Peritraumatic Psychological and Somatoform Dissociation in Predicting PTSD Symptoms

A Prospective Study

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Abstract: The present prospective study evaluates the predictive value of 2 different types of dissociation (psychological and somatoform peritraumatic dissociation), and dysfunctional cognitions on symptoms of posttraumatic stress disorder (PTSD) at 6 months. Assessment of dissociation, PTSD symptoms, and dysfunctional cognitions took place in 49 participants approximately 3 weeks after a traumatic event. Six months later PTSD symptoms were assessed again. The effect of both psychological and somatoform peritraumatic dissociation disappeared after controlling for initial PTSD numbing symptoms. Dysfunctional cognitions predicted PTSD at 6 months after controlling for initial numbing symptoms. The present study indicates that peritraumatic dissociation may not be a predictor of PTSD. In contrast, maladaptive posttraumatic coping behavior like persistent dissociation and dysfunctional cognitions may be predictors.

Key Words: Peritraumatic dissociation, posttraumatic stress disorder, cognitions, numbing.

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People’s reactions during the time of a traumatic event have been studied intensively in the last decade, suggesting an important predictive role of peritraumatic variables in the development of posttraumatic stress disorder (PTSD). In a meta-analysis, peritraumatic dissociation was strongly related to later PTSD (Ozer et al., 2003). Theoretically, peritraumatic dissociation is thought to interfere with adequate processing of trauma information leading to poorly integrated representations of the trauma in autobiographical memory, which subsequently leads to PTSD intrusions and flashbacks (Brewin et al., 1996; Ehlers and Clark, 2000).

However, many of the studies in the meta-analysis were retrospective or noncontrolled. An increasing body of literature is now questioning the usefulness of peritraumatic dissociation in the prediction of PTSD. Indeed, some recent controlled studies found the relationship between peritraumatic dissociation and PTSD disappeared after controlling for initial PTSD symptoms (e.g., Marshall and Schell, 2002). This may be the result of the overlap between dissociative and PTSD symptoms, more specifically numbing symptoms. Moreover, several studies successfully induced peritraumatic dissociation in an experimental context but failed to find a relationship with trauma-related intrusions (Hagenaars, Van Minnen, Holmes, Brewin, and Hoogduin, In press; Holmes et al., 2004; Holmes, Oakley, Stuart, and Brewin, 2006).

Some studies have shown that not peritraumatic dissociation but persistent dissociation predicts PTSD development (Briere et al., 2004; Murray et al., 2002). Unlike peritraumatic dissociation, an automatic reaction evoked by threat, persistent dissociation indicates a dysfunctional coping strategy. This is in line with the cognitive model of PTSD (Ehlers and Clark, 2000), which states that it is not the traumatic event itself, but one’s coping with the event and its sequelae that predicts PTSD. The cognitive model also posits that dysfunctional cognitions about the trauma or one’s own reactions during or after the trauma predict the development of PTSD symptoms. Persistent dissociation is seen as a maladaptive coping behavior that prevents the person from changing negative appraisals of the trauma, therefore maintaining PTSD. Indeed, some studies found that posttrauma dysfunctional cognitions and dysfunctional strategies like avoidance or numbing were related to the onset and maintenance of PTSD (Dunmore et al., 1999).

In contrast with the large number of studies on peritraumatic psychological dissociation, mostly using the Peritraumatic Dissociative Experiences Questionnaire (PDEQ; Marmar et al., 1997), peritraumatic somatoform dissociation has been underreported. However, this somatoform reaction to threat seems rather common in both animals and humans (Moskowitz, 2004). For example, it was found that two-thirds of the rape victims froze and were unable to move during the assault (Galliano et al., 1993). This peritraumatic tonic immobility was related to an increased passive attitude after the assault. In a recent review, it was argued that 2 qualitatively
distinct forms of dissociation can be distinguished, namely detachment and compartmentalization (Holmes et al., 2005). PDEQ items typically refer to detachment. Somatoform dissociation on the other hand typically refers to compartmentalization. In this respect, it would be interesting to study whether these 2 types of dissociation have different influences on PTSD development.

In conclusion, it is important to gain more insight in the impact of distinct forms of peritraumatic dissociation and confounding factors on PTSD development. The present study is set up to investigate prospectively the relationship between 2 types of dissociation—psychological and somatoform peritraumatic dissociation—and dysfunctional cognitions on PTSD symptoms 6 months posttrauma. Because of the substantial overlap between dissociative and numbing symptoms, and because numbing concerned persistent (and not peritraumatic) symptoms, we controlled for initial numbing symptoms.

METHODS

Forty-nine participants were recruited from 3 areas where a disaster had taken place in the home environment. In the first area (27 participants), the balconies of 5 apartments came down with a lot of noise and 2 people died under the wreckage. In the second area (8 participants) a gas explosion took place. Three houses were destroyed, but no one was injured. In the third area (14 participants) a truck drove into a supermarket, destroying the supermarket and the 2 houses next to it, and 3 people died. All participants had witnessed or were involved in the trauma. Participants were contacted if they were potential witnesses or potentially involved, that is, if their residence was close to the place where the disaster took place. In addition, individuals who were in the supermarket (third area) were contacted via the owner of the supermarket. After receiving a written description of the study, written informed consent was obtained for all participants. Three participants (all from the first area) withdrew their participation after the procedure was explained, leaving a total of 46 participants (25 men and 21 women, mean age 51.5 years, SD = 15.3), who completed the first assessment. The first assessment was approximately 20 days (SD = 5.9 days) after the trauma. Thirty-two participants completed the second assessment at 6 months posttrauma.

At the first assessment 2 types of dissociation were measured. Peritraumatic psychological dissociation was measured by the Peritraumatic Dissociation Experiences Questionnaire—10-Self Report Version, which is a reliable and valid instrument (Marmar et al., 1997). The Somatoform Dissociation Questionnaire—Peritraumatic (SDQ-P; Nijenhuis et al., 2001) was used to measure peritraumatic somatoform dissociation. Dysfunctional trauma-related cognitions were measured by the Posttraumatic Cognition Inventory (PTCI), which has good-to-excellent psychometric properties (Foa et al., 1999).

Both at the first assessment and second assessment, the Posttraumatic Symptoms Scale—Self Rating (PSS-SR; Foa et al., 1993) was used to assess PTSD symptoms as defined by DSM-IV. Psychometric properties of the total scale and its subscales are good (Foa et al., 1993). As a dissociation-related construct, numbing was defined using the 3 numbing items of the PSS-SR avoidance subscale (e.g., Litz, 1992) that refer to diminished interest, detachment or estrangement, and restricted range of affect. Note that the assessment of numbing concerned persistent symptoms (e.g., symptoms in the week before the assessment), whereas assessments of psychological and somatoform dissociation concerned symptoms at the time of the trauma.

Regression analyses were used to predict PTSD symptoms at 6 months. In all analyses, the criterion for significance was 0.05.

RESULTS

First, the effect of psychological and somatoform peritraumatic dissociation and dysfunctional cognitions on PTSD symptoms at 6 months was studied. To establish the effect of these predictors without controlling for initial numbing symptoms, a stepwise regression was conducted with PSS-SR at 6 months as the dependent variable and peritraumatic psychological and somatoform dissociation and posttraumatic dysfunctional cognitions as predictors (measured by PDEQ, SDQ, and PTCI respectively). Peritraumatic psychological and somatoform dissociation and posttraumatic dysfunctional cognitions all uniquely predicted PTSD at 6 months (ΔR² = 0.42, p < 0.001, ΔR² = 0.11, p = 0.01, and ΔR² = 0.10, p = 0.01, respectively).

However, these results changed after controlling for initial PTSD numbing symptoms. For this purpose, initial PTSD numbing symptoms were entered in the first block of a hierarchical regression (R² = 0.57, β = 0.68, p < 0.001). Peritraumatic psychological and somatoform dissociation and posttraumatic dysfunctional cognitions (PDEQ, SDQ, and PTCI respectively) were entered and analyzed stepwise in the second block. Besides numbing symptoms that continued to be significant (β = 0.51, p = 0.001), only posttraumatic dysfunctional cognitions contributed to the prediction (ΔR² = 0.13, β = 0.40, p = 0.006). The 2 peritraumatic dissociation measures did not add to the prediction of later PTSD symptoms above initial numbing symptoms and dysfunctional cognitions.

DISCUSSION

The present study investigated the influence of 2 forms of dissociation on PTSD symptom development. It was found that both psychological and somatoform peritraumatic dissociation failed to predict PTSD symptoms after controlling for numbing symptoms. Although peritraumatic (psychological) dissociation was strongly related to PTSD development in many studies (Ozer et al., 2003), recent controlled studies also failed to find this association (Briere et al., 2004; Marshall and Schell, 2002). In line with the cognitive model of PTSD (Ehlers and Clark, 2000), dysfunctional cognitions did predict later PTSD symptoms on top of initial numbing symptoms. Our findings are in line with other prospective studies, suggesting that sustained posttraumatic and not peritraumatic reactions are associated with PTSD development (Dunmore et al., 1999).
The results have implications for theories on PTSD development, especially with regard to the uniqueness of trauma memory. It is often thought that peritraumatic dissociation, being a disruption of usually integrated processes of consciousness and restricting verbal encoding, leads to inadequate encoding of trauma information. This leads to information being stored as sensory, visuospatial fragments without temporal context, i.e., intrusions (Brewin et al., 1996). The absence of a relationship between peritraumatic dissociation and later PTSD symptoms may indicate that this mechanism of inadequate encoding during the trauma is not relevant with respect to PTSD development. Maybe this is the case because inadequate encoding can later be replaced by adequate information processing. It is possible that PTSD is developed if inadequate encoding is not followed by adequate information processing leading to sustained inadequate information processing. Indeed, the association between persistent dissociation and PTSD development suggests that the absence of repeated processing of trauma information post-trauma plays a role in PTSD development. Numbing and avoidance may prevent the traumatic information from being repeatedly and emotionally processed. It is also possible that numbing prevents dysfunctional cognitions to be reevaluated, which leads to the development and maintenance of PTSD (Ehlers and Clark, 2000). Indeed, a post hoc analysis of our data showed that both initial active avoidance ($\beta = 0.32, p = 0.04$) and numbing ($\beta = 0.50, p = 0.002$) predicted later PTSD symptoms ($\Delta R = 0.53, p < 0.001$), whereas reexperiences ($\beta = 0.12, p = 0.44$) and arousal ($\beta = 0.16, p = 0.36$) did not.

The uniqueness of trauma memory and processing of trauma information are complicated and intriguing issues. In this respect, future research focusing on the relationship between (peritraumatic and persistent) dissociation and information processing would be helpful. With respect to future research on dysfunctional cognitions, it would be interesting to control for pretrauma dysfunctional thinking or a concept like neuroticism, which is characterized by dysfunctional thinking. This would clarify if either a general tendency to dysfunctional cognitions that is sustained after the traumatic event, or dysfunctional cognitions that are developed merely as a posttrauma reaction, predict PTSD.

An important limitation of the study concerns the relatively small number of participants. However, the number of tests was in accordance with this small sample. On the other hand, the strength of the study is its prospective design, and the fact that it controlled for numbing symptoms at baseline. In addition, to our knowledge, it is the first to compare different forms of dissociation with respect to the development of PTSD symptoms. Especially somatoform peritraumatic dissociation has not been studied in a prospective design. However, the study merits replication with larger samples and different traumata.

**CONCLUSIONS**

In conclusion, we did not find that psychological or somatoform peritraumatic dissociation predicted PTSD. However, dysfunctional cognitions and numbing (an avoidant coping style) did predict PTSD. Our results suggest that sustained and not peritraumatic dissociation is of relevance. Our reactions during a traumatic event may be as they are; it is the way we cope with them that makes the difference.

**REFERENCES**


