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Peritraumatic dissociation and posttraumatic stress after pregnancy loss: a prospective study

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Abstract

This study examined (1) predictors for peritraumatic dissociation, (2) its relations with acute and chronic symptoms of posttraumatic stress disorder (PTSD), and (3) pathways regarding these relations in response to pregnancy loss. In early pregnancy, about 1370 women volunteers completed questionnaires for neuroticism, control over emotions, dissociative tendencies, absorption, and prior life events. Of these, 126 subsequently experienced pregnancy loss and most of them completed measures 1 month ($N = 118$) and 4 months ($N = 104$) later. At 1 month, peritraumatic dissociation, memory of pregnancy loss (degree of fragmentation, sensory impressions, and emotional intensity), thought suppression, and PTSD symptoms were assessed, and at 4 months, PTSD symptoms were re-assessed. Peritraumatic dissociation was predicted by prior low control over emotions, dissociative tendencies, and lower education. It was not predicted by neuroticism, absorption, and prior life events. Peritraumatic dissociation was related to acute PTSD symptoms and LISREL analyses indicated that self-reported memory fragmentation and thought suppression of pregnancy loss mediated this relation. It also predicted chronic PTSD symptoms, and this relation was mediated by acute PTSD symptoms.

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Peritraumatic dissociation involves acute alterations in cognitive and perceptual functioning at the time of a traumatic event. It includes depersonalization, derealization, narrowing of attention, time distortion, and confusion (Marmar, Weiss, & Metzler, 1997; Spiegel & Cardeña, 1991).

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Longitudinal studies strongly linked peritraumatic dissociation to posttraumatic stress disorder (PTSD) after various traumatic events (e.g., Ehlers, Mayou, & Bryant, 1998; Koopman, Classen, & Spiegel, 1994; Shalev, Peri, Canetti, & Schreiber, 1996; Ursano et al., 1999). This was independent of objective markers of trauma severity, initial (<1 week) PTSD symptoms (Shalev et al., 1996), and prior PTSD (Ursano et al., 1999).

In fact, peritraumatic dissociation is considered one of the most important predictors for PTSD (Marmar et al., 1997). The question is what are its determinants? Peritraumatic dissociation relates to peritraumatic perceived threat (Griffin, Resick, & Mechanic, 1997; Marmar, Weiss, Metzler, & Delucchi, 1996), and dissociative reactions are related to hyperarousal (Rainey et al., 1987; Southwick et al., 1993). However, research of its *premorbid* psychological determinants is scant. This is partly because prospective trauma studies are difficult to do. Generally, potential premorbid factors are assessed after trauma, although effects of the event or memory distortions might contaminate such reports. Nevertheless, cross-sectional studies identified potential determinants. Fullerton and colleagues (2000) found that prior depression was related to peritraumatic dissociation in response to a motor vehicle accident (MVA). Marmar and colleagues (1996) assessed emergency workers after critical incident exposure and found that traits similar to neuroticism related to peritraumatic dissociation. This suggests that *neuroticism*, which predicts PTSD symptoms (McFarlane, 1989, but see also Engelhard, van den Hout, & Kindt, submitted), increases risk for peritraumatic dissociation. Marmar and colleagues (1996) also found external locus of control to be a strong correlate. Low *control over emotions* is involved in PTSD (Jones & Barlow, 1990), and might lead to more disorganizing effects of panic during threat. Moreover, *general dissociative tendencies* and ‘*absorption*’, that is, openness to self-altering experiences (see Kihlstrom, Glisky, & Angiulo, 1994) bear a close phenomenological resemblance to peritraumatic dissociation (absorption correlates with hypnotizability; see Roch & McConkey, 1990). These tendencies relate to PTSD (Spiegel, Hunt, & Dondershine, 1988; Stutman & Bliss, 1985) and may enhance spontaneous dissociation during trauma. It has also been suggested that *prior traumatic events*, particularly before adulthood, lower the threshold for acute dissociation (van der Kolk, van der Hart, & Marmar, 1996). Prior trauma predicts PTSD (Breslau, Chilcoat, Kessler, & Davis, 1999), but recently, Fullerton et al. (2000) found it to be unrelated to peritraumatic dissociation. Finally, *lower intelligence* strongly predicts PTSD, beyond objective trauma exposure (Macklin et al., 1998). Fewer cognitive resources might render individuals more overwhelmed during threat.

Furthermore, little is understood about the processes by which peritraumatic dissociation results in PTSD. For a better understanding, it might be useful to distinguish processes leading to acute and chronic (>3 months; APA, 1994) PTSD. To begin with, regarding the onset of PTSD, peritraumatic dissociation might explain the nature of trauma representations that are later involuntary re-experienced (van der Kolk & Fisler, 1995). Traumatic memories related to PTSD appear different in structure and content from traumatic memories unrelated to PTSD. Intentional recall of the former is fragmented and disorganized, rather than whole and coherent (Foa, Molnar, & Cashman, 1995; Harvey & Bryant, 1999). For example, trauma-narratives reported by individuals with acute stress disorder (ASD) involve more disjointed, confused, and repetitive elements than those reported by individuals without ASD (Harvey & Bryant, 1999), and decreased fragmentation is related to reduced PTSD symptoms (Foa et al., 1995). Moreover, sensory and emotional impressions rather than thoughts typically characterize re-experiencing phenomena (Ehlers & Steil, 1995; Ehlers & Clark, 2000). Dissociation during trauma may be responsible for incoherence

of events at the time of encoding, resulting in *memory fragmentation* at the time of recall, and may overlap with the processing of *sensory and emotional impressions* of the event rather than its meaning.

Peritraumatic dissociation might also predict acute PTSD by being an early marker for disengagement with the traumatic event. In the aftermath of a traumatic event, oscillation occurs between intrusions and cognitive avoidance (Horowitz, Wilner, Kaltreider, & Alvarez, 1980). The more memories evoke emotions, the more the individual attempts to suppress them. However, for individuals who develop PTSD, avoidance increases with time (Shalev et al., 1996). There is evidence that peritraumatic dissociation strongly relates to avoidance (Griffin et al., 1997) and escape coping (Marmar et al., 1996). Moreover, it relates to less physiological reactivity during re-exposure to trauma-related material (Griffin et al., 1997). Possibly, individuals with high dissociation during trauma are more likely to use *avoidance strategies* to deal with traumatic memories. The tendency to suppress thoughts is particularly relevant as it paradoxically increases their recurrence (e.g., Lavy & van den Hout, 1990).

Regarding the maintenance of PTSD, studies showed that the longer PTSD persists, the less trauma exposure explains symptoms (McFarlane & Yehuda, 1996). Once symptoms have emerged, the way the individual interprets them appears more critical in determining their course (Ehlers et al., 1998; Steil & Ehlers, 2000). For example, Ehlers et al. (1998) found that peritraumatic dissociation and negative interpretations of intrusions at three months predicted PTSD symptoms at one year. After controlling for PTSD symptoms at three months, peritraumatic dissociation was no longer significant, whereas negative interpretations remained significant. This suggests that the relation between peritraumatic dissociation and chronic PTSD is mediated by acute PTSD symptoms.

In sum, peritraumatic dissociation appears related to neuroticism, emotional control, general dissociative tendencies, absorption, prior trauma, and lower intelligence. Note that these may not be independently associated. In order to elucidate their relative importance and degree of interdependence, the first goal of this study was to measure all factors in a prospective study. We also studied the magnitude of peritraumatic dissociation and these factors in predicting acute PTSD symptoms. Second, we investigated whether traumatic memory characteristics (fragmentation, sensory impressions and emotional intensity) and thought suppression mediate the relation between peritraumatic dissociation and acute PTSD symptoms. Finally, we examined whether acute PTSD symptoms mediate the relation between peritraumatic dissociation and chronic PTSD symptoms. Earlier, we reported a prospective longitudinal study of PTSD symptoms after pregnancy loss (Engelhard, van den Hout, & Arntz, 2001; Engelhard et al., submitted), which provided us with a way to measure premorbid factors before pregnancy loss. The results presented here are part of that study. Participants were sent questionnaires by mail. In early pregnancy, they completed measures for neuroticism, expected emotional control in case of pregnancy loss, general dissociative tendencies, absorption, and prior negative events. Highest level of education attained was used as a proxy measure of intelligence. There are no Dutch measures of intelligence to be administered by mail and the two are highly related in the Netherlands. For example, Bögels, Notermans, Arntz, and van den Hout (in preparation) found a correlation of $r = 0.62$ between highest level of education and gender and age-controlled IQ. One month after pregnancy loss, participants completed measures for peritraumatic dissociation, memory (degree of fragmentation, sensory impressions, and emotional intensity), thought suppression, and PTSD symptoms. At four months, PTSD symptoms were re-assessed.

1. Method

1.1. Participants

As described in detail elsewhere (Engelhard et al., 2001), about 1370 pregnant women responded to ads in Dutch family magazines recruiting participants (≤ 12 weeks pregnant) for study of pregnancy experiences. They completed baseline questionnaires at about 8 weeks of pregnancy. For every 2 months thereafter until 1 month after the due-date of birth, they completed pregnancy-related surveys that also asked them to inform the researchers if they had a pregnancy loss. A total of 118¹ out of 126 women (9%) who had a pregnancy loss completed questionnaires about 1 month later (response of 94%). Respondents were 31 years ($SD = 4$), nearly all were married or cohabited, about 40% were college-educated, and about 30% were childless. The mean age of the loss was 12 weeks ($SD = 6$; range 5–40), and 95% were before the 20th week. A total of 104 women (88%) also completed measures at 4 months. Relative to completers, the 14 drop-outs reported more peritraumatic dissociation, $M = 21.2$ vs $M = 26.0$, $F(1, 116) = 5.1$, $p = 0.03$, and acute PTSD symptoms, $M = 17.3$ vs $M = 24.4$, $F(1, 116) = 7.9$, $p < 0.01$. They did not differ in age and pregnancy length.

1.2. Measures

Neuroticism was assessed with the widely used Eysenck Personality Questionnaire (EPQ-N; Eysenck & Eysenck, 1975).

Perceived control over emotional reactions was measured with the following item: “If my pregnancy does not turn out well, I will definitely be able to completely control my emotions”, rated on a five-point scale (1=absolutely not, 5=absolutely).

General dissociative tendencies were measured with an adapted form of the Peritraumatic Dissociative Experiences Questionnaire (PDEQ; see below) in which all items were rewritten to general terms. Participants rated to which extent each item characterized their experiences (e.g., “I find myself acting on ‘automatic pilot’”, “I have moments of losing track of what is going on”, “What happens to me seems unreal to me, like I am in a dream or watching a movie or play”, using a five-point scale (1=not at all, 5=extremely). Cronbach’s alpha was 0.75.

Absorption was measured with the 34-item Tellegen Absorption Scale (TAS; Tellegen & Atkinson, 1974). Items are scored on a true or false format (e.g., “My thoughts often occur as images rather than words”), and ‘true’ responses are summed. Prior research supports the reliability and validity of the TAS (Roche & McConkey, 1990).

Negative life events were assessed with a scale of 21 discrete events (e.g., verbal, physical, and sexual abuse, serious accidents, and disaster), and an open-ended question for events not included (Morgan & Janoff-Bulman, 1994). For each negative event participants had experienced, they reported how many times it occurred and their age at the time. Negative lifetime events and events before adulthood (< 18 years) were calculated.

¹ Five women were not included in our previous report as they enrolled at a late stage and experienced a late loss.

Highest level of education attained was assessed with a seven-point scale (1=elementary school, 7=graduate school).

Dissociation at the time of pregnancy loss was measured with the 10-item PDEQ (Marmar et al., 1997). Each item (e.g., “I had moments of losing track of what was going on”) is rated on a five-point scale (1=not at all true, 5=extremely true) and ratings are summed to generate severity of peritraumatic dissociation. A matching rater-administered version of this scale has established internal consistency and validity (Marmar et al., 1997).

Memory of pregnancy loss was rated for fragmentation (“How much does your memory of the event exist of fragmented pieces as opposed to a whole entity?”), sensory impressions (i.e., visual images, sounds, smells, taste, and bodily sensations; e.g., “How much does your memory of the event exist of visual images?”), and emotional intensity (“How intense are emotions in your memory of the event?”). Items were rated on a seven-point scale (1=not at all, 7=extremely), and sensory-impressions were summed.

Thought suppression was measured with four items of the White Bear Suppression Inventory (WBSI; Wegner & Zanakos, 1994) that measure individual differences in the tendency to suppress unwanted thoughts (see Muris, Merckelbach, & Horselenberg, 1996). These were rated for the suppression of pregnancy-related thoughts, using a five-point scale (1=strongly disagree, 5=strongly agree). Cronbach’s alpha for these items was 0.73.

PTSD symptom severity was assessed with the PTSD Symptom Scale (PSS; Foa, Riggs, Dancu, & Rothbaum, 1993), which comprises the 17 DSM-IV (APA, 1994) symptoms for PTSD. Items are rated on a four-point severity scale (0=not at all, 3=almost always). The PSS has established reliability and validity (Foa et al., 1993).

1.3. Data analysis

SPSS 10.0.5 estimated the few missing values by regression with residual estimation adjustment. Pregnancy length and life events were skewed to the left and log-transformed for analyses. Pearson correlations were computed between the variables and multiple regression tested the relative contribution of predictors. The PRELIS 2.30 program computed polychoric, polyserial, and product moment correlations, and LISREL 8.0 evaluated the degree to which hypothesized pathways were consistent with the data, with the weighted least squares method. Several model fit criteria were used (Schumacker & Lomax, 1996). The *Goodness-of-Fit Index* (GFI) and *Non-Normed Fit Index* (NNFI) do not explicitly depend on sample size and values above 0.90 represent a good fit. The *Root-Mean-Square Residual Error of Approximation* (RMSEA) considers the error of approximation in the population and precision of the fit measure itself. A value up to 0.08 reflects an acceptable error. A χ^2 -difference test compared hypothesized pathways with an alternative and indicates whether the improvement in fit for an additional relationship is sufficient to warrant the cost of one degree of freedom. The significance level was 5%.

2. Results

Table 1 displays means, standard deviations, and intercorrelations of the variables. Most participants (70%) reported at least one item on the PDEQ (rated ≥ 4 , “intense”). The most common

Table 1
Psychometric data and intercorrelations of variables at baseline ($N = 118$), 1 month ($N = 118$) and 4 months ($N = 104$) post-loss^a

	<i>M</i> (<i>SD</i>)	1	2	3	4	5	6	7	8	9	10	11	12
<i>Baseline</i>													
1. EPQ-N	7.3 (4.6)												
2. Control emotions	2.0 (1.0)	0.00											
3. PDEQ-modified	13.8 (4.4)	0.23*	0.00										
4. TAS	13.3 (7.8)	-0.09	-0.01	0.41*									
5. Prior negative events	1.5 (1.1)	0.03	0.03	0.11	0.20*								
6. Education	4.8 (1.5)	0.06	0.07	0.08	0.11	-0.22*							
<i>One month post-loss</i>													
7. PDEQ	21.8 (7.6)	0.06	-0.20*	0.26*	0.12	0.10	-0.27*						
8. Fragmented memory	3.6 (2.1)	0.11	-0.08	0.10	-0.01	0.07	-0.08	0.19*					
9. Sensory impressions	4.9 (1.6)	-0.02	0.04	0.12	0.08	0.35*	-0.27*	0.29*	0.08				
10. Emotional intensity	4.4 (1.7)	-0.01	-0.28*	-0.06	0.15	0.10	-0.03	0.34*	-0.05	0.14			
11. WBSI	8.2 (3.3)	0.05	-0.08	0.25*	0.19*	0.10	-0.15	0.32*	0.20*	0.27*	0.42*		
12. PSS-SR	18.1 (9.1)	0.25*	-0.24*	0.31*	0.23*	0.23*	-0.15	0.46*	0.32*	0.30*	0.50*	0.56*	
<i>Four months post-loss</i>													
13. PSS-SR	9.5 (8.5)	0.26*	-0.10	0.30*	0.22*	0.14	0.02	0.29*	0.27*	0.15	0.34*	0.34*	0.62*

^a Possible ranges of scores were EPQ-N, 0–22; Control emotions, 1–5; PDEQ (modified), 10–50; TAS, 0–34; Education, 1–7; Fragmented memory, sensory impressions, emotional intensity, 1–7; WBSI, 4–20; PSS-SR, 0–51. * $p < 0.05$.

were being on automatic pilot (27%), distorted body (23%), feeling as though watching a movie or play (23%), time change (18%), and losing track of what was going on (17%). Residual symptoms were endorsed by 6–15%. First, we studied predictors of peritraumatic dissociation. Negative lifetime events and events before adulthood were strongly related [$r(118) = 0.73, p < 0.001$] and we only report negative events before adulthood. Neuroticism, absorption, and prior negative events did not predict peritraumatic dissociation. In contrast, expected emotional control ($\beta = -0.17, t = 2.03, p = 0.045$), general dissociative tendencies ($\beta = 0.27, t = 3.21, p = 0.002$), and education ($\beta = -0.28, t = 3.23, p = 0.002$) independently predicted peritraumatic dissociation, accounting for 18% of its variance, $F(3, 114) = 8.17, p < 0.001$. Next, these predictors were studied in relation to acute PTSD symptoms. Neuroticism ($\beta = 0.22, t = 2.54, p = 0.013$), emotional control ($\beta = -0.24, t = 2.96, p = 0.004$), general dissociative tendencies ($\beta = 0.18, t = 1.94, p = 0.054$), and prior negative events ($\beta = 0.19, t = 2.33, p = 0.022$) accounted for 23% of acute PTSD symptoms, $F(4, 113) = 8.54, p < 0.001^2$. Absorption ($\beta = 0.14, t = 1.55, p = 0.124$) was not significant. Then peritraumatic dissociation was included. Peritraumatic dissociation ($\beta = 0.36, t = 4.48, p < 0.001$), neuroticism ($\beta = 0.22, t = 2.75, p = 0.007$), emotional control ($\beta = -0.17, t = 2.23, p = 0.028$), and negative events ($\beta = 0.16, t = 2.13, p = 0.035$) accounted for 33% of acute PTSD symptoms, $F(4, 113) = 13.67, p < 0.001$. General dissociative tendencies ($\beta = 0.10, t = 1.08, p = 0.282$) and absorption ($\beta = 0.14, t = 1.64, p = 0.103$) were not significant. Finally, the pathways linking peritraumatic dissociation to PTSD symptoms at 1 month [$r(118) = 0.46, p < 0.001$] and 4 months [$r(104) = 0.29, p = 0.003$] were investigated (see Fig. 1 for β weights). To begin with, peritraumatic dissociation was allowed to have an indirect relationship with acute PTSD symptoms through memory fragmentation, sensory impressions, emotional intensity, and thought suppression of pregnancy loss. This model fit well to the data, GFI = 0.97, NNFI = 0.97, RMSEA = 0.04, and explained 46% of the variance of acute PTSD symptoms. However, sensory impressions and emotional intensity were not significantly linked to PTSD, and emotional intensity appeared related to PTSD through thought suppression. Then, a direct link between peritraumatic dissociation and acute PTSD symptoms was added to the model. This direct link ($\beta = 0.19, t = 0.64$) and the difference between the models with and without this link were not statistically significant [$\Delta\chi^2(1) = 0.40, p = 0.53$]. We concluded that the relationship between peritraumatic dissociation and acute PTSD symptoms was mainly mediated by fragmented memory and thought suppression of pregnancy loss. Next, we allowed the relationship between peritraumatic dissociation and chronic PTSD symptoms to be mediated by acute symptoms, GFI = 0.97, NNFI = 1.01, RMSEA = 0.0, which fit well to the data, and explained 37% of chronic PTSD symptoms. Then, we also included a direct link between peritraumatic dissociation and chronic symptoms in the model. This direct link ($\beta = 0.07, p = 0.82$) and the difference between the models with and without this link [$\Delta\chi^2(1) = 0.77, p = 0.38$] were not statistically significant. We concluded that the relationship between peritraumatic dissociation and chronic PTSD symptoms was mediated by acute symptoms. Finally, we included a relationship between acute and chronic PTSD symptoms in the model for acute PTSD, which

² Pregnancy length was significantly related to acute PTSD symptoms ($r = 0.31, p = 0.001$), but not to peritraumatic dissociation ($r = 0.08, p = 0.37$)

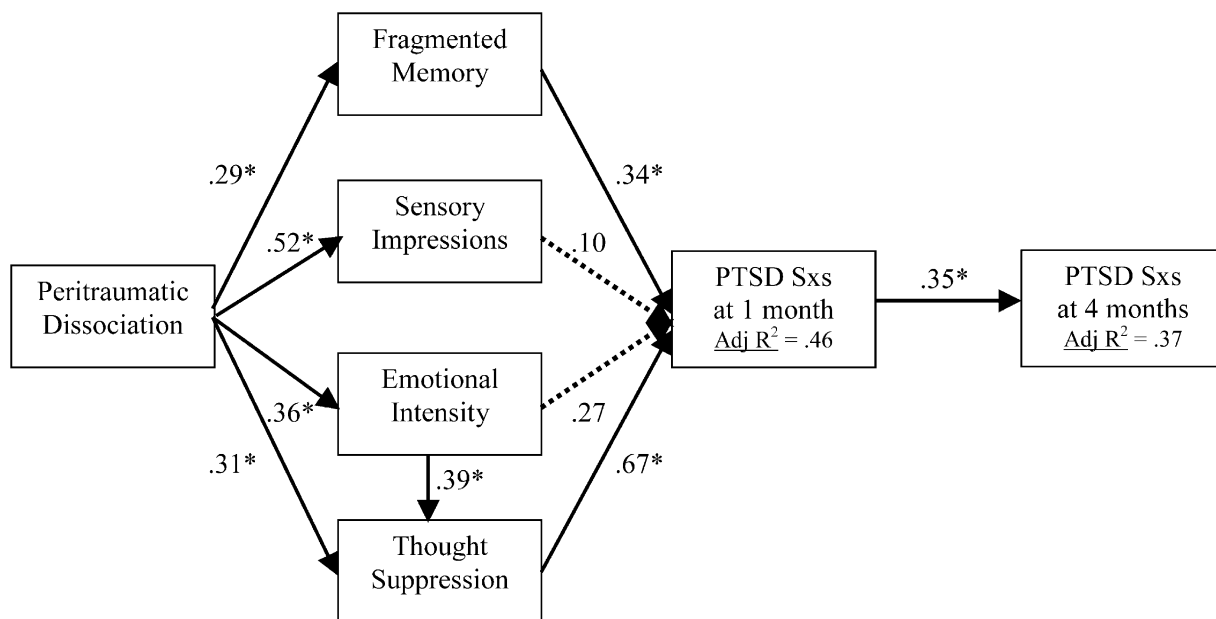


Fig. 1. Path analyses and beta weights of factors linking peritraumatic dissociation and PTSD symptoms 1 and 4 months after pregnancy loss.

fit well, GFI = 0.98, NNFI = 1.09, RMSEA = 0.0. The mediators of acute PTSD symptoms were each allowed to directly relate to chronic PTSD symptoms, but none of these links (largest $\beta = 0.2$, largest $t = 0.67$) were significant.

3. Discussion

The results of this study can be summarized as follows. Peritraumatic dissociation was commonly reported (70%) after pregnancy loss, which was comparable to studies of MVA (79%; Ursano et al., 1999) and civilian injury (50%; Shalev et al., 1996). It was strongly related to acute and chronic PTSD symptoms, which is consistent with other longitudinal studies. Its determinants were less control over emotions, dissociative tendencies, and lower education. Peritraumatic dissociation related to acute PTSD symptoms even after controlling for premorbid neuroticism, emotional control, general dissociative tendencies, absorption, and negative events. This relation was largely mediated by memory fragmentation and thought suppression, and the relation between peritraumatic dissociation and chronic PTSD symptoms was mediated by acute symptoms.

Previously, dissociation was associated with physiological hyperarousal (Rainey et al., 1987; Southwick et al., 1993). Although the construct of peritraumatic dissociation is poorly defined, taken together, the cluster of symptoms might be conceived of as a marker for the individual's inability to tolerate acute and later arousal and intrusions. The highest level of education negatively predicted peritraumatic dissociation but did not predict PTSD. Shalev et al. (1996) found the opposite for total years of education. It is unclear what is responsible for this. Highest level of education still might be too crude a proxy measure of intelligence, since lower intelligence

was previously found to be a strong predictor of PTSD in Vietnam veterans (Macklin et al., 1998). Peritraumatic dissociation was not predicted by neuroticism, which was inconsistent with cross-sectional data of Marmar et al. (1996), and it was not predicted by absorption. In contrast, both factors predicted PTSD symptoms. The nature of the link between neuroticism and PTSD is unclear (Engelhard et al., submitted). To our knowledge, this is the first prospective study linking absorption to PTSD. Although imagery is thought to play an important role in PTSD (Brett & Ostroff, 1985), absorption did not predict PTSD beyond the contribution of general dissociative tendencies. Finally, prior life events did not predict peritraumatic dissociation, which was in line with recent findings of Fullerton et al. (2000), but did directly predict acute PTSD symptoms.

Peritraumatic dissociation related to acute PTSD symptoms through fragmentation and suppression of memories of pregnancy loss. The data are silent about causality, given the cross-sectional nature of these relations. However, theory suggests that fragmented memories impair the organization of memories and their connection with existing schemas, and excessive avoidance is thought to impede this integration further (Foa & Riggs, 1993). Prior studies showed that suppression of unwanted thoughts increases the frequency of such thoughts (e.g., Lavy & van den Hout, 1990). Moreover, after instructed suppression of trauma-related thoughts, individuals with PTSD, relative to non-PTSD traumatized controls, reported a drastic increase in trauma-related thoughts (Shipherd & Beck, 1999). Possibly, the inability to suppress trauma-related thoughts predisposes to persistent symptoms. Self-reported sensory impressions were unrelated to PTSD symptoms. This may be attributed to the nature of memory measurement. That is, we assessed evaluation of memory (“meta”-memory) rather than memory content. Perceptual memories relevant to PTSD might not be deliberately accessed (Brewin, 2001). Future studies with more comprehensive measures might clarify this issue further. Sensory impressions might also have been measured too early. Most individuals report sensory impressions and intrusive recollections immediately after trauma and initial intrusions are not a good predictor of PTSD (Ehlers & Steil, 1995). The significance of sensory impressions might be different at a later stage. Dissociation at the time of pregnancy loss did not predict chronic PTSD symptoms beyond acute PTSD symptoms. Holen (1993) also found that peritraumatic dissociation was more important in predicting short term rather than long term adjustment. As noted by Marmar et al. (1997), reports of peritraumatic dissociation months or years later might be confounded by current distress, resulting in stronger relationships with PTSD.

The results are in line with cognitive models of PTSD (e.g., Foa & Riggs, 1993; Ehlers & Clark, 2000) that underscore the importance of elaboration of fragmented trauma memory into coherence and alteration of avoidance strategies. However, many questions remain. For example, how can be assessed when integration of the trauma memory has occurred? When are avoidance strategies dysfunctional, and what factors determine them? Finally, several limitations of this study need to be taken into account. First, the LISREL analyses were partly cross-sectional and do not elucidate causality. Future longitudinal studies ought to use shorter assessment intervals to clarify causality. Second, there were several imperfections in measures. Although showing good internal consistency, general dissociative tendencies were measured with an unvalidated modified PDEQ and thought suppression was measured with only part of the WBSI. Moreover, memory measures were not validated. Third, pregnancy loss is generally a brief event and it is unclear how the results relate to more severe or prolonged events. Risk factors for PTSD at low

exposure levels may lose significance at high exposure, although the dose–response model generally does not withstand scrutiny (Bowman, 1999). Strengths of the study were the prospective assessment of predictors and rather homogeneous traumatic event. However, further study is needed to establish causal pathways between peritraumatic dissociation and acute PTSD symptoms.

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