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The impact of dissociation and depression on the efficacy of prolonged exposure treatment for PTSD☆

Muriel A. Hagenaars [a,*], Agnes van Minnen [b], Kees A.L. Hoogduin [c]

[a] Department of Clinical-, Health, and Neuropsychology, PO Box 9555, 2300 RB Leiden, The Netherlands
[b] GGZ Nijmegen, Outpatient Clinic of Anxiety Disorders, Tarweweg 2, 6535 AM Nijmegen, The Netherlands
[c] Department of Clinical Psychology, Radboud University Nijmegen, PO Box 9104, 6500 HE Nijmegen, The Netherlands

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ABSTRACT

This study investigates the impact of dissociative phenomena and depression on the efficacy of prolonged exposure treatment in 71 patients with posttraumatic stress disorder (PTSD). Diagnoses, comorbidity, pretreatment depressive symptoms, PTSD symptom severity, and dissociative phenomena (trait dissociation, numbing, and depersonalization) were assessed at pretreatment using semi-structured interviews and questionnaires. In a pretreatment behavioral exposure test, patients were imaginally exposed to (part of) their trauma memory for 9 min, during which subjective fear was assessed. At posttreatment and 6 months follow-up PTSD, depressive and dissociative symptoms were again assessed in the completers ($n = 60$). Pretreatment levels of dissociative and depressive symptoms were similar in dropouts and completers and none of the dissociative phenomena nor depression predicted improvement. Against expectations, dissociative phenomena and depression were associated with enhanced rather than impeded fear activation during the behavioral exposure test. However, these effects disappeared after controlling for initial PTSD severity. Hence, rather than supporting contraindication, the current results imply that patients presenting with even severe dissociative or depressive symptoms may profit similarly from exposure treatment as do patients with minimal dissociative or depressive symptoms.

Exposure-based treatments have proven to be effective in reducing PTSD symptoms (Foa et al., 1999, 2005; Marks, Lovell, Noshirvana, Livanou, & Thrasher, 1998; Resick, Nishith, Weaver, Astin, & Feuer, 2002). Yet, despite this high efficacy, some patients do not (sufficiently) profit from exposure therapy (Bradley, Green, Russ, Dutra, & Westen, 2005). Both from a theoretical and a clinical point of view, i.e., to improve treatment indication and treatment efficacy, it is important to identify these patients. Thus far, few stable predictors of treatment outcome have been identified (Van Minnen, Arntz, & Keijsers, 2002). Remarkably, although in numerous studies (peritraumatic) dissociation has been associated with PTSD development (for a review see Ozer, Best, Lipsey, & Weiss, 2003), its impact on PTSD treatment has been alluded to (Jaycox & Foa, 1996; Shalev, Bonne, & Eth, 1996), but not systematically studied. The present study therefore investigates the impact of several dissociative phenomena on the efficacy of prolonged exposure treatment. The impact of depression is also studied, as it seems to be related to dissociative phenomena like numbing (Monson, Price, Rodriguez, Ripley, & Warner, 2004).

First, it is important to understand how dissociation and depression may interfere with prolonged exposure treatment. Foa and Kozak (1986) developed the emotional processing theory, a theoretical framework that conceptualizes PTSD pathology and ways to correct this pathology in treatment. In this theory, it is proposed that the traumatic memory can be represented as a fear structure that includes representations of trauma-related stimuli, responses and their meaning (Foa & Kozak, 1986; Foa & Rothbaum, 1998; Foa, Steketee, & Rothbaum, 1989). In PTSD, this fear structure is characterized by a large number of stimulus representations associated with danger and strong response elements. For a treatment to be effective, the pathological elements of the fear structure must be corrected. This can only be achieved if the fear structure is activated and if new information is introduced that is incompatible with the existing information in the fear structure. In addition to the emotional processing theory, cognitive models of PTSD have also emphasized the role of fear activation in effective treatment. For example, Ehlers and Clark (2000) underscore the need to relive

☆The work was done at the Department of Clinical Psychology, Radboud University Nijmegen, PO Box 9104, 6500 HE Nijmegen, The Netherlands.

* Corresponding author. Tel.: +31 71 5273748; fax: +31 71 5274678.
E-mail addresses: mhagenaars@fsw.leidenuniv.nl (M.A. Hagenaars), avanminnen@ggznijmegen.nl (A. van Minnen), c.a.l.hoogduin@hsk.nl (K.A.L. Hoogduin).

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the trauma so elaboration and contextualization of the trauma memory can take place and negative assumptions about recalling the trauma can be tested.

If fear activation is important with respect to successful treatment of PTSD, factors that impede fear activation should have a negative impact on recovery. Some studies on PTSD treatment indeed found that higher (increase in) subjective fear during exposure was related to more improvement (Jaycox, Foa, & Morral, 1998; Van Minnen & Hagenaars, 2002). Also, factors that impede fear activation, like anger (Foa, Riggs, Massie, & Yarczower, 1995; Speckens, Ehlers, Hackmann, & Clark, 2006) and the use of benzodiazepines (Van Minnen et al., 2002), had a negative impact on treatment outcome in PTSD. Several authors have suggested that dissociation might be such an impeding factor as well (Hembree, Marshall, Fitzgibbons, & Foa, 2001; Jaycox & Foa, 1996). For example, emotional numbing (emotional non-responsiveness and non-engagement in activities, feelings or other people) may be associated with difficulties in recognizing, describing and regulating emotions, including fear (Monson et al., 2004). This lack of fear may frustrate adequate fear activation. In accordance, Ehlers and Clark (2000) suggested that depersonalization, derealization and numbing may interfere with recovery by impeding the elaboration of the trauma memory and its integration into the autobiographical memory knowledge base. Some empirical evidence supports this dissociative non-responsiveness. For example, high dissociators, relative to low dissociators, demonstrated suppressed autonomic activity when recounting their trauma (Griffin, Resick, & Mechanic, 1997), and lower resting heart rate (Bryant, Harvey, Guthrie, & Moulds, 2000). Nixon and Bryant (2005) did not find any differences in psychophysiological responses during trauma recounting between these two patient types, though.

In clinical practice, highly dissociative patients are often excluded from exposure treatment. Only a handful of studies have explored its suggested negative impact though. Taylor et al. (2001) found a non-significant trend for partial responders to cognitive behavior therapy (CBT) to have higher levels of pretreatment numbing and depressive symptoms relative to full responders. Moreover, in the partial responders, numbing symptoms had not declined following treatment, whereas the other PTSD symptom clusters had. However, many partial responders also suffered from comorbid major depressive disorder, so their numbing symptoms may have been elevated as a result of the depression. Another indication for numbing hindering effective treatment comes from Ehlers et al. (1998), who found that pretreatment alienation, a concept that resembles numbing symptoms like restricted range of affect and feelings of detachment from others, was related to poorer treatment outcome in PTSD after controlling for initial PTSD symptoms. In contrast, Jaycox et al. (1998) found no differences in trait dissociation between patients that did or did not engage in exposure treatment and did or did not habituate. In addition, Speckens et al. (2006) found that pretreatment dissociation did not affect the reduction of intrusions during CBT.

In the current study depression was investigated as an additional variable because of its relatedness to dissociative constructs like emotional numbing, and because depression may thus similarly hamper adequate fear activation. Although depression did not seem to be related to exposure treatment outcome (Van Minnen et al., 2002), comorbidity (mostly depression) was associated with more CBT sessions (Gillepsie, Duffy, Hackmann, & Clark, 2002). Depressive symptoms (BDI scores) on the other hand, tend to decline after treatment along with PTSD symptoms in prolonged exposure treatment studies (Foa et al., 1999; Foa, Rothbaum, Riggs, & Murdock, 1991; Tarrier et al., 1999). However, considering the substantial overlap between depression and PTSD, these may be PTSD related symptoms, decreasing after successful PTSD treatment.

Dissociation is a complicated construct that includes a variety of symptoms but is often studied as a general construct. Bryant (2007) correctly points out that this does not help to delineate the exact mechanisms that are involved. He suggests that to study dissociation, a deconstruction into more specific factors may be a better approach. Distinct dissociative symptoms may indeed have different effects on, in this case, exposure treatment efficacy. Therefore, in the present study several dissociative phenomena were investigated separately and not combined into one dissociation construct. We chose for depersonalization and emotional numbing because these have been associated with stress, PTSD development or reduced efficacy of PTSD treatment (Ehlers & Clark, 2000; Taylor et al., 2001). A general tendency to dissociate was added because its association with PTSD. Moreover, individuals with high dissociative tendencies are expected to respond to stress (in this case imaginal exposure) with dissociative symptoms (Kihlstrom, Glisky, & Angiulo, 1994), which may impede fear activation and adequate information processing. Besides the theoretical importance to study dissociative phenomena separately, there is also some empirical evidence to support this approach. For example, general dissociative tendency, emotional numbing and depression were found to be distinct constructs with distinct effects on PTSD development (Feeny, Zoellner, Fitzgibbons, & Foa, 2000). The correlation between emotional numbing, anomalous body experience and alienation from surroundings was only moderate in patients suffering from depersonalization disorder, again suggesting distinct symptom domains (Sierra, Baker, Medford, & David, 2005). On the other hand, the moderate correlations also indicate that all constructs mentioned show substantial overlap and are somehow related. Depression seems to be a related construct as well, especially with respect to numbing, which also includes diminished positive affect. Several studies found the correlation between numbing and depression to be higher than the one between active avoidance and depression (e.g., Taylor et al., 2001), confirming a symptom overlap or relationship. However, other studies find that although related, numbing and depression are distinct constructs (Litz et al., 1997).

The present study investigates the impact of dissociation, i.e., trait dissociation, depersonalization, and numbing, and depression on the efficacy of exposure treatment for PTSD. We first examined the course of two dissociative phenomena, depersonalization and numbing, and depressive symptoms during treatment. Subsequently, we analyzed the predictive value of pretreatment dissociative phenomena and depression on improvement. Adhering to Foa and Kozak's (1986) conditions for effective exposure, we hypothesized that treatment would be less effective for patients with elevated levels of dissociation compared to those with lower levels. We additionally studied whether dissociation does indeed impede fear activation, expecting pretreatment elevated levels of dissociation to be associated with less fear during exposure. We applied the same hypotheses with regard to depression in order to stay consistent with the theoretical model.

Method

Participants

Of 95 patients referred for PTSD treatment to an outpatient clinic specialized in the treatment of anxiety disorders 74 met the inclusion criteria as they met the DSM-IV-TR criteria (APA, 2000) for PTSD according to the Mini-International Neuropsychiatric Interview (MINI; Sheehan et al., 1998a) and the Clinician-Administered PTSD Scale (CAPS; Blake et al., 1995). Patients who were suicidal, or involved in ongoing traumatization, those fulfilling the DSM-IV-TR criteria for substance dependence or a psychotic

disorder, and those that did not speak Dutch were excluded. Three patients (4%) refused to participate in the study, resulting in an intent-to-treat (ITT) sample of 71 patients.

Mean age of the ITT sample was 35.75 (*SD* 11.74; range 18–63). Twelve patients (17%) were male, 59 (83%) were female. Educational level was low in 5 (i.e. 6 years; 7%), low-extended in 11 (i.e. 8 years; 16%), medium in 29 (i.e. 10–12 years; 41%), high in 15 (i.e. 12–15 years; 21%), higher than a bachelor degree in 9 (i.e. 16 years or more; 12%), and unknown for 2 patients (2%). Thirty patients (42%) were employed, 20 (28%) employed but currently at home because of their PTSD symptoms, 8 (11%) were unemployed, 11 (16%) either attended university or took care of the housekeeping, and for 2 patients (3%) the occupation was unknown.

The patients had suffered mixed traumas, the index trauma being: sexual assault ($n = 17$, 24%), nonsexual assault ($n = 21$, 30%), both sexual assault and nonsexual violence ($n = 12$, 17%), accidents ($n = 8$, 11%), and miscellaneous (like being trapped in a fire, attacked by dog, and war experiences; $n = 13$, 18%). Thirty-seven patients (52%) had been traumatized repeatedly, and 34 (48%) patients had experienced a single trauma. At the beginning of the treatment, the mean time elapsed since the traumatic event was 10 years and 4 months ($SD = 11.07$ years, range 6 months–52 years). Comorbidity was high with many patients meeting the criteria of more than one comorbid disorder: 26 (37%) were diagnosed with panic disorder with agoraphobia, 13 (18%) with social phobia, 8 (11%) with generalized anxiety disorder, 2 (3%) with obsessive-compulsive disorder, 16 (23%) with a current and another 12 (17%) with a past mood disorder, 4 (6%) with somatoform disorder, and 3 (4%) with an eating disorder. In addition, 12 patients (17%) were diagnosed with cluster C personality disorders, 1 (1%) with paranoid personality disorder, 1 (1%) with borderline personality disorder, and 1 (1%) with personality disorder NOS. In total, 49 patients (69%) had a comorbid axis I or II DSM disorder, and 22 (31%) did not.

Of the 71 ITT patients, 60 (84.5%) completed the treatment and 11 (15.5%) dropped out prematurely. The mean number of sessions for the dropouts was 5.82 ($SD = 2.75$). Dropouts and completers did not differ in age ($t(69) = -.51$, $p = .62$), educational level ($t(69) = .63$, $p = .53$), gender ($\chi^2(1, N = 71) = .57$, $p = .45$), comorbidity ($\chi^2(1, N = 71) = .08$, $p = .77$), multiple or single traumatization ($\chi^2(1, N = 71) = .03$, $p = .86$), or pretreatment PTSD symptoms (PSS-SR: $t(69) = .08$, $p = .94$).

Measures

Diagnostic measures

Mini-International Neuropsychiatric Interview (MINI). The MINI (Sheehan et al., 1998a) is a structured interview using closed-end questions based on DSM-IV and ICD-10 to establish DSM-IV psychiatric diagnoses. Its inter-rater reliability proved to be good (kappa values of all diagnostic subscales are above .75; Sheehan et al., 1997). In addition, comparison of the MINI with the SCID-I has shown that, in general, MINI-diagnoses are characterized by good or very good kappas (except for current drug dependence with a kappa below .50; kappa for PTSD = .78), good sensitivity (>.70 except for dysthemia, obsessive–compulsive disorder and current drug dependence), and high specificities and negative predictive values (>.85; Sheehan et al., 1998b).

Structured clinical interview for DSM-IV (SCID-I and SCID-II). Both the SCID-I and SCID-II are standardized, semi-structured interviews for diagnosing DSM-IV psychiatric axis I (First, Spitzer, Gibbon, & Williams, 1996) and axis II disorders (First, Gibbon, Spitzer, Williams, & Benjamin, 1997). The reliability of the SCID-I in different patient samples was shown to be good with overall kappas of .61 for current and .68 for lifetime diagnoses. The reliability of the Dutch version of the SCID-II was shown to be good: in an outpatient population kappas ranged from .77 for obsessive–compulsive personality disorder to .82 for avoidant personality disorder. Weighted kappa for all personality disorders was .80. The inter-rater agreement proved to be fair to excellent (Intraclass Correlation Coefficients (ICC) ranging from .41 to .88), except for the dependent personality disorder (ICC < .40; Weertman, Arntz, Dreessen, Van Velzen, & Vertommen, 2003).

Outcome measures

Clinician-Administered PTSD Scale (CAPS-1). The CAPS-1 is a structured interview designed to test for the presence of the 17 DSM-IV-TR criteria for PTSD and to establish PTSD severity in the previous month (Blake et al., 1995). Each symptom is scored on two dimensions, i.e., frequency and intensity, using 5-point scales. The inter-rater diagnostic agreement proved excellent (Blake et al., 1990), and test-retest reliability for the three symptom clusters ($r = .77$ to .96) and total scale ($r = .90$ to .98) was good (Blake et al., 1995). The internal consistency for all CAPS-1 items proved to be high ($\alpha = .94$; Blake et al., 1995) and the concurrent validity adequate (correlation with Mississippi Scale for Combat-related PTSD: $r = .70$ to .91, correlation with MMPI PTSD subscale $r = .77$ to .84; Blake et al., 1990, 1995). We used a Dutch version of the CAPS-1 (Hovens, Luinge, & Van Minnen, 2005).

Posttraumatic stress symptom Scale–Self-Report (PSS-SR). The PSS-SR is a 17-item self-report questionnaire that measures the frequency of PTSD symptoms using 4-point Likert scales (Foa, Riggs, Dancu, & Rothbaum, 1993). Each item corresponds to one of the DSM-IV-TR criteria for PTSD, and has three symptom subscales: reexperiencing, avoidance and arousal. Analyses showed a high internal consistency (Cronbach's alpha for the total score was .91), and a good test-retest reliability of the overall severity (.74; Foa et al., 1993). The Dutch version also shows good internal consistency ($\alpha = .92$; Mol et al., 2005).

Beck Depression Inventory (BDI). The BDI (Beck, Ward, Mendelson, Mock, & Erbaugh, 1961) is a 21-item self-report questionnaire assessing the severity of depressive symptoms (score range per item is 0–3). Its internal consistency for both psychiatric and nonpsychiatric samples was shown to be high (α coefficients range from .76 to .95 and .73 to .92 respectively). Its concurrent validity was also high in both psychiatric and nonpsychiatric samples (correlation with clinical ratings: $r = .55$–.96, correlation with Hamilton Rating Scale for Depression: $r = .61$–.86, correlation with Zung: $r = .57$–.86, correlation with MMPI-D: $r = .41$–.75; Beck, Steer, & Garbin, 1988). The Dutch version of the BDI also showed good internal consistency ($\alpha = .91$; Schotte, Maes, Cluydts, De Doncker, & Cosyns, 1997).

Subjective Unit of Distress Scale (SUDS). Subjective fear was measured using the SUDS, a visual analogue scale on which the respondent indicated the degree of fear felt at that moment by placing a number from 0 (no fear) to 10 (panic) on the 10-cm horizontal line. To establish pretreatment fear levels, patients took a 9-minute behavioral exposure test, during which they were asked to indicate their current fear level every 3 min. Following Jaycox et al. (1998) a patient's level of fear activation was defined as his/her mean SUDS score during this test.

Dissociative phenomena

Trait dissociation was measured with the Dissociative Experiences Scale (DES; Bernstein & Putnam, 1986; Carlson & Putnam, 1993), a 28-item self rating scale that measures the tendency to

experience dissociative experiences in daily life (total range: 0–100). For each item the respondent states how often a specific dissociative symptom occurs. Compared to other instruments gauging dissociation, the convergent validity of the DES was shown to be very good and its reliability good (overall Cohen's $d = 1.82$ and mean alpha reliability = .93 respectively; Van IJzendoorn & Schuengel, 1996). In our sample, using the Dutch translation of the DES, reliability was similarly high (Cronbach's alpha = .92). Furthermore, it was found that the DES assesses a single dimension of dissociation (Holtgraves & Stockdale, 1997). Bremner et al. (1998) found some evidence for the DES measuring general dissociative tendencies and not state dissociation, as their Clinician-Administered Dissociative States Scale (CADSS), which measures dissociative states, was only moderately correlated to the DES.

Depersonalization was evaluated using by the 3 dissociation items of the associated features of PTSD from the CAPS (CAPS-D; reduced awareness, derealization, and depersonalization). We used the items' mean score in the analyses.

Numbing was defined based on the three numbing items from the PSS-avoidance subscale (PSS-SR-N; detachment from others, restricted affect, diminished interest in activities; see e.g., Litz, 1992). Like all PSS-SR items, the numbing items typically inquire after symptoms in the preceding week. The items' mean score was used in the analyses.

Procedure

The participants cooperated on a voluntary basis and all assessments were conducted by trained, independent assessors that were naïve with respect to the patients' developments in treatment. Pretreatment screening (establishing diagnosis, comorbidity, and inclusion and exclusion criteria) comprised the SCID-I, and later MINI, and SCID-II. Within a week, included patients subsequently completed all pretreatment questionnaires and took part in the CAPS interview. Inter-rater reliability was not checked for the clinical interviews, but SCID and MINI PTSD diagnoses were compared to and confirmed by the CAPS in all cases. Lastly, to establish pretreatment levels of distress they took the 9-minute behavioral exposure test which was delivered by two trained independent experimenters. Imaginal exposure during this test was consistent with the protocol for prolonged exposure treatment for PTSD the patients would be attending later (see next paragraphs; Dancu & Foa, 1993). The patients and the experimenter selected the first frightening (part of the) trauma from the exposure hierarchy for this purpose. Subjective levels of distress were rated by the patient at 0, 3, 6, and 9 min.

One week after the abovementioned assessments, patients entered a standardized prolonged exposure treatment program (Dancu & Foa, 1993) comprising 8–12 weekly sessions that lasted 45 min. Note, however, that in 10 cases treatment was ended before the 8th session because the patients concerned had already achieved (full) recovery: they no longer met the DSM-IV-TR PTSD criteria according to the CAPS, their PSS-SR total score had dropped below 10, and their SUDS scores recorded during the 3 last exposure sessions and the subsequent homework assignments were low (<5). The mean number of session of these early completers was 4.38 ($SD = 1.71$).

The first therapy session included a presentation of the treatment rationale, education about the disorder and common reactions to trauma and information gathering. The subsequent sessions consisted of 30 min imaginal exposure: patients were asked to close their eyes and talk about the traumatic event in the first person and in the present tense, recollecting as many sensory details as vividly as possible, i.e., as if the trauma was happening "here and now". Each imaginal exposure session was audiotaped and patients were instructed to listen to the tape at home five times a week. From the 4th session onwards in vivo exposure assignments were an integrated part of the treatment. These included exposure to fearful stimuli associated with the trauma, such as visiting trauma-related places or listening to trauma-related sounds. Each session started with a review of the patients' homework and ended with homework assignment. At the start of each treatment session patients also completed the PSS-SR. Treatment fidelity was rated after each session and in addition audiotapes of treatment sessions were randomly selected and coded for treatment protocol adherence by independent raters. The treatment protocol was indeed followed closely by all therapists. All therapists ($n = 9$) involved were supervised weekly by the second author. Furthermore, therapists were not aware of the patients' initial depression or dissociation scores. Their experience ranged from 0 years (just graduated) to 12 years (experienced, registered psychotherapists with postdoctoral training). Following treatment conclusion, all patients again completed the questionnaires measuring state symptoms and participated in the posttreatment CAPS interview.

Follow-up assessments comprising all questionnaires measuring state symptoms and the CAPS were conducted six months after the posttreatment assessment.

Table 1
Correlations between dissociative and depressive symptoms ($N = 71$).

	BDI	DES	CAPS-D	PSS-SR-N
BDI		.53*	.31*	.49*
DES			.29*	.38*
CAPS-D				.44*
PSS-SR-N				

Note. BDI = Beck Depression Inventory, DES = Dissociative Experiences Scale, CAPS-D = Clinician-Administered PTSD Scale-Depersonalization, PSS-SR-N = Posttraumatic Stress Symptoms-Self Rating Scale-Numbing.
*$p < .05$.

Statistical analyses

Repeated measures analyses were conducted to analyze treatment effect for PTSD symptoms and dissociative phenomena using the intent-to-treat (ITT) sample. Because no posttreatment data were available for the dropouts, data were analyzed with the last observation carried forward (LOCF) to establish improvement for the entire ITT sample. As we wished to study the effect of dissociation and depression on improvement, we only included the data of the completers in our subsequent linear regression analyses. Although this may seem to provide a distorted image, it is in fact addressing the research question, which concerns the impact of dissociation and depression when someone completes treatment. These results are very important from a clinical point of view.

Several distinct statistical analyses were conducted to address the various research questions. To begin with, correlations between the DES, CAPS-D, PSS-SR-N, and BDI were calculated to check whether these indeed reflected associated but distinct constructs. As all correlations were indeed significant but moderate (Table 1) we did not use a composite variable but instead analyzed all dissociation and depression variables separately. Next, repeated measures analyses with PSS[1], CAPS-D, PSS-SR-N, and BDI as independent variables were used to analyze the effect of

[1] CAPS-data are not reported in this analysis or any of the following analyses, because the CAPS was introduced in the research at a later stage of our study, resulting in CAPS-data of only 47 completers. However, all analyses were recomputed with the CAPS, leading to the similar results.

exposure treatment on PTSD, depersonalization, numbing and depressive symptoms.

The impact of dissociative and depressive symptoms on improvement was analyzed in two ways: 1) overall regression analyses and 2) comparing extreme symptom profiles. In the simultaneous entry regression analyses the three pretreatment dissociation measures (DES, CAPS-D and PSS-SR-N) and depressive symptoms (BDI) were entered as independent variables. We used PSS-SR residual gain scores as a dependent variable to reflect change in PTSD symptoms and control for initial PTSD severity (Steketee & Chambless, 1992)[2]. Still, it was possible that overall analyses would not yield any effects because dissociation and depression have an impact on treatment efficacy only in patients with high levels of these symptoms. We hence divided the patients into high and low dissociation and depression groups, based on their pretreatment DES, CAPS-D, or PSS-SR-N scores (mean ±.5 *SD*). With this procedure we eliminated patients whose scores were in the middle range, thereby possibly neutralizing effects. It also allowed us to compare substantial groups of high- and low-scoring patients (about 50% of the total sample), minimizing losses of power. With regard to depression, patients were divided into 3 groups based on their DSM-IV-TR diagnosis: 1) current depression, 2) depressive episode(s) in the past but no current depression, 3) and no current or past depression. Improvement in these high- and low-scoring patients was subsequently compared using 2 (high versus low) × 3 (pre, post, follow-up) repeated measures MANOVAs and χ^2-tests.

Finally, to study whether dissociation and depression were associated with impeded fear activation correlations were calculated between DES, CAPS-D, PSS-SR-N, and BDI with mean SUDSs during the behavioral exposure test, using Bonferroni adjustments. The entire ITT sample ($N = 71$) was used in these analyses.

Results

Treatment outcome

Treatment was successful in linearly decreasing PTSD symptoms from pretreatment to follow-up in the ITT sample ($F(1, 70) = 126.84$, $p < .001$) and in the completers sample ($F(1, 59) = 154.37$, $p < .001$). Means, SDs and within subject effect sizes, controlling for repeated measurements, of the outcome measures are listed in Table 2. End-state functioning was defined as being at or below 20 on the PSS-SR and at or below 10 on the BDI, following Foa et al. (1999). Based on these criteria, 58% of the completers achieved good end-state functioning. Remarkably, there was a great discrepancy between patients achieving good end-state functioning using the criteria for the PSS and those for the BDI, with 90% of the completers achieving the PSS criterion and 57% the BDI criterion. Pretreatment PTSD severity and improvement were comparable to other PTSD studies evaluating prolonged exposure treatment (e.g., Foa et al., 1999). Considering the range of (multiple) traumas in our patient cohort, the improvement rate was quite high. The linear decrease of numbing symptoms, depersonalization, and depressive symptoms from pretreatment to follow-up was also significant in the ITT sample (PSS-SR-N: $F(1, 70) = 36.11$, $p < .001$; CAPS-D: $F(1, 54) = 7.16$, $p < .01$; BDI: $F(1, 70) = 51.98$, $p < .001$) and in the completers sample (PSS-SR-N: $F(1, 59) = 40.84$, $p < .001$; CAPS-D: $F(1, 46) = 7.30$, $p < .01$; BDI: $F(1, 59) = 66.71$, $p < .001$). In sum, prolonged exposure treatment successfully reduced PTSD, including numbing symptoms, depersonalization and depressive symptoms.

Table 2
Means (SDs) of outcome measures for the completers sample ($N = 60$).

	Pretreatment	Posttreatment	Follow-up	Partial η^2	Cohen's *d*
PSS-SR*	25.52 (8.70)	10.95 (9.75)	9.20 (8.40)	.77	2.70
CAPS*	66.17 (16.81)	27.50 (26.24)	22.08 (22.08)	.82	3.07
BDI*	20.21 (10.50)	12.24 (10.05)	10.11 (9.23)	.53	2.18
DES	18.41 (13.44)	–	–	–	–
CAPS-D*	3.18 (3.88)	1.06 (2.39)	1.44 (3.27)	.28	.96
PSS-SR-N*	3.70 (2.50)	1.53 (2.22)	1.38 (1.97)	.47	1.81
Mean SUDS during the Behavior Exposure Test	7.58 (1.74)	–	–	–	–

Note. Effect sizes and significance values concern pretreatment to follow-up repeated measures analyses. PSS-SR = Posttraumatic Stress Symptoms-Self Rating Scale, CAPS = Clinician-Administered PTSD Scale, BDI = Beck Depression Inventory, DES = Dissociative Experiences Scale, CAPS-D = Clinician-Administered PTSD Scale-Depersonalization, PSS-SR-N = Posttraumatic Stress Symptoms-Self Rating Scale-Numbing, SUDS = Subjective Unit of Distress Scale.
*$p < .01$.

Effect of dissociation and depression on treatment efficacy

As treatment efficacy concerns both improvement and dropout, we first tested whether dropouts showed more pretreatment dissociative and depressive symptoms than completers. Dropouts did not differ from completers on trait dissociation (DES: $t(69) = .09$, $p = .93$), depersonalization (CAPS-D: $t(69) = .20$, $p = .84$), numbing (PSS-SR-N: $t(69) = .90$, $p = .37$), depressive symptoms (BDI: $t(69) = .24$, $p = .81$), or in the presence of a current or past mood disorder ($\chi^2(1, N = 71) = .17$, $p = .68$). Means for dropouts and completers were respectively: DES: 18.80 ($SD = 11.74$) and 18.26 ($SD = 13.09$), CAPS-D: 3.45 ($SD = 5.32$) and 3.18 ($SD = 3.88$), PSS-SR-N: 4.46 ($SD = 2.84$) and 3.70 ($SD = 2.50$), and BDI: 20.82 ($SD = 6.71$) and 20.06 ($SD = 10.19$). Current or past depressions were present in 18% of the dropouts and 38% of the completers.

Regression analyses showed that none of the three dissociation variables, nor depressive symptoms predicted pre-to-posttreatment PTSD reduction ($\Delta R^2 = .13$ $p = .17$, all β's ns), indicating that neither pretreatment dissociation nor depression had affected improvement. Similarly, none of the dissociation variables, nor depressive symptoms proved to predict pretreatment to follow-up PTSD reduction ($\Delta R^2 = .10$ $p = .25$, all β's ns). The results of this latter regression analysis must be interpreted with caution, though, because 7 (12%) of the patients from the completers sample did not take part in the follow-up assessment.[3]

Next, extreme symptom groups were compared. The parallel lines in Fig. 1[4] indicate a similar pretreatment to follow-up decline in PTSD symptoms in patients with high ($n = 15$, M DES = 34.91, $SD = 9.59$) and low trait dissociation ($n = 21$, M DES = 6.19, $SD = 2.39$), high ($n = 25$, M CAPS-D = 7.82, $SD = 3.38$) and low depersonalization ($n = 17$, M CAPS-D = 0, $SD = 0$), high

[2] As some items of the PSS-SR were used to assess emotional numbing, thereby creating an overlap between the dependent (PSS-SR) and independent (PSS-SR-N) variable, the same analysis was also executed with the CAPS and with the PSS-SR minus the numbing items. Results of those analyses were similar to the ones reported here.

[3] Regression analyses were also executed with the ITT sample, again showing no effect of any of the variables on PTSD symptom reduction from pre-to-posttreatment ($\Delta R^2 = .06$ $p = .47$, all β's ns), or from pretreatment to follow-up ($\Delta R^2 = .08$ $p = .32$, all β's ns).

[4] Because the number of sessions varied between patients, the PSS-SR of the middle session was used in the graphs to show the course of PTSD symptoms during treatment.

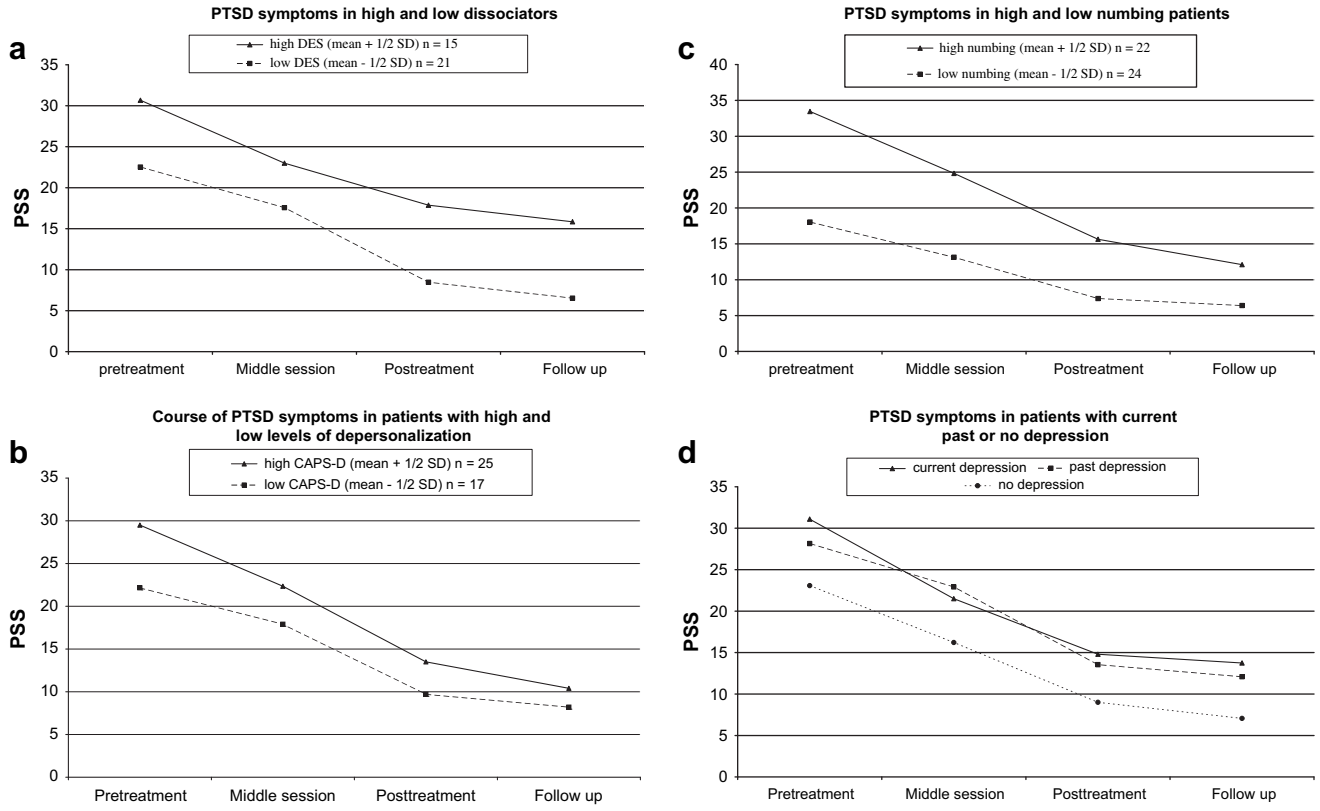

Fig. 1. a. The course of PTSD symptoms in patients with high and low trait dissociation. b. The course of PTSD symptoms in patients with high and low levels of depersonalization. c. The course of PTSD symptoms in patients with high and low levels of numbing. d. The course of PTSD symptoms in patients with current, past, and no depression.

($n = 22$, M PSS-SR-N = 6.53, $SD = 1.23$) and low ($n = 24$, M PSS-SR-N = 1.22, $SD = .85$) numbing, and current ($n = 12$, M BDI = 27.60, $SD = 13.33$), past ($n = 13$, M BDI = 23.92, $SD = 7.33$), or no current or past depression ($n = 35$, M BDI = 16.39, $SD = 8.22$). Note that a substantial number ($n = 11$, i.e., 18% of the entire completers sample) of the high DES patients were severely dissociative based on a cut-off score of 30. The 2 × 3 repeated measures pretreatment to follow-up MANOVA did not show an interaction effect for DES ($F(1, 30) = .18$, $p = .68$), or depression ($F(1, 51) = .32$, $p = .57$), indicating PTSD symptoms declined similarly in high and low DES and in current/past and no depression groups. There was an interaction effect for PSS-SR-N ($F(1, 36) = .14.35$, $p = .001$), but in a surprising direction: "high numbing" patients showed a greater reduction in pre-to-follow-up PTSD symptoms than "low numbing" patients. There was a similar trend for patients with high versus low levels of depersonalization ($F(1, 34) = 3.27$, $p = .08$) too. As expected, time also proved to be significant ($F(1, 36) = 122.75$, $p < .001$).

The parallel improvement curves and the absence of an interaction effect for the high and low DES and depression groups suggested that the high DES patients and those with a current/past depression would exhibit more PTSD symptoms at follow-up. This indeed proved the case; these two groups of patients met PTSD criteria at follow-up more often than patients with low DES ($\chi^2(1, N = 32) = 11.79$, $p = .002$) and those without depression ($\chi^2(1, N = 53) = 4.41$, $p = .04$). That is, 10% of the low versus 69% of the high DES group, and 18% of the non-depressed versus 45% of the past/current depressed met PTSD criteria at follow-up. Interestingly, patients with a current or a past depression did not differ from each other at follow-up ($\chi^2(1, N = 20) = .04$, $p = .85$). There was no difference in meeting PTSD criteria between patients with high and low levels of depersonalization ($\chi^2(1, N = 37) = .02$, $p = .99$), and high and low numbing patients ($\chi^2(1, N = 39) = 1.37$, $p = .24$). That is, 27% of the patients with low levels of depersonalization versus 29% of those with high levels, and 20% of the low numbing versus 31% of the high numbing patients met PTSD criteria at follow-up.

Dissociation and fear activation

DES ($r = .38$, $p < .01$), CAPS-D ($r = .27$, $p < .05$), PSS-SR-N ($r = .37$, $p < .01$), and BDI ($r = .37$, $p < .01$) were significantly related to mean SUDSs, albeit in the opposite direction as hypothesized: higher levels of dissociative and depressive symptoms were associated with higher levels of subjective fear during the behavioral exposure test. As the fear habituation curves during the behavioral exposure test were similar for high and low DES, high and low CAPS-D, high and low PSS-SR-N, and current or past versus no depression, Fig. 2 only depicts the habituation curves for the high and low numbing patients (mean ±.5 SD). Because the relationship between high levels of dissociative and depressive symptoms and elevated fear levels during the behavioral exposure test could be due to the fact that these were patients experiencing more severe PTSD symptoms to begin with, partial correlations were calculated while controlling for initial PTSD symptoms (pretreatment PSS-SR). Indeed, in these analyses all significant correlations between dissociative and depressive symptoms and fear during the behavioral exposure test disappeared (all *p*s ns).

In sum, neither the three pretreatment dissociative phenomena nor depression were associated with poorer improvement after exposure treatment. In addition, rather than impeding fear activation, numbing, depersonalization, trait dissociation, and

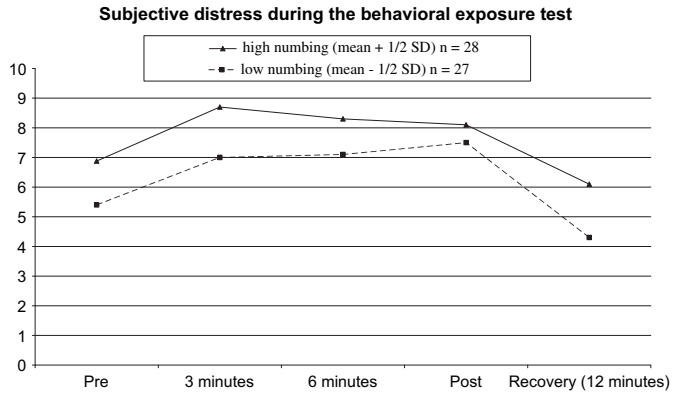

Fig. 2. Pretreatment SUDS scores during the behavioral exposure test for patients with high and low levels of numbing.

depressive symptoms were related to higher fear levels during the behavioral exposure test, although this finding was explained by higher levels of pretreatment PTSD symptoms.

Discussion

The present study examined the impact of three dissociative phenomena and depression on exposure treatment efficacy in PTSD patients. The prolonged exposure treatment not only reduced PTSD symptoms, but also numbing, depersonalization and depressive symptoms. In contrast to our hypothesis, pretreatment trait dissociation, depersonalization, numbing, and depressive symptoms did not predict improvement or dropout. In fact, patients with high levels of trait dissociation, depersonalization, or numbing as well as patients with a past or current depression showed a similar reduction of PTSD symptoms from pre-to-posttreatment and pre-to-follow-up as patients with low levels of these dissociative phenomena or patients that had no (history of) depression. Patients with high levels of trait dissociation or depressive patients (current and past) showed more severe PTSD symptoms at posttreatment and follow-up, but also at pretreatment, thus showing similar improvement as patients with low levels of trait dissociation or no (history of) depression. Surprisingly, "high numbing" patients (also having more severe pretreatment PTSD symptoms) even showed a greater reduction in PTSD symptoms from pretreatment to follow-up than "low numbing" patients, and a trend in the same direction was found for patients with high levels of depersonalization. As a result, there was no difference between patients with high or low levels of depersonalization or numbing in the number of patients meeting PTSD criteria at follow-up. In sum, dissociation and depression had no predictive value with respect to improvement from treatment. With respect to depression, our results confirm earlier findings that depressive symptoms improve as a function of exposure treatment for PTSD (e.g. Foa et al., 1999); the findings on dissociation are quite novel.

The results have some important implications for clinical practice. Most importantly, PTSD patients with elevated levels of dissociation and those with a comorbid depressive disorder seem to improve similarly as a result of exposure treatment as patients without these symptoms. They are also not more likely to dropout of treatment. Thus, it would be ill-advised to exclude these patients from prolonged exposure treatment. Note that as suicidal patients were excluded from the present study, these conclusions should not be generalized to depressive PTSD patients with suicidal intent. The finding that patients with high levels of dissociation gained from treatment as much as others may also be relevant with respect to treating PTSD patients that suffered sexual abuse and subsequently have developed dissociative symptoms. The present study included childhood sexual abuse and dissociation did not affect improvement. This is consistent with an earlier study of Van Minnen et al. (2002), who found the type of trauma (childhood versus adulthood trauma) not to be a relevant predictor of exposure treatment outcome. Indeed, other studies with childhood sexual abuse victims also showed that CBT can be effective in this group (Resick et al., 2008). Note that this latter study as well as our own study did not use any stabilization or emotional skill training phases (Levitt & Cloitre, 2005), but instead started cognitive therapy or exposure immediately. Still, more research is required before any firm conclusions can be drawn about the efficacy of exposure therapy and phase-based treatment in victims of childhood (sexual) trauma.

The results also have some implications for current theories on exposure treatment for PTSD. Interestingly, not only did dissociation and depression not hamper effective exposure treatment, in fact, symptoms of depersonalization, numbing and depression even declined as a result of exposure therapy. This actually makes sense because one of the aims of exposure treatment is to help patients engage and experience their fear, thereby reducing emotional numbing during exposure. This may have contributed to the reduction of numbing and depersonalization symptoms. Alternatively, as dissociation is no longer needed as a means of coping with anxiety (Elzinga, Bermond, & Van Dyck, 2002) it may have faded out when PTSD symptoms diminish. Similarly, Foa and Rauch (2004) found that exposure alone was just as effective in reducing negative cognitions as exposure plus cognitive restructuring was. This latter finding could indicate the main working mechanism of exposure is inhibitory learning, for example, disconfirmation of harm expectancy leads to new CS–US associations (Craske et al., 2008). Our results possibly indicate that the presence of dissociative symptoms does not hinder the formation of such new associations or are not present at critical times during the exposure.

Another interesting finding concerns the similar decline of PTSD symptoms in patients with higher levels of dissociation and (current or past) depression, compared to patients with lower levels of dissociation and no (history of) depression. At the 6-month follow-up, the number of patients meeting PTSD criteria was similar for patients with high and low numbing or depersonalization. As they started off with more severe PTSD symptoms, the patients with high levels of numbing or depersonalization may have needed more time to recover. However, because treatment was not controlled during the 6 months from posttreatment to follow-up, it is impossible to say what happened during this period. Patients with high trait dissociation levels and patients with a current or past depression improved like the others, but did meet PTSD criteria at follow-up more often than patients with low trait dissociation levels and patients without (a history of) depression. High trait dissociation may reflect a personality trait that makes one chronically more vulnerable to stress. These patients may hence have higher stress levels chronically, perhaps also before the onset of their PTSD. Their end-state functioning is then limited to symptoms associated with a stress-related personality trait. Indeed, recent studies have shown trait dissociation to be related to neuroticism (Goldberg, 1999; Kwapil, Wrobel, & Pope, 2002) or even psychiatric symptoms in general (Spindler & Elklit, 2003). Moreover, in the current study, trait dissociation was related specifically to the PTSD arousal ($r = .41$, $p < .01$) and avoidance ($r = .40$, $p < .01$) symptom clusters, and not to the reexperiences cluster ($r = .23$, ns). Interestingly, there was no difference in meeting PTSD criteria between patients with a current and those with a past depression, again suggesting that an underlying stress-related trait is responsible for the maintenance of a somewhat increased and chronic symptom level. In this respect, it would be

interesting to also address neuroticism in future studies, because it is possible that patients with high trait dissociation levels and those with a current/past depression show a similar end-state of PTSD symptoms as patients high on neuroticism.

Finally, trait dissociation, depersonalization, numbing, and depression did not impede fear activation during exposure. On the contrary, all three were related to higher fear levels during exposure, although this association disappeared after controlling for initial PTSD severity. This suggests that dissociation may not protect the individual against experiencing distress but instead may be an epiphenomenon of high levels of distress. Other studies (Fikretoglu et al., 2006, 2007) showed similar results: high levels of peritraumatic dissociation were associated with high levels of peritraumatic distress. In fact, in the present study, initial PTSD severity (indicating higher levels of distress) was responsible for the association between dissociative and depressive symptoms, and fear during exposure. An additional explanation may be that numbing is a coping reaction to reduce distress (Litz et al., 1997), which can be switched off during high stress. Because exposure therapy aims to induce fear and distress, it thereby halts coping symptoms like numbing, thus allowing the patient access to the fear network. It has been suggested that numbing and hyperarousal are related symptom clusters (Litz et al., 1997; Yoshihama & Horrocks, 2005), which is consistent with our finding that the "high numbing" patients reacted with higher distress during the behavior exposure test (hyperarousal) than the "low numbing" patients. More explicitly, in the absence of numbing, the patients experienced new, and thus extra, distress. May be numbing symptoms should not be interpreted as an inability to *experience* emotions (i.e., fear during exposure), but instead as an inability to adequately *regulate* emotions (in this case anxiety). Note that there is same debate about the necessity of fear activation as an essential aspect of exposure therapy. That is, fear activation may not be an adequate indicator of extinction learning or long-lasting improvement. It is beyond the scope and not the focus of the present article, but the interested reader can find an elegant explanation in Craske et al. (2008).

Although dissociation is believed to negatively affect the efficacy of exposure treatment in PTSD, to our knowledge, the present study is the first to address this issue directly. The study is strong in that it assesses three well-defined types of dissociation (trait dissociation, depersonalization, and numbing), and the related construct of depression, thereby recognizing that dissociation covers a wide range of symptoms. Moreover, it included a relatively large sample and numerous types of traumas (including sexual abuse), allowing the results to be generalized to exposure treatment of a broad range of traumas. Nevertheless, not all traumas were represented (e.g., war trauma) and our results hence warrant replication in other treatment studies that include these other trauma populations. The formation of extreme dissociative and depressive subgroups is a procedure that is often used to study sample extremities. However, forming high and low symptom profile groups may also result in decreased power, hereby not detecting possible effects. These analyses therefore warrant replication in preferably large samples. In addition, although a substantial number of patients were severely dissociative (Carlson et al., 1993), the study also merits replication in patients with dissociative disorders included in the sample. Perhaps a randomized control trial would be an elegant design for this purpose. Other types of dissociation, like amnesia, must also be studied in order to determine their impact on treatment efficacy. It would furthermore be interesting to not only administer the DES at pretreatment but also following treatment cessation because, even though the DES is thought to measure trait dissociation and therefore considered to be stable, it is possible that part of the DES-score depends on the level of PTSD symptoms. The DES-score may therefore also decline after treatment. Moreover, although the DES is a widely used and accepted instrument to assess general dissociative tendencies, it has also been criticized. For example, it may be sensitive to response biases (Van IJzendoorn & Schuengel, 1996), which could possibly affect results. It also seems to assess the current perception of past experiences, although this does not have to be a problem in our study, because we used the DES to assess current and not past dissociative symptoms. On the other hand, its excellent predictive validity would be a reason to include the DES in predictive studies. With respect to dissociation measures, it would be interesting to further explore the effect of dissociation during exposure using the PDEQ or the CADSS, instruments that specifically assess "peri-event" dissociation. Results may very well be similar, as the CADSS for example proved related to the DES (Bremner et al., 1998), but this has yet to be proved. Finally, previous studies on fear activation in PTSD usually used SUDS measures derived from exposure treatment sessions. However, as exposure duration may vary and the exposure treatment is conducted by the patient's own therapist, we used a behavioral exposure test in order to control for any resultant confounders. Moreover, all behavioral exposure tests were conducted by the same two independent experimenters.

In conclusion, although the prevailing view in clinical practice is that dissociation and depression have a negative impact on exposure treatment for PTSD, we found no evidence to support this belief. Conversely, we found depersonalization, numbing and depressive symptoms to have declined after exposure treatment. There was no difference in dissociative and depressive symptoms between dropouts and completers. Furthermore, relative to patients with low levels of dissociation (trait dissociation, depersonalization and numbing) and no depression, fear activation was not impeded in patients with high levels of dissociation and those with a current or past depression, these latter patients showed a similar decline of PTSD symptoms during therapy, and did not relapse after 6 months. These findings have clear clinical relevance as they indicate that PTSD patients with serious comorbid dissociative or depressive symptoms are just as likely to profit from effective treatment programs like exposure as those with low dissociative and depressive symptom levels.

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