposed that intrusions constitute involuntary memory recall from an imagery-based memory system in the temporal-occipital region storing the sensory aspects of an event. In this view, peritraumatic dissociation may be the causal agent behind intrusions, as it is assumed that the integration into the autobiographical memory system is inhibited during dissociative processing.<sup>2</sup>

## Persistent Dissociation Upon Exposure to Trauma Cues

Persistent dissociation is a common feature of chronic PTSD and typically consists of depersonalization, derealization, alterations in time sense, and analgesia.<sup>9,10</sup> In persons who experienced acute dissociative responses to psychological trauma, persistent dissociation can be evoked by reminders of the original trauma and minor stressors.<sup>11</sup> Recently, it has been proposed to differentiate a dissociative subtype of PTSD,<sup>12</sup> as a subset of PTSD patients showing differential autonomic reactions<sup>13,14</sup> and brain activation patterns during exposure to trauma cues<sup>15,16</sup> has been identified. Typically, autonomic hypoarousal, instead of hyperarousal, is associated with dissociative reactions,<sup>17</sup> which seems to interfere with emotional learning.<sup>18</sup> This suggests that persistent dissociation might

impair extinction learning and that highly dissociative patients may need therapeutic modules focusing on emotion regulation before being able to fully profit from standard, exposure-based psychotherapies.<sup>19</sup>

### Childhood Abuse and Dissociation

Two meta-analyses<sup>20,21</sup> identified prior traumatization as a major risk factor for the development of PTSD after an acute traumatic event during adulthood, and, in the National Comorbidity Survey, physical and sexual childhood abuse were uniquely related to adult PTSD.<sup>22</sup> Repeated childhood sexual or physical abuse and emotional neglect have been associated with the development of dissociative symptoms,<sup>23,24</sup> raising the question whether the relationship between childhood maltreatment and PTSD development is mediated by peritraumatic and persistent dissociation in acutely traumatized, adult subjects. Specifically, it is unknown whether the adverse effects of peritraumatic dissociation are predominantly due to the alterations in memory formation during the traumatic event or, alternatively, to the persistence of dissociative reactions upon exposure to trauma cues after the traumatic event.

### Neural Correlates of Dissociative Processing

Neuroimaging studies of dissociation occurring during the scan in PTSD have shown altered activation of brain regions implicated in arousal modulation and emotion regulation, especially the medial prefrontal cortex (Brodmann areas [BAs] 9 and 10), the anterior cingulate cortex (BAs 32 and 24), and the limbic system including the amygdala.<sup>15,16,25</sup> Convergent with evidence that activation of the medial prefrontal cortex correlates negatively with amygdala activity during script-driven imagery,<sup>26</sup> these studies suggest that the activation pattern during acute dissociation may be described as one of overmodulation<sup>12</sup> of limbic activity.

### Aims of the Study

The aim of this study was to clarify the mechanism by which peritraumatic dissociation is related to PTSD development and to explore the neural correlates of peritraumatic dissociation during posttraumatic adjustment. To this end, we combined a prospective questionnaire study with assessments at 1 to 2 weeks, 5 to 6 weeks, and 3 months post trauma with a neuroimaging paradigm in an acutely traumatized sample. In the functional magnetic resonance imaging (fMRI) investigation, we contrasted the neural correlates of peritraumatic dissociation and dissociation elicited by script-driven trauma recall during the scan. We hypothesized that peritraumatic dissociation would be associated with activation in either memory-related areas as seen during intrusive recall or emotion regulation areas as seen during dissociation elicited by traumatic script-driven imagery.<sup>15,26</sup>

In addition, the predictive power of accounts of childhood abuse and posttraumatic dissociative symptoms were examined comparatively by using self-report measures

- Peritraumatic dissociation is a risk factor for the development of posttraumatic stress disorder and can help to determine who is at risk and should thus be offered close monitoring or early interventions in the acute period following a traumatic event.
- Peritraumatic dissociation therefore constitutes an important marker that should be routinely assessed shortly after the traumatic event.
- Subjects who experience peritraumatic dissociation are most likely to exhibit persistent dissociation following the traumatic event and may therefore require treatment that focuses on a reduction of dissociative processing.

## METHOD

### Participants and Procedure

This study included 121 subjects (mean  $\pm$  SD age =  $38.2 \pm 13.7$  years; women,  $n = 84$ ) who presented consecutively to the emergency department at the London Health Sciences Center in London, Ontario, Canada, or to the Department of Emergency Medicine at the University of Alberta in Edmonton, Canada, after they had been involved in a motor vehicle crash ( $n = 104$ ), workplace accident ( $n = 9$ ), physical assault ( $n = 4$ ), or other traumatic events ( $n = 4$ ). Subjects were assessed shortly after admission, and those meeting *DSM-IV* criterion A for PTSD were included in the study. Subjects were assessed 3 times within the first 3 months post trauma. A convenience subsample of 21 subjects (mean  $\pm$  SD age =  $38.2 \pm 12.1$  years; women,  $n = 17$ ; motor vehicle crash,  $n = 16$ ; workplace accident,  $n = 3$ ; physical assault,  $n = 1$ ; other traumatic event,  $n = 1$ ) underwent an fMRI scan 2 to 4 months post trauma. Exclusion criteria included (1) significant head injury; (2) history of neurologic disorders; (3) lifetime history of bipolar disorder or schizophrenia; and (4) psychotropic or steroid medication (for the fMRI sample only). The study was approved by the research ethics boards at University of Western Ontario and University of Alberta. Informed written consent was obtained from all participants.

### Functional Imaging Paradigm

The script-driven imagery procedure was adapted to fMRI according to well-established methods.<sup>27</sup>

### fMRI Procedures

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. A whole-head hybrid birdcage radio frequency coil was used for transmission and detection of signals. Each functional brain volume was acquired by using a navigator echo-corrected, interleaved multi-shot (4 shots), echo-planar imaging pulse sequence with a  $128 \times 128$  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

For each series, all volumes were realigned to the first volume of series to reduce the effects of head motion using SPM 2 (Wellcome Department of Imaging Neuroscience, London, England). The realigned functional images were then spatially normalized to an echo-planar imaging template supplied by SPM 2. The functional data were spatially smoothed with an 8-mm (full width at half maximum) isotropic Gaussian kernel.

### Statistical Mapping

Images were analyzed using a 2-stage mixed-effects analysis. At the first level, each subject's functional data were analyzed separately by modeling the evoked blood oxygen level–dependent (BOLD) responses for each task epoch of interest as a boxcar function convolved with a hemodynamic response function. Baseline brain activation was calculated on the basis of the average activation patterns 60 seconds before each recollection of the traumatic event, and brain activation during the recall was calculated on the basis of the average activation patterns during the final 30 seconds of recall. 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 with the Detailed Assessment of Posttraumatic Stress<sup>31</sup> peritraumatic dissociation scores and Responses to Script-Driven Imagery Scale<sup>17</sup> acute dissociation scores were conducted separately and as partial correlation analysis with the Responses to Script-Driven Imagery Scale scores and results were masked with the Detailed Assessment of Posttraumatic Stress correlation. All analyses were thresholded at  $\kappa > 10$  and  $P = .001$ , requiring contiguous voxels to meet the small-volume correction according to random field theory. In addition, the time since the traumatic event was used as a covariate in all analyses.

### Measures

The Acute Stress Disorder Scale<sup>28</sup> ( $\alpha = .945$ ) is a 19-item questionnaire measuring the severity of acute stress disorder symptoms on a 5-point Likert scale (1 = not at all, 5 = very much; cutoff of 56 to establish diagnostic status 1 to 2 weeks post trauma).<sup>28</sup>

The Clinician-Administered PTSD Scale<sup>29</sup> ( $\alpha = .952$ ) is a semistructured clinical interview assessing posttraumatic symptoms as defined in *DSM-IV*, and a cutoff score of 50 was employed to establish diagnostic status at 5 to 6 weeks and 3 months post trauma. Severity scores were calculated as the sum of frequency and intensity ratings across all *DSM-IV* symptoms of PTSD.

The Childhood Trauma Questionnaire Short Form<sup>30</sup> consists of 25 clinical items assessing emotional abuse, emotional neglect, physical abuse, physical neglect, and sexual abuse on a 5-point Likert scale (1 = never true to 5 = very often true).

The Detailed Assessment of Posttraumatic Stress<sup>31</sup> is a 104-item inventory assessing peritraumatic and posttraumatic symptoms. The subscale peritraumatic dissociation consists of 6 items scored on a 5-point Likert scale (0 = not at all to 5 = very much) and was assessed at 5 to 6 weeks post trauma as a retrospective report of dissociation during the traumatic event (“Your mind went blank”; “Things around you felt unreal or strange”). In 13 subjects not available at this time, the Detailed Assessment of Posttraumatic Stress was filled out 3 months post trauma.

The Multiscale Dissociation Inventory<sup>32</sup> is a standardized self-report test of dissociative symptomatology (“Knowing you must be upset, but not being able to feel it”; “Feeling like you were in a dream”). Using a 5-point Likert scale (0 = never to 5 = very often), it measures the frequency of 6 types of dissociative responses within the last month: disengagement, depersonalization, derealization, emotional constriction, memory disturbance, and identity dissociation. This scale was used as a measure of persistent dissociation at 5 to 6 weeks post trauma.

The Responses to Script-Driven Imagery Scale<sup>17</sup> is an 11-item self-report measure of PTSD and dissociative symptoms evoked by script-driven imagery using a 7-point Likert scale (0 = not at all to 6 = a great deal). The dissociation subscale consists of 4 items specifically assessing dissociation during the fMRI scan, ie, the extent to which the subject acutely experiences derealization, depersonalization, and a sense of disconnection from his or her body during the scan (“Did you feel like you were in a fog?” “Did you feel like you were a spectator watching what was happening to you, like an observer or outsider?”). This measure was administered immediately following the scan to assess the level of dissociation induced by the exposure to individualized trauma scripts during the scanning procedure.

### Statistics

Group statistics were calculated as Spearman rank correlations and 2-sample Mann-Whitney *U* test due to extensive positive skew in most variables. A stepwise, binary logistic regression analysis with Wald  $\chi^2$  statistics was employed to predict diagnostic status. Variables were entered in blocks in order of occurrence, so the first block contained Childhood Trauma Questionnaire Short Form scores, the second block contained Detailed Assessment of Posttraumatic Stress scores, and the third block contained Multiscale Dissociation Inventory scores. Statistical analyses were carried out using Statistical Package for Social Sciences, Version 15.0 (SPSS Inc, Chicago, Illinois).

## RESULTS

### fMRI Results

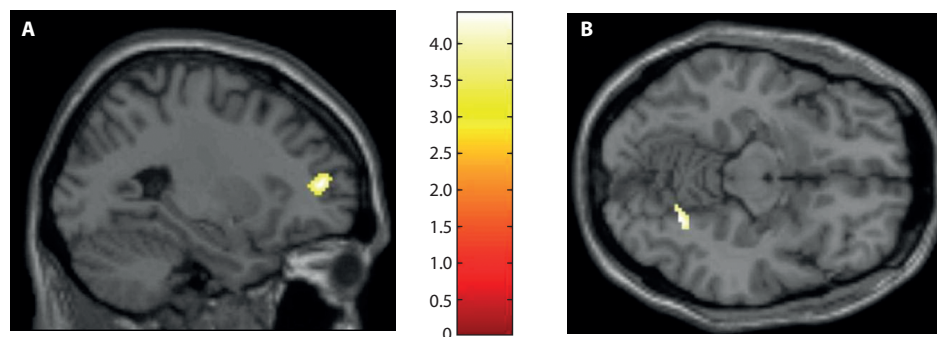
During script-driven imagery of the traumatic versus the neutral event, Responses to Script-Driven Imagery Scale ratings for dissociation during the scan correlated positively with BOLD signal intensity in the bilateral prefrontal cortex (Table 1 and Figure 1), ie, the left middle frontal (BA 10) and

**Table 1. Correlation Between Peritraumatic Dissociation and Dissociation at the Time of the Scan and Brain Activation During Trauma Script-Driven Imagery (n = 21)<sup>a</sup>**

MNI Coordinates (local maxima)	z Score	Cluster Size, Voxels	Brain Region
Positive correlations with DAPS peritraumatic dissociation scores			
26, -60, -12	3.64	55	Fusiform gyrus, BA 19
26, -64, -10	3.47		Fusiform gyrus, BA 19
10, -78, -8	3.37	43	Lingual gyrus, BA 18
8, -82, -6	3.29		Lingual gyrus, BA 18
4, -80, -8	3.27		Lingual gyrus, BA 18
14, -70, -10	3.20		Lingual gyrus, BA 18
40, -44, -10	3.25	11	Parahippocampal gyrus, BA 19
Positive correlations with RSDI scores for acute dissociation			
-26, 48, 10	4.12	105	Middle frontal gyrus, BA 10
10, 52, 46	4.16	32	Superior frontal gyrus, BA 8
Positive correlations with DAPS scores after covariation of RSDI scores			
26, -60, -12	3.55	54	Fusiform gyrus, BA 19
10, -78, -8	3.21	10	Lingual gyrus, BA 18

<sup>a</sup>All analyses are covaried for time since trauma, significant at  $P = .001$ , small volume corrected.

Abbreviations: BA = Brodmann area, DAPS = Detailed Assessment of Posttraumatic Stress, MNI = Montreal Neurological Institute, RSDI = Responses to Script-Driven Imagery Scale.

**Figure 1. Neural Correlates of Acute and Peritraumatic Dissociation<sup>a</sup>**

<sup>a</sup>Numbers in the color bar indicate  $t$  values.

Image A depicts neural activations in the middle frontal gyrus (-26, 48, 10) correlated with Responses to Script-Driven Imagery Scale scores for acute dissociation during the scan, with time since trauma as a covariate ( $P = .001$ ) and small-volume correction.

Image B depicts neural activation in the fusiform gyrus (26, -60, -12) correlated with Detailed Assessment of Posttraumatic Stress scores for peritraumatic dissociation; the Responses to Script-Driven Imagery Scale scores for acute dissociation during the scan and time since trauma were used as covariates ( $P = .001$ ), with small-volume correction.

the right superior frontal gyri (BA 8). Peritraumatic dissociation scores were positively correlated with activation in the right occipital lobe, ie, the lingual (BA 18), fusiform (BA 19), and parahippocampal (BA 19) gyri. After covariation of dissociation during the scan, peritraumatic dissociation remained positively correlated with activation in the right lingual (BA 18) and fusiform (BA 19) gyri.

### Descriptive Statistics and Zero-Order Correlations

Descriptive statistics for all scales are listed in Table 2. Thirty-three of the 90 subjects (36.7%) assessed with the Acute Stress Disorder Scale 1 to 2 weeks post trauma met the diagnostic cutoff for acute stress disorder. Five to 6 weeks post trauma, 25 of the 102 subjects (24.5%) who underwent the Clinician-Administered PTSD Scale interview were diagnosed with PTSD. At 3 months post trauma, 92 subjects were available for interviewing with the Clinician-Administered PTSD Scale, 12 (13.0%) of whom met diagnostic criteria for PTSD. The subgroup undergoing fMRI scanning did not differ significantly

**Table 2. Descriptive Statistics of the Total and fMRI Samples**

Scale	Total Sample		fMRI Subsample		$P$
	n	Mean $\pm$ SD	n	Mean $\pm$ SD	
CTQ	106	43.49 $\pm$ 18.58	16	45.50 $\pm$ 24.18	.846
DAPS peritraumatic dissociation	121	14.28 $\pm$ 6.17	21	14.33 $\pm$ 4.92	.739
ASDS	90	46.93 $\pm$ 19.52	14	20.43 $\pm$ 5.46	.548
CAPS (5–6 weeks post trauma)	102	31.95 $\pm$ 26.05	18	32.28 $\pm$ 5.19	.693
CAPS (3 months post trauma)	92	22.50 $\pm$ 21.34	15	19.67 $\pm$ 20.22	.582
MDI-disengagement	103	9.05 $\pm$ 3.45	16	9.19 $\pm$ 2.76	.545
MDI-depersonalization	100	5.65 $\pm$ 1.62	16	5.75 $\pm$ 2.27	.603
MDI-derealization	103	6.34 $\pm$ 2.12	16	6.50 $\pm$ 1.75	.466
MDI-emotional constriction	102	6.37 $\pm$ 2.63	15	6.80 $\pm$ 3.05	.869
MDI-memory lapse	103	6.38 $\pm$ 2.14	16	6.31 $\pm$ 1.58	.580
MDI-identity dissociation	102	5.22 $\pm$ 0.78	16	5.56 $\pm$ 1.36	.161

Abbreviations: ASDS = Acute Stress Disorder Scale, CAPS = Clinician-Administered PTSD Scale, CTQ = Childhood Trauma Questionnaire Short Form, DAPS = Detailed Assessment of Posttraumatic Stress, fMRI = functional magnetic resonance imaging, MDI = Multiscale Dissociation Inventory, PTSD = posttraumatic stress disorder.



**Table 3. Zero-Order Correlations**

	2	3	4	5	6	7	8	9	10	11 MDI-Identity Dissociation
1 ASDS	0.577**	0.569**	0.266*	0.656**	0.501**	0.435**	0.298**	0.358**	0.352**	0.060
2 CAPS (5–6 weeks post trauma)		0.654**	0.326**	0.320**	0.421**	0.266**	0.150	0.218*	0.236*	0.191
3 CAPS (3 months post trauma)			0.307**	0.410**	0.373**	0.284*	0.122	0.285*	0.195	0.063
4 CTQ				0.175	0.364**	0.342**	0.263**	0.292**	0.271**	0.094
5 DAPS-peritraumatic dissociation					0.467**	0.441**	0.354**	0.258**	0.270**	0.144
6 MDI-derealization						0.555**	0.560**	0.477**	0.448**	0.209*
7 MDI-disengagement							0.375**	0.557**	0.655**	0.226*
8 MDI-depersonalization								0.321**	0.389**	0.278**
9 MDI-emotional constriction									0.486**	0.167
10 MDI-memory lapse										0.207*

\* $P = .05$ . \*\* $P = .001$ .

Abbreviations: ASDS = Acute Stress Disorder Scale, CAPS = Clinician-Administered PTSD Scale, CTQ = Childhood Trauma Questionnaire, DAPS = Detailed Assessment of Posttraumatic Stress, MDI = Multiscale Dissociation Inventory, PTSD = posttraumatic stress disorder.

**Table 4. Stepwise, Binary Logistic Regression Analyses**

Regressand	Block	$\chi^2$	Nagelkerkes $R^2$	Predictor Variable	Nonstandardized Coefficient $\beta$	Wald	$P$
PTSD at 5–6 weeks <sup>a,b</sup>	1	9.498*	0.145	CTQ	.037	8.582	.003
	2	15.544**	0.230	CTQ	.034	6.243	.012
				DAPS	.105	5.703	.017
	3	19.625**	0.284	CTQ	.036	6.644	.010
				DAPS	.091	4.035	.045
				MDI-identity dissociation	.688	2.779	.096
PTSD at 3 months <sup>c</sup>	1	5.104*	0.129	CTQ	.038	5.048	.025
	2	10.471	0.256	CTQ	.031	3.029	.082
				DAPS	.157	4.793	.029

<sup>a</sup>Diagnostic status of PTSD was defined as CAPS score  $< 50$  or  $\geq 50$ . <sup>b</sup> $n = 92$ . <sup>c</sup> $n = 72$ .\* $P = .05$ . \*\* $P = .001$ .

Abbreviations: CTQ = Childhood Trauma Questionnaire, DAPS = Detailed Assessment of Posttraumatic Stress, MDI = Multiscale Dissociation Inventory, PTSD = posttraumatic stress disorder.

from the remaining sample in terms of psychopathology (see Table 2).

The Detailed Assessment of Posttraumatic Stress scores for peritraumatic dissociation were significantly related to symptom severity at 1 to 2 weeks ( $r = 0.656$ ), 5 to 6 weeks ( $r = 0.320$ ), and 3 months ( $r = 0.410$ ) post trauma (Table 3). These correlations indicate that stronger peritraumatic dissociation was related to more severe PTSD symptomatology throughout the first 3 months after the traumatic event. Peritraumatic dissociation was not significantly correlated with exposure to childhood trauma as measured with the Childhood Trauma Questionnaire Short Form ( $r = 0.175$ ,  $P = .072$ ). In turn, Childhood Trauma Questionnaire Short Form scores were significantly correlated with PTSD symptom severity at 1 to 2 weeks ( $r = 0.266$ ), 5 to 6 weeks ( $r = 0.326$ ), and 3 months ( $r = 0.307$ ) post trauma. Both Childhood Trauma Questionnaire Short Form scores and Detailed Assessment of Posttraumatic Stress scores for peritraumatic dissociation correlated significantly with severity of dissociative symptoms at 5 to 6 weeks post trauma on all Multiscale Dissociation Inventory subscales, except for the one measuring identity dissociation.

### Regression Analyses

In a stepwise, binary logistic regression analysis, both Childhood Trauma Questionnaire Short Form scores and Detailed Assessment of Posttraumatic Stress scores were

identified as significant predictors of diagnostic status at 5 to 6 weeks post trauma. In the third block containing all Multiscale Dissociation Inventory subscales, only the subscale identity dissociation was identified as a predictor, but it failed to reach significance (Table 4). In the prediction of diagnostic status 3 months after the traumatic event, Childhood Trauma Questionnaire Short Form scores were a significant predictor in the first block, but they were rendered insignificant by the inclusion of the Detailed Assessment of Posttraumatic Stress peritraumatic dissociation scores.

### DISCUSSION

To our knowledge, this is the first study investigating the neural correlates of peritraumatic dissociation during trauma recall in an acutely traumatized sample. The neuroimaging results suggest that peritraumatic dissociation is associated with greater activation of the right occipital lobe (BAs 18 and 19), regions previously implicated in vivid autobiographical memory recall of highly emotional events. These findings may indicate that peritraumatic dissociation directly leads to the formation of intrusive memories. In conjunction, we analyzed the predictive value of peritraumatic and persistent dissociation in a prospective questionnaire study. Results suggest that peritraumatic dissociation does not fully mediate the relationship between childhood trauma and posttraumatic adjustment; however, both variables

constitute significant predictors for the development of PTSD at 5 to 6 weeks after the traumatic event. For the prediction of diagnostic status 3 months post trauma, only peritraumatic dissociation was identified as a significant predictor.

### fMRI Study

Consistent with previous reports,<sup>15,16,25</sup> dissociation during the scan was strongly correlated with bilateral activations of prefrontal cortex regions implicated in emotion regulation.<sup>33,34</sup> The left-sided activation in the middle frontal gyrus (BA 10) is consistent with the extensive activations characterizing dissociative versus intrusive PTSD symptoms reported by Hopper et al<sup>16</sup> and directly coincides with regions implicated in modulation of emotion.<sup>33,34</sup>

Peritraumatic dissociation was correlated with BOLD signal intensity in the right occipital lobe. This association remained significant in the right medial lingual and fusiform gyri after covariation of dissociation during the scan. Previous studies have implicated these regions in emotional autobiographic memory recall (for a meta-analysis see Svoboda et al<sup>35</sup>) as well as visual imagery.<sup>36,37</sup> In a study by Shergill et al,<sup>38</sup> activation in the fusiform region was correlated with visual imagery characterized by a first-person perspective. Convergingly, Gilboa et al<sup>39</sup> reported a relationship between BOLD signal intensity in the lingual gyrus and greater vividness of the autobiographical memory recall. The overlap in areas of visual processing and autobiographic memory during trauma recall could be interpreted as enhanced visual processing for highly emotional arousing events, possibly driven by feed-forward amygdala connectivity.<sup>40</sup> In conjunction, these results indicate that peritraumatic dissociation is associated with brain activations underlying intrusive trauma recall in this script-driven paradigm and thus strongly support the model proposed by Conway.<sup>8</sup> Interestingly, similar activations in the fusiform gyrus have previously been associated with the recall of unresolved life events in a sample of patients with borderline personality disorder<sup>41</sup>—a population that is characterized by high rates of traumatization.

### Childhood Trauma, Peritraumatic Dissociation, and PTSD Symptoms

Childhood trauma was significantly correlated with symptom severity at all 3 time points as well as with dissociative symptoms as measured with all Multiscale Dissociation Inventory subscales except identity dissociation. The significant correlations between childhood trauma and persistent dissociation explain why the results reported here differ from those reported previously<sup>5</sup>: after accounting for childhood trauma and peritraumatic dissociation, persistent dissociation, as measured with the Multiscale Dissociation Inventory, did not improve the prediction of PTSD. This indicates that even in an acutely traumatized sample of adult subjects, childhood trauma accounts for such a significant portion of variance in persistent dissociation that persistent dissociation is rendered insignificant in the prediction of PTSD development. This finding is in line with earlier studies<sup>23,24</sup> demonstrating a direct link between childhood trauma and

the development of persistent dissociation. Childhood trauma was positively correlated with peritraumatic dissociation, but it failed to reach statistical significance. Peritraumatic dissociation, in turn, was positively correlated with symptom severity at all 3 time points as well as with persistent dissociation, including dissociative amnesia. A nonsignificant correlation emerged between the Multiscale Dissociation Inventory subscale identity dissociation measuring symptoms indicative of dissociative identity disorder and peritraumatic dissociation. This may result from a sampling bias, as this disorder is likely underrepresented in an acutely traumatized sample recruited in the emergency department.

### Limitations

First, due to the nature of traumatic events, it is difficult to assess peritraumatic dissociation at the time of occurrence. While some authors have argued that the posttraumatic assessment might lead to a scoring bias, a recent meta-analysis has shown that the predictive value of peritraumatic dissociation assessments did not vary with the assessment date.<sup>42</sup>

Second, we did not include acute stress disorder symptoms in the regression analysis, as it has been shown before that the symptom overlap with peritraumatic dissociation is so substantial that acute stress disorder as the more proximal predictor eliminates the predictive value of peritraumatic dissociation scores.<sup>43</sup> However, it would be most informative to differentiate between the influence of dissociation strictly occurring peritraumatic and dissociation occurring within the first days of posttraumatic adjustment. Future studies should therefore assess dissociation repeatedly within the first 2 weeks post trauma.

Third, this pilot investigation relies on a comparatively small number of patients from 2 centers. This is particularly true for the assessment of symptom severity at 3 months post trauma and the neuroimaging study. The small sample size and associated limited power may have prevented us from detecting additional brain regions associated with peritraumatic dissociation and prevented us from analyzing the predictive value of the fMRI data. Future studies should therefore seek to predict symptom severity on the basis of BOLD signal changes in a larger sample. In addition, we cannot rule out that the use of a convenience sample in the neuroimaging study introduced a systematic bias. Therefore, generalizations can only be accepted with caution.

### CONCLUSION

The results of the regression analysis indicate that both childhood trauma and peritraumatic dissociation are valuable predictors of future PTSD symptom development and therefore can guide the identification of individuals at risk. However, peritraumatic dissociation is a more proximal variable and carried greater predictive power of chronic PTSD in the current sample. Further evidence for the latter stems from the neuroimaging data that link peritraumatic dissociation to intrusive memory recall and strongly support the model proposed by Conway<sup>8</sup>; in this model, intrusions represent vivid

memory recall from an episodic memory system in the posterior temporal-occipital regions storing the sensory aspects of the traumatic event.

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