Research Article

THE IMPACT OF DISSOCIATION ON PTSD TREATMENT WITH COGNITIVE PROCESSING THERAPY

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Background: *This secondary analysis of data from a randomized controlled trial of cognitive processing therapy (CPT) and its constituent components investigated whether dissociation decreased over the course of treatment primarily targeting symptoms of posttraumatic stress disorder (PTSD) and explored whether levels of dissociation predicted treatment outcome differentially by treatment condition.* **Methods:** *An intention to treat sample of 150 women were randomized to CPT, cognitive therapy only (CPT-C) or written trauma accounts only (WA). Dissociation was measured by the dissociation subscale of the Traumatic Stress Inventory and the Multiscale Dissociation Inventory.* **Results:** *Multilevel regression analyses revealed significant decreases in dissociation that did not vary as a function of treatment condition. Growth curve modeling revealed significant treatment condition by dissociation interactions such that the impact of pretreatment levels of dissociation impacted the treatment conditions differently.* **Conclusions:** *Women who endorsed low pretreatment levels of dissociation responded most efficiently to CPT-C, whereas women with the highest levels of dissociation, in particular high levels of depersonalization, responded better to CPT. Depression and Anxiety 00:1–12, 2012.*

Key words: *posttraumatic stress disorder; dissociation; cognitive processing therapy; randomized controlled trial*

INTRODUCTION

A dissociative subtype of posttraumatic stress disorder (PTSD) is being considered for the new edition of the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM*).[1] Therefore, it is important to determine whether dissociation makes any difference in PTSD treatment. A range of research indicates that individuals with PTSD and dissociation are different than those who have PTSD without dissociation. In one of the first illustrations that highly dissociative trauma victims respond differently than victims with lower levels of dissociation, Griffin, Resick, and Mechanic[2] found that although women's subjective reports of peritraumatic dissociation fell on a relatively normal distribution, their physiological responses during recall of trauma were nonlinear. When relaying their trauma accounts, individuals with low and medium levels of peritraumatic dissociation showed elevations in skin conductance and heart rate; whereas, those with high levels of peritraumatic dissociation showed decreases in both physiological measures. Interestingly, there were no differences in the levels of subjective distress reported by the two groups.

Using functional magnetic resonance imaging (fMRI), several studies have found different patterns of response among participants with chronic PTSD who dissociate during script-driven imagery tasks,[3–5] compared to

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those who do not dissociate. Individuals with PTSD typically demonstrate deactivation of the medial prefrontal cortex and anterior cingulate cortex—brain regions that play an important role in arousal and emotion regulation; however, dissociative participants showed abnormally high activation in these regions.[6] Thus, Lanius et al.[6] recently proposed that there is sufficient neurobiological evidence to warrant a dissociative subtype of PTSD based on emotion dysregulation that involves emotional overmodulation. Because of this overmodulation, the authors expressed concern about conducting exposure therapy with highly dissociative patients because emotional engagement with the trauma memory would be prevented and recommended treating the dissociation first.

Epidemiological studies also indicate that dissociation is a distinct taxon or subtype of PTSD. For example, using the Dissociative Experiences Scale (DES),[7] Waelde, Slivern, and Fairbank[8] found a taxonomic group of highly dissociative Vietnam Veterans with severe PTSD. Specifically, among participants with a current PTSD diagnosis, 32% belonged to the dissociative taxon, suggesting a subtype of severe PTSD with elevated dissociation.

A remaining question is whether dissociation severity negatively impacts PTSD treatment. Two primary mechanisms of change are thought to underlie efficacious PTSD treatment. Emotional processing theory,[9] the theoretical model associated with prolonged exposure therapy (PE), hypothesizes that PTSD reduction requires repeated activation of a pathological fear network associated with the traumatic event, along with the corrective information that will make the fear network less threatening and anxiety provoking, resulting in extinction. Cognitive models and related treatments; e.g. cognitive processing therapy (CPT) propose that directly changing maladaptive beliefs and assumptions associated with traumatic events results in changes in emotions and significant reductions in PTSD symptoms,[10] and may be more efficient than repetitions of the trauma account.[11] As described above, research has shown that dissociation can interfere with emotional responding. Moreover, there are documented cognitive difficulties and related neuropsychological impairments associated with dissociation.[12] Therefore, it is reasonable to hypothesize that dissociation might interfere with the effectiveness of PTSD treatment. However, thus far, treatment research on PTSD has found mixed results with regard to this question. In fact, sometimes dissociation has been used as the basis of exclusion from studies, operating on the assumption that it would interfere with treatment.[13]

Studies that have examined this question have generally not found dissociation to be predictive of PTSD treatment outcome. However, most of these studies had small sample sizes and may have been underpowered.[14–17] Findings have been inconsistent with regard to whether dissociation changes over the course of PTSD treatment. For example, McDonagh et al.[18] conducted a randomized controlled trial (RCT) with 74 childhood sexual abuse (CSA) survivors with PTSD. They compared PE versus present centered therapy or a wait-list (WL) group. Neither of the active treatments had a significant effect on dissociation as measured by the DES, nor was there a group by time interaction. Rothbaum[19] compared eye movement desensitization and reprocessing versus WL ($n = 21$ rape victims) and found that both groups decreased equally on the DES.

In contrast, some studies have found improvements in dissociation while treating PTSD without specifically targeting dissociation. Zlotnick et al.[20] compared affect management to WL ($n = 48$ CSA survivors with PTSD) and found that the treatment group exhibited less dissociation, as measured by the DES, than the WL group at posttreatment. Cloitre et al.[21] compared a two-phase treatment, skills training in affective and interpersonal regulation followed by modified PE (STAIR/MPE), to a WL among 58 women with CSA histories. Interestingly, there was no improvement in dissociation in the first, skills focused, phase of treatment; however, there was a significant improvement in dissociation as measured by the Dissociation Scale (DISS)[22] during the exposure portion of treatment. Chard[23] included the DES-II[24] in a study of CPT for 71 women with CSA histories and PTSD (55 treatment completers). Women in the CPT group demonstrated significant improvements on dissociation at posttreatment relative to the WL group. The large gains were maintained through 1 year follow-up.

As discussed by Bryant[25] and Hagenaars et al.,[14] dissociation is a complicated phenomenon that includes a variety of symptoms usually studied as a general construct. The use of a range of general dissociation scales may be one reason why there is a variety of outcomes in research to date. Bryant[25] argues that future research should deconstruct dissociation into distinct factors (e.g. time distortion, derealization, depersonalization, reduced awareness) in order to evaluate whether there are differential treatment effects by factors to better understand mechanisms of change in PTSD treatment. For example, if there is a dissociative subtype of PTSD, the effects on treatment outcome may be nonlinear and may be missed without a nuanced examination of the data.

Thus, the present study examines dissociation as a predictor of PTSD treatment outcome and explores effects of PTSD treatments on dissociation using data from a RCT of PTSD treatment conducted with female victims of interpersonal violence by Resick et al.[11] The purpose of the larger RCT was to conduct a dismantling study of CPT comparing the complete protocol (CPT) to its constituent components: cognitive therapy only (CPT-C) and written trauma accounts only (WA) for the treatment of PTSD and comorbid symptoms. The trial included multiple measures of dissociation that have not been examined previously and may contribute answers to the question of whether it is relevant to

consider a dissociative subtype of PTSD in the context of psychotherapy.

METHODS

PARTICIPANTS

The study included 150 adult women with PTSD, secondary to an index event of a sexual or physical assault in childhood or adulthood, who participated in a larger dismantling study of CPT for PTSD.[11] Participants were randomized to receive CPT ($n = 53$), WA; ($n = 50$), or the CPT-C ($n = 47$). Sample characteristics and clinical scores for the three treatment conditions are reported in Table 1. With the exception of household income, there were no differences among groups on these variables. All women met *DSM-IV*[26] criteria for PTSD. Dissociation was not an exclusion criterion, nor were any comorbid axis I or II disorders, except current substance dependence or psychosis. Most participants reported multiple episodes of physical and/or sexual assaults (see Table 1).

Of the 150 women in the intent-to-treat (ITT) sample, 24 never returned for the first session of therapy, 126 women completed one or more therapy sessions, and 86 women completed all 12 hr of therapy (i.e. treatment completers), of whom four did not return for the follow-up assessment. There were no significant differences in dropout rates among the three treatment conditions (CPT = 34%, CPT-C = 22%, and WA = 26%). There were 127 women who completed at least one of the posttreatment

TABLE 1. Pretreatment sample characteristics by study condition ($N = 150$)

	CPT ($n = 53$)	WA ($n = 50$)	CPT-C ($n = 47$)	Statistical test F or χ^2
Demographic characteristics				
Age, mean (*SD*)	36.25 (12.55)	34.24 (12.47)	35.83 (12.30)	$F(2, 146) = 0.36$
Education, mean (*SD*)	13.57 (3.11)	13.86 (2.89)	14.04 (2.50)	$F(2, 147) = 0.37$
Race/ethnicity, percentage (*n*)				$\chi^2(8) = 4.80$
White	60.4 (32)	32.0 (31)	63.8 (30)	
African American	34.0 (18)	34.0 (17)	34.0 (16)	
Other	5.6 (3)	4.0 (2)	2.1 (1)	
Household income, percentage (*n*)				$\chi^2(10) = 26.68^{**}$
Less than $5,000	28.8 (15)	6.3 (3)	17.4 (8)	
$5,000–$10,000	15.4 (8)	8.3 (4)	15.2 (7)	
$10,000–$20,000	34.6 (18)	27.1 (13)	13.0 (6)	
$20,000–$30,000	9.6 (5)	16.7 (8)	15.2 (7)	
$30,000–$50,000	9.6 (5)	25.0 (12)	15.2 (7)	
Greater than $50,000	1.9 (1)	16.7 (8)	23.9 (11)	
Years since index event, mean (*SD*)	14.29 (13.98)	14.59 (13.84)	14.82 (15.57)	$F(2, 147) = 0.02$
On psychotropic medicine, percentage (*n*)	32.1 (17)	46.0 (23)	46.8 (22)	$X^2(2) = 2.90$
Interpersonal traumas, percentage (*n*)				
Child physical abuse	73.1 (38)	71.7 (33)	76.6 (36)	$\chi^2(2) = 0.31$
Child sexual abuse	73.1 (38)	83.0 (39)	78.7 (37)	$\chi^2(2) = 1.43$
Adult physical assault	84.6 (44)	82.6 (38)	85.1 (40)	$\chi^2(2) = 0.12$
Adult sexual assault	82.7 (43)	80.4 (37)	78.7 (37)	$\chi^2(2) = 0.25$
Study variables				
CAPS, mean (*SD*)	70.19 (15.50)	70.38 (18.65)	73.87 (21.04)	$F(2, 147) = 0.61$
PDS, mean (*SD*)	29.15 (9.54)	29.35 (9.72)	28.48 (9.51)	$F(2, 144) = 0.11$
BDI-II, mean (*SD*)	27.51 (11.75)	26.31 (10.99)	25.72 (11.33)	$F(2, 144) = 0.32$
MDI dissociation[a], mean (*SD*)	64.23 (20.79)	65.81 (24.77)	61.66 (24.10)	$F(2, 128) = 0.34$
MDI disengagement	14.54 (4.14)	14.95 (4.93)	13.49 (4.42)	$F(2, 128) = 1.18$
MDI depersonalization	8.90 (4.21)	9.69 (4.91)	8.85 (4.94)	$F(2, 128) = 0.43$
MDI derealization	10.69 (4.60)	11.43 (5.10)	9.73 (4.85)	$F(2, 128) = 1.28$
MDI memory disturbance	9.98 (4.33)	10.26 (5.09)	9.51 (4.35)	$F(2, 128) = 0.28$
MDI emotional constriction	13.02 (5.61)	12.48 (6.07)	12.76 (5.86)	$F(2, 128) = 0.10$
MDI multiplicity	7.10 (3.70)	7.00 (2.78)	7.32 (4.64)	$F(2, 128) = 0.08$
TSI dissociation, mean (*SD*)	63.04 (11.99)	64.56 (12.10)	62.53 (12.43)	$F(2, 146) = 0.37$
Caseness for dissociation[b], percentage (*n*)	48.1 (25)	46.0 (23)	40.4 (19)	$\chi^2(2) = 0.62$

Note: CAPS, Clinician Administered PTSD Scale; PDS, Posttraumatic Diagnostic Scale; BDI-II, Beck Depression Inventory, II; MDI, Multiscale Dissociation Inventory; TSI, Trauma Symptom Inventory. [a]MDI dissociation represents the mean score for trauma-specific dissociation on the MDI. [b]Caseness for dissociation was determined using the standard clinical cutoff (a T score of 65 or higher)[32] on the trauma-specific dissociation subscale of the MDI.

$^{**}P < .01$.

assessments regardless of treatment participation, resulting in a 15% study dropout rate.

MEASURES

Interview assessment measures included the *Structured Clinical Interview for DSM-IV Axis I Disorders–Patient Edition* (*SCID-P*)[27] to diagnose major depressive disorder, alcohol dependence, substance dependence, and panic disorder as well as to screen for active psychosis; and the *Clinician Administered PTSD Scale* (*CAPS*),[28] a semi-structured interview for the diagnosis of PTSD that also assesses PTSD severity. Self-report measures included the *Posttraumatic Diagnostic Scale* (*PDS*)[29] and the *Beck Depression Inventory-II* (*BDI-II*)[30] to assess PTSD and depressive symptom severity. Both the PDS and BDI-II were administered weekly as well as during the three major assessment points. Psychometric properties of the aforementioned measures can be found in the parent trial.[11] We included two measures of dissociation. First, we used the Dissociation Scale of the *Trauma Symptom Inventory* (*TSI*).[31] The TSI is a 100-item measure of trauma symptoms and contains 10 clinical scales, including a dissociation subscale. The standard clinical cutoff (a T-score $\geq$ 65)[32] was used to index persistent dissociation on the trauma-specific dissociation scale. Second, we used the 30-item *Multiscale Dissociation Inventory* (*MDI*),[32] which includes a trauma-specific dissociation scale as well as six scales designed to reflect the multifaceted nature of dissociation: disengagement (emotional and cognitive separation from one's immediate environment), depersonalization (alteration in one's perception of one's body or self), derealization (alteration in one's perception of the external world), memory disturbances (loss of memory for specific personal events), emotional constriction (reduced emotionality or diminished emotional responsivity), and multiplicity (the perception or experiences that there exists more than one person or self within one's mind). Cronbach's α values for dissociation measures are presented in Table 2.

PROCEDURES

A full description of the recruitment procedures and interventions are described in the primary outcome paper.[11] Treatment consisted of once or twice weekly sessions for a total of 12 hr of therapy over a 6-week period, depending upon the condition. Participants in each condition were assigned practice assignments between sessions as well as the in-session therapy. As an ITT design, participants who were randomized but never attended or dropped out of therapy were invited to return for follow-up assessments. Women completed self-report measures of PTSD and depression severity at pretreatment, every week during their 6-weeks of treatment, posttreatment, and at 6 month follow-up, resulting in a total of nine assessments. A brief description of the three treatments follows:

CPT. CPT is predominantly a cognitive therapy for PTSD.[33] Session 1 includes psychoeducation about the theory of PTSD, rationale for treatment, and an assignment to write an impact statement about the perceived cause and personal meaning of the index (worst) event. After reading and discussing the meaning of the index trauma in Session 2, patients learn to identify relationships among events, thoughts, and emotions (including worksheets). At the end of Session 3, patients are assigned to write a detailed account of the worst trauma, including sensory details, thoughts, and emotions. Patients are instructed to read the account every day. In Sessions 4 and 5, patients read the trauma accounts aloud to the therapists who assist the patients in processing emotions and challenging maladaptive thoughts about the meaning of the event through the use of Socratic dialogue. Writing about additional traumatic events may occur after Session 5, but the focus of CPT shifts to teaching patients to challenge and change beliefs about the meaning of the event and the implications of the trauma for their lives. Patients are first taught to challenge a thought by asking themselves a series of questions. In Session 6, they are then taught to identify problematic patterns of thinking that have come to represent a style of responding. Beginning with Session 7, patients use worksheets that incorporate the earlier ones and are asked to develop and practice alternative, more balanced thoughts. From Sessions 7–12, patients are asked to focus on one theme each week (safety, trust, power–control, esteem, or intimacy) and correct any overgeneralized beliefs related to that theme. At Session 11, patients are also asked to rewrite their impact statements to reflect their current beliefs, and these revised statements are then used in the final session to evaluate treatment gains and areas in which patients wish to continue working.

CPT-C. The CPT-C protocol is identical to CPT, except for the omission of the detailed written trauma accounts. More trauma-focused worksheets and Socratic dialogue by the therapist are substituted. There is also additional emphasis on cognitive skills, including further applications of worksheets for homework in Sessions 5 and 6.

WA. The configuration of the WA protocol was developed to maintain the integrity for the spirit of the written account procedures from the complete CPT protocol (described above). Specifically, in WA the written accounts are designed to be implemented faithfully to CPT but also modeled after imaginal exposure. Patients write their accounts during sessions. Sessions 1 and 2 are an hour each and consist of an overview of the treatment, education regarding PTSD, instructions regarding subjective units of distress (SUDS) and script construction. During the remaining five sessions (2 hr each), patients briefly meet with the clinician and then spend 45–60 min writing about their index trauma. Patients provide SUDS ratings at the beginning and at the end of the writing assignment, as well as ratings of strongest emotions. After completing the written assignment, the patient reads the account to the therapist. Therapists make nondirective and supportive comments, facilitate emotional processing, and provide education, but do *not* conduct any cognitive therapy or try to challenge the patient's cognitive distortions. For homework, patients are asked to complete their written account if they did not during the session, read it daily, and record SUDS ratings. If they complete their work on their index trauma, the therapy may move to focus on additional traumas.

TABLE 2. Bivariate correlations between pre-treatment PTSD and dissociation measures

	Sample size	CAPS	PDS
TSI dissociation ($\alpha = 84$)	149	.49***	.56***
MDI disengagement ($\alpha = 82$)	131	.37***	.46***
MDI depersonalization ($\alpha = 85$)	131	.42***	.41***
MDI-derealization ($\alpha = 88$)	131	.36***	.37***
MDI emotional constriction/numbing ($\alpha = 92$)	131	.42***	.46***
MDI memory disturbance ($\alpha = 85$)	131	.29**	.37***
MDI multiplicity ($\alpha = 87$)	131	.15	.11
MDI total dissociation ($\alpha = 96$)	131	.42***	.46***

Note: CAPS, Clinician Administered PTSD Scale; PDS, Posttraumatic Diagnostic Scale; MDI, Multiscale Dissociation Inventory; α, Cronbach's alpha.
**$P < .001$, ***$P < .000$.

RESULTS

DATA ANALYSES

We obtained and began to use one of the dissociation measures, the MDI, after data collection had begun, so this measure was not administered to the first 19 participants enrolled (5 CPT, 8 WA, and 6 CPT-CT). Therefore, the sample size for the analyses examining the MDI as a predictor of treatment response was 131. Because multilevel regression is efficient in dealing with missing data, the analyses examining change over time in MDI included 147 (out of 150) participants who completed the MDI on at least one assessment occasion. All other analyses included the entire ITT sample.

Simple bivariate correlations were conducted first to examine the relationship between PTSD and dissociation at pretreatment. Hierarchical Linear and Nonlinear Modeling (HLM6)[34] was used to conduct multilevel regression analyses. Time was modeled using dummy-coded variables to examine change over time in dissociation, which was assessed at the three primary assessments, and whether this change varied as a function of treatment condition. This model is analogous to repeated measures ANOVA but capitalizes on the benefits of multilevel regression (e.g. efficiency with handling missing data). Growth curve modeling (GCM) within a multilevel regression framework[35,36] was used to examine change over time in PTSD symptoms for all nine assessment occasions and whether change over time in PTSD symptoms varied as a function of dissociation levels at pretreatment. We capitalized on multilevel regression GCM's flexibility in how time can be modeled. Our primary time variable began at zero (pretreatment assessment) and increased by one for subsequent assessments. In treatment studies, the completion of a treatment session is more important in terms of change over time in the outcome than just the mere passage of time. The primary disadvantage is that this approach does not account for any variation in the timing of assessments, both between assessment points and across individuals. Therefore, we controlled for the variability in the timing of assessments by including the number of days since pretreatment assessment as a time varying covariate. Treatment condition was analyzed using dummy-coded variables.[37] Dissociation variables were mean centered, and product terms were used to evaluate dissociation × treatment condition interactions.[37] We used full maximum likelihood estimation and change in the log-likelihood-based deviance statistic (Δdev) to assess the statistical significance of the joint impact of multiple variables. HLM does not produce standardized coefficients, so we report squared partial correlation coefficients as an index of effect size. Kirk[38] suggests .01, .06, and .14 for small, medium, and large effect sizes, respectively. ANOVA with the TSI dissociation scale as the outcome and MANOVA with the MDI subscales as outcomes were also conducted to see if those who never started therapy or stopped after only one session had higher levels of dissociation than those who received more therapy.

RESULTS OF ANALYSES

Correlations between PTSD and dissociation severity are presented in Table 2. PTSD and dissociation severity were positively correlated, with the exception of the multiplicity scale which was not associated with PTSD severity. Total MDI score was strongly correlated with TSI dissociation ($r = .67$, $P < .001$), whereas correlations among MDI subscales ranged from .35 (multiplicity and disengagement) to .84 (derealization and depersonalization). Results of analyses examining change over time in dissociation are summarized in Table 3. Significant time effects emerged for each dissociation variable, with most effect sizes in the large range. All dissociation variables exhibited significant decreases from pre to posttreatment with no additional change from posttreatment to the 6-month follow-up. No significant time × treatment condition interactions emerged, suggesting that change in dissociation did not differ as a function of treatment condition.

Preliminary GCMs suggested that modeling time using linear and quadratic (i.e. squared) session number fit the data best. This model depicted a curvilinear pattern of change with large initial decreases in PTSD that flattened out over subsequent sessions. To depict the nature of the effects, follow-up analyses with time modeled as the natural log of session number were conducted. The advantage of the natural log model, an alternative procedure to depict nonlinear change, is that it produces only one change parameter, which assists in probing significant group × dissociation × time interactions.

Preliminary GCMs indicated that initial status and change over time in PDS were significantly correlated, such that higher initial PTSD scores were associated with larger decreases in PTSD. We controlled for initial PTSD levels by including CAPS severity scores as a covariate of all change parameters to ensure that any effect of dissociation was not an artifact stemming from the combination of the relationship between dissociation and PTSD at pretreatment and regression toward the mean.

As shown in Table 4, there were no statistically significant dissociation × time interactions, indicating that dissociation did not have a meaningful impact on PTSD treatment response when averaged across treatment conditions. However, significant dissociation × treatment × time three-way interactions emerged for five of the dissociation variables (TSI dissociation total scores, TSI dissociation caseness, MDI total, MDI disengagement, MDI depersonalization); the MDI derealization × treatment condition × time interaction approached statistical significance ($P = .06$). These significant three-way interactions suggest that dissociation had a different impact on PTSD treatment response as a function of treatment condition. Examining the GCM coefficients in Table 3 reveals that the differences were between CPT

TABLE 3. Change over time in dissociation

Variable	*n*	Pretreatment *M* (*SD*)		Posttreatment *M* (*SD*)		6-month follow-up *M* (*SD*)		r^2 Time	Δdev ($df = 2$) Time	*P* Time	Δdev ($df = 2$) Time × con	*P* Time × con
TSI dissociation	150	63.93	(24.49)	49.25	(19.07)	47.05	(20.69)	.25	69.79	.00	5.66	.23
MDI total	147	63.93	(22.96)	49.25	(18.91)	47.05	(20.61)	.25	69.79	.00	5.66	.23
MDI disengagement	147	14.34	(4.48)	10.98	(4.39)	10.82	(4.53)	.26	70.20	.00	6.43	.17
MDI depersonalization	147	9.14	(4.64)	6.90	(3.38)	6.80	(4.24)	.16	41.74	.00	3.15	.53
MDI derealization	147	10.63	(4.83)	7.84	(4.00)	7.64	(4.13)	.21	55.86	.00	3.35	.50
MDI Emot. Constriction	147	12.76	(5.78)	9.66	(5.30)	8.79	(5.24)	.18	49.94	.00	6.37	.17
MDI Mem. Disturbance	147	9.92	(4.55)	7.94	(3.71)	7.44	(3.49)	.14	37.43	.00	6.17	.19
MDI multiplicity	147	7.14	(3.73)	5.92	(2.25)	5.81	(2.35)	.07	21.21	.00	1.82	.77

Note: MDI, Multiscale Dissociation Inventory; TSI, Trauma Symptom Inventory; con, condition; Δdev, change in the deviance statistic.

and CPT-C; however, the WA group did not differ significantly from either of the two conditions in terms of the impact of dissociation on PTSD treatment response.

Figure 1 depicts the nature of this interaction for TSI dissociation (TSI DIS) as a moderator of treatment response by examining trajectories at 1 *SD* above (1a) and 1 *SD* below (1b) the mean of TSI DIS. Figure 1 indicates that CPT-C and CPT differed in how dissociation affected treatment response. We further explored the nature of this interaction by examining the regression equation at different levels of TSI DIS. These probes revealed that at levels of dissociation around the mean, the trajectories of the CPT-C and CPT conditions were very similar. Beginning around 1 *SD* below the mean (57.33), the trajectories of CPT and CPT-C began to differ such that CPT-C was associated with a trajectory characterized by initial large decreases in PTSD that flattened out over time, whereas CPT was associated with a more gradual, steady decrease that was initially smaller than CPT-C and did not exhibit the degree of deceleration as the CPT-C condition. This difference between the two conditions became more pronounced at lower levels of TSI DIS. In contrast, beginning at about 2 *SD*s above the mean of TSI DIS (87.63), the trajectories of the CPT and CPT-C conditions began to differ in the opposite manner. CPT initially exhibited larger decreases that flattened out overtime, whereas CPT-C exhibited smaller decreases initially that did not flatten out overtime. However, at no levels of TSI DIS did the CPT-C exhibit significant differences at the posttreatment or six-month follow-up assessments. Therefore, these differences at low and high levels of TSI DIS were in *rate* of change and not in overall amount of change. The CPT-C condition produced faster change at low dissociation levels and CPT producing faster change at high levels of TSI DIS.

MDI depersonalization was the MDI scale associated with the largest three-way interaction. Figure 2 depicts the nature of the three-way treatment condition × MDI depersonalization × time interaction with the data depicted at 1 *SD* below the mean (2a) and 1 *SD* above the mean (2b) on depersonalization severity. The nature of this effect was similar to the interaction involving TSI DIS, but more pronounced, particularly the differences between the two conditions at high levels of depersonalization. At the mean level of depersonalization (9.14), CPT and CPT-C did not differ in change in PDS scores or at posttreatment and 6-month follow-up levels of PDS. At a half *SD* below the mean (MDI depersonalization = 6.81) the trajectories began to differ mirroring the results described above at low levels of TSI DIS. At even the lowest levels of MDI depersonalization, the two groups did not differ on PTSD levels at the posttreatment or 6-month follow-up assessment. At higher levels of MDI depersonalization, beginning at about 1 *SD* above the mean (13.79), the trajectories began to differ in a manner similar to the effect described for TSI DIS. However, at high levels of MDI depersonalization, the posttreatment PDS differences between CPT and

TABLE 4. Summary of growth curve analyses examining dissociation × time two-way interactions and treatment condition × dissociation × time three-way interactions

Analysis	Variable	Power polynomials			ln session			
		β	t	pr^2		β	t	pr^2
1a: Session × TSI-DIS total (T-score)								
		$\Delta DEV\ (2) = 5.70, P = .057$						
	Lin session × dissociation	0.02	1.10	.01				
	Quad session × dissociation	0.01*	2.21	.03				
1b: Session × TSI DIS total × treatment condition								
		$\Delta DEV\ (4) = 9.96, P < .05$						
	Lin session × dissociation				ln session × dissociation			
	CPT	−0.01[a]	−0.48	.00	CPT	−0.17[a]*	−2.26	.01
	WA	0.03	0.93	.01	WA	−0.05	−.44	.00
	CPT-C	0.08[b]*	2.55	.04	CPT-C	0.17[b]	1.87	.00
	Quad session × dissociation							
	CPT	0.02	1.95	.03				
	WA	0.02*	2.22	.03				
	CPT-C	0.00	0.59	.00				
2a: Session × TSI DIS caseness								
		$\Delta DEV\ (2) = 5.70, P = .057$						
	Lin session × dissociation	0.05	1.10	.01				
	Quad session × dissociation	0.03*	2.21	.03				
2b: Session × TSI DIS caseness × treatment condition								
		$\Delta DEV\ (4) = 9.96, P < .05$						
	Lin session × dissociation				ln session × dissociation			
	CPT	−0.03[a]	−0.48	.00	CPT	−0.77	−0.36	.00
	WA	0.07	0.93	.01	WA	−0.37	−0.16	.00
	CPT-C	0.15[b]*	2.55	.04	CPT-C	4.85*	2.23	.01
	Quad session × dissociation							
	CPT	0.05	1.95	.03				
	WA	0.03*	2.22	.03				
	CPT-C	0.01	0.59	.00				
3a: Session × MDI total								
		$\Delta DEV\ (2) = 4.19, ns$						
	Lin Session × dissociation	0.02	1.34	.01				
	Quad ses × dissociation	0.01	1.62	.02				
3b: Session × MDI total × treatment condition		$\Delta DEV\ (4) = 11.19, P < .05$						
	Lin session × dissociation				ln session × dissociation			
	CPT	−0.01	−0.39	.00	CPT	−0.10[a]	−1.78	.02
	WA	0.03	1.69	.02	WA	0.04	0.73	.00
	CPT-C	0.03	1.48	.02	CPT-C	0.09[b]*	2.26	.04
	Quad session × dissociation							
	CPT	0.01*	2.22	.04				
	WA	0.01	1.53	.02				
	CPT-C	0.00	−0.04	.00				

TABLE 4. Continued

Analysis	Variable	Power polynomials			ln session			
		β	t	pr^2		β	t	pr^2
4a: Session × MDI disengagement (DIS)								
		$\Delta DEV\ (2) = 2.87$, *ns*						
	Lin session × dissociation	0.00	− 0.33	.00				
	Quad session × dissociation	0.04	0.52	.00				
4b: Session × MDI DIS × treatment condition								
		$\Delta DEV\ (4) = 10.43$, $P < .05$						
	Lin session × dissociation				ln session × dissociation			
	CPT	− 0.07	− 0.57	.00	CPT	− 0.71*	− 2.36	.04
	WA	0.08	0.89	.01	WA	0.00	0.00	.00
	CPT-C	0.10	0.84	.01	CPT-C	0.23	0.91	.01
	Quad session × dissociation							
	CPT	0.08*	2.01	.03				
	WA	0.00	0.09	.00				
	CPT-C	0.03	1.36	.01				
5a: Session × MDI derealization (DER)								
		$\Delta DEV\ (2) = 5.06$, *ns*						
	Lin session × dissociation							
	Quad session × dissociation							
5b: Session × MDI DER × treatment condition		$\Delta DEV\ (4) = 9.10$, $P = .06$						
	Lin session × dissociation				ln session × dissociation			
	CPT	0.00	− 0.02	.00	CPT	− 0.41[a]	− 1.56	.02
	WA	0.11	1.29	.01	WA	0.05	0.18	.00
	CPT-C	0.17	1.86	.03	CPT-C	0.50[b]*	2.64	.05
	Quad session × dissociation							
	CPT	0.06*	2.19	.04				
	WA	0.04	1.71	.02				
	CPT-C	0.00	− 0.19	.00				
6a: Session × MDI depersonalization (DEP)								
		$\Delta DEV\ (2) = 3.94$, *ns*						
	Lin session × dissociation	0.09	1.24	.01				
	Quad session × dissociation	0.03	1.61	.02				
6b: Session × MDI DEP × treatment condition		$\Delta DEV\ (4) = 22.54$, $P < .001$						
	Lin session × dissociation				ln session × dissociation			
	CPT	− 0.08	− 0.59	.00	CPT	− 0.85[a]*	− 2.41	.04
	WA	0.13	1.34	.01	WA	0.25[b]	1.01	.01
	CPT-C	0.19*	1.98	.03	CPT-C	0.61[b]*	3.25	.08
	Quad session × Dissociation							
	CPT	0.10[a]*	2.10	.03				
	WA	0.01	0.69	.00				
	CPT-C	− 0.01[b]	− 0.38	.00				
7a: Session × MDI emotional constriction (EC)								
		$\Delta DEV\ (2) = 1.70$, *ns*						
	Lin session × dissociation	0.04	0.52	.00				
	Quad session × dissociation	0.03	1.72	.02				

TABLE 4. Continued

Analysis	Variable	Power polynomials β	Power polynomials t	Power polynomials pr^2	ln session β	ln session t	ln session pr^2
7b: Session × MDI EC × treatment condition							
		$\Delta DEV\ (4) = 7.61$, *ns*					
	Lin session × dissociation				ln session × dissociation		
	CPT	−0.08[a]	−1.33	.01	CPT −0.18	−0.83	.01
	WA	0.14	1.75	.02	WA 0.18	0.92	.01
	CPT-C	0.10[b]	1.44	.02	CPT-C 0.06	0.27	.00
	Quad session × dissociation						
	CPT	0.00	0.04	.00			
	WA	0.02	1.32	.01			
	CPT-C	0.02	1.29	.01			
8a: Session × MDI memory disturbance (Mem)							
		$\Delta DEV\ (2) = 1.40$, *ns*					
	Lin session × dissociation	0.04	0.62	.00			
	Quad session × dissociation	0.02	1.03	.01			
8b: Session × MDI Mem × treatment condition							
		$\Delta DEV\ (4) = 5.74$, *ns*					
	Lin session × dissociation				ln session × dissociation		
	CPT	−0.04	−0.32	.00	CPT −0.36[a]	−1.55	.02
	WA	0.11	1.09	.01	WA 0.04	0.16	.00
	CPT-C	0.06	0.68	.00	CPT-C 0.33[b]	1.54	.02
	Quad session × dissociation						
	CPT	0.04	1.22	.01			
	WA	0.04	1.52	.02			
	CPT-C	−0.02	−0.91	.01			
9a: Session × MDI multiplicity (MUL)							
		$\Delta DEV\ (2) = 4.77$, *ns*					
	Lin session × dissociation	0.11	1.33	.01			
	Quad session × dissociation	−0.01	−0.37	.01			
9b: Session × MDI MUL × treatment condition							
		$\Delta DEV\ (4) = 7.11$, *ns*					
	Lin session × dissociation				ln session × dissociation		
	CPT	0.06	0.68	.00	CPT 0.05	0.11	.00
	WA	0.33	2.28	.00	WE 0.72*	2.57	.05
	CPT-C	0.02	0.24	.04	CT 0.58*	2.32	.04
	Quad session × dissociation						
	CPT	0.02	0.67	.00			
	WA	0.03	0.59	.00			
	CPT-C	−0.04	−3.76	.10			

Note: *$P < .05$; Superscripts [a]and [b] have been used to denote significant difference among treatment conditions. TSI-DIS, the dissociation subscale of the Trauma Symptom Inventory; MDI, Multiscale Dissociation Inventory; ln session, the natural log transformation of session number; *b*, unstandardized regression coefficient, *t*, t-statistic, pr^2, squared partial correlation coefficient; Δ*dev*, change in the deviance statistic.

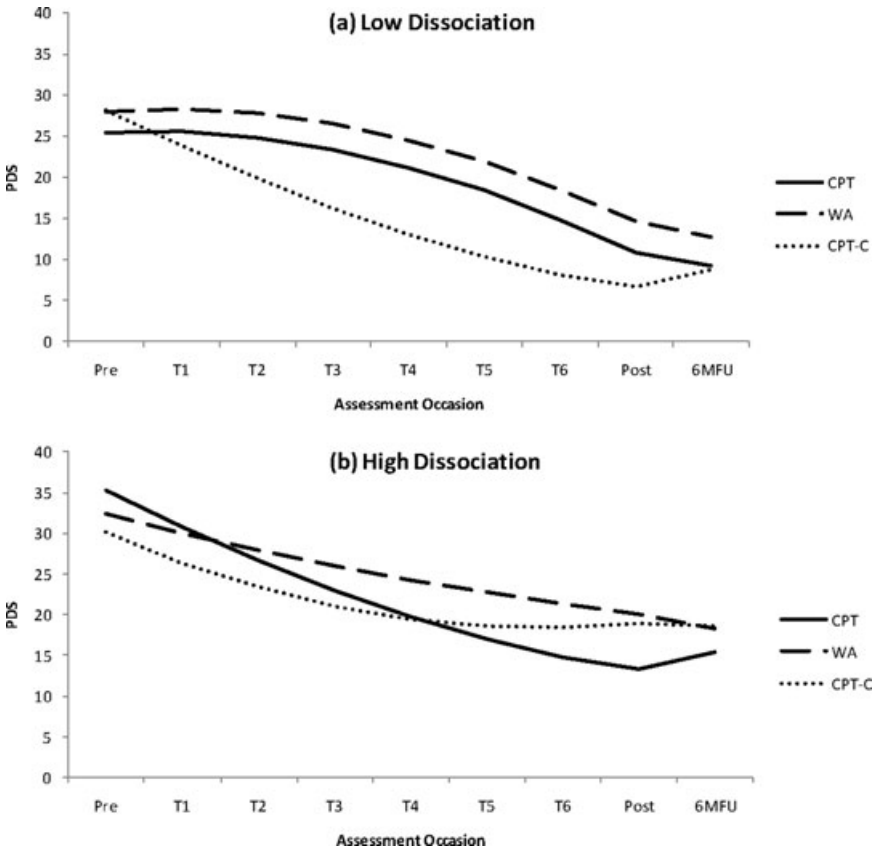

Figure 1. Treatment condition × TSI DIS × time interaction. la depicts change over time on the PDS as a function of treatment condition at 1 *SD* below the mean level of TSI DIS. lb depicts change in PDS as a function of treatment condition at 1 *SD* above the mean level of TSI DIS.

CPT-C started to become meaningful. For example, at 2 *SD*s above the mean level of MDI depersonalization (18.45), the regression equation predicted posttreatment PDS values of 7.75 and 27.93 for the CPT and CPT-C conditions, respectively, which represents a 1.12 *SD* difference. This difference increased as MDI depersonalization values increased. Therefore, at very high levels of depersonalization, the treatment conditions differ in both rate and overall amount of PTSD symptom change, with the CPT condition exhibiting faster and larger decreases.

In two of the three therapy conditions, the patient discussed the impact of the index trauma on their lives and why they think the traumas occurred in the early sessions; therefore, we did not consider those who had two or more sessions to be untreated. Also, results from the parent study revealed that participants in CPT-C experienced clinically significant improvement by the fourth session,[11] and thus, drop-out from the protocol is not necessarily considered to be a problem. As such, we divided the sample into those who had one session or less as untreated ($N = 36$) and those with two or more sessions as treated ($N = 112$). An ANOVA on the TSI DIS measure was nonsignificant, $F(1,147) = 1.5$, *ns*. A MANOVA on the subscales of the MDI was also nonsignificant, $F(5,125) = .49$, Pillai's Trace = .89, *ns*. Additionally, individual analyses of the subscales were all nonsignificant, indicating that pretreatment level of dissociation was unrelated to starting therapy.

DISCUSSION

This study evaluated whether: (1) level of dissociation at pretreatment predicts PTSD treatment outcomes; (2) dissociation improves over the course of PTSD treatment; and (3) whether there are differences in the impact of dissociation on PTSD treatment response across the three treatment conditions in the trial. Overall, pretreatment levels of dissociation did not impact change in PTSD symptoms. However, significant dissociation × time × treatment condition interactions emerged such that, across dissociation subscales, individuals with high dissociation who were receiving CPT had better outcomes than those receiving CPT-C. Of note, these findings were particularly pronounced for individuals reporting high levels of pretreatment depersonalization. In contrast, participants with lower levels of dissociation who were in the CPT-C condition evidenced more efficient treatment response relative to those with lower levels of dissociation in the CPT condition.

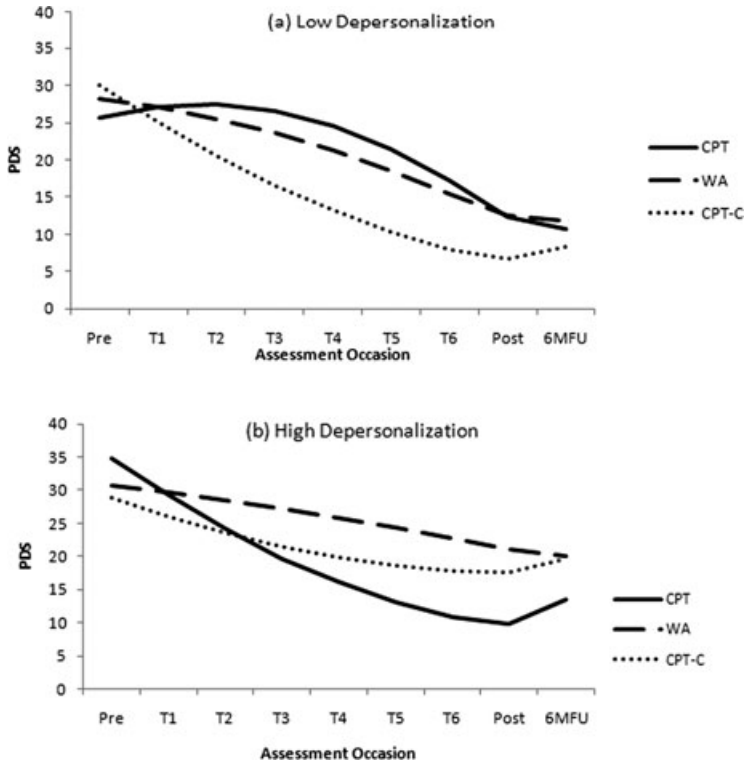

Figure 2. Treatment condition × MDI depersonalization × time interaction. 2a depicts change over time on the PDS as a function of treatment condition at 1 *SD* below the mean level of MDI depersonalization. 2b depicts change in PDS as a function of treatment condition at 1 *SD* above the mean level of MDI depersonalization.

These findings suggest that the combination of writing the trauma account and cognitive therapy provided by CPT was most effective for patients exhibiting high levels of dissociation. High levels of dissociation at pretreatment may be reflective of dissociation levels during the trauma, which likely resulted in fragmented memories for the traumatic event. It appears that psychotherapy that assists the patient in reconstructing the traumatic event and then focuses on the meaning of the event is optimal for PTSD treatment response for highly dissociative patients. In CPT, writing the trauma account may assist in reconstructing the fragmented memory and placing the event into context, allowing for emotional processing as well as more balanced trauma-related cognitions. However, if only writing the account was sufficient for patients with high levels of dissociation, then the WA treatment should have been the most efficient because WA provides more opportunities to write and read the account. These findings indicate that the combination of reconstructing the traumatic event paired with Socratic examination of the patient's interpretation of the event is the more efficient method than CPT-C or WA for those with high levels of dissociation. Thus, providers should assess specific dissociation symptoms and consider matching treatment accordingly. For example, the current findings suggest the utility of offering CPT to patients with high levels of dissociation, especially depersonalization.

These findings also suggest that patients who have low levels of dissociation do not need to write a trauma account, and in fact, do better with a cognitive therapy that focuses exclusively on thinking and talking *about* the trauma rather than reconstructing the sensory experience of event. Patients who do not dissociate probably have good recall of the traumatic event and may be distracted by parts of the event that are distressing but not maintaining their PTSD (e.g. "the smell of alcohol on his breath" rather than "it is all my fault that it happened"). In the parent study,[11] it was found that CPT-C appeared to be more efficient without the written accounts and clinical change occurred earlier in treatment than in either CPT or WA. That study did not examine the effects of dissociation, which was equally distributed across the three treatment conditions. The current findings extend this previous work by demonstrating that the relative efficiency of CPT-C is particularly important when level of dissociation is taken into account.

Regarding the question of whether dissociation improves during PTSD treatment, the current study found that dissociation decreased significantly from pre to posttreatment, and these decreases were maintained at the 6-month follow up. These results did not differ by treatment condition, suggesting that cognitive therapy and exposure-type therapy have a positive impact on dissociation. These results are consistent with those of Cloitre et al.[21] and Chard[23] in that they found significant

decreases in dissociation following modified exposure or modified CPT. Treating the dissociation in advance of trauma work does not appear to be necessary.

As noted above, however, results across studies have been inconsistent, with some demonstrating significant reductions in dissociation following treatment[20,21,23] and others finding no changes.[18,19] It is important to note that most previous studies used the DES,[7] which provides a total score of dissociation but is weighted toward dissociative disorders. The current study provides a more nuanced evaluation of dissociation as a treatment outcome. Although this hypothesis warrants future testing, it is possible that the TSI DIS and the subscales of the MDI represent more ongoing dissociation that is associated with PTSD, rather than symptoms of dissociative disorders. Additionally, the study design and data analytic methods used in this study allowed us to elucidate the differential effects of therapies on dissociation in a way that was not possible in previous studies.

The current study was limited by the absence of a control group, because the focus was on evaluating primary components of CPT. Further, subsamples of various ethnic groups were too small to make statistical comparisons. Thus, it is unclear to what extent these results would generalize to diverse groups of women or to men. The strength of this study is that it was conducted with interpersonal trauma survivors, a population that may be particularly prone to dissociation; however, it remains unknown whether the current findings would generalize to survivors of other forms of trauma, such as combat, natural disasters, or vehicular accidents.

The current findings support the inclusion of patients with even high levels of dissociation in PTSD treatment. Moreover, PTSD patients with high levels of dissociation may experience particularly positive treatment response in CPT relative to cognitive therapy or exposure-type therapy alone; whereas patients who exhibit lower levels of dissociation are treated most efficiently with CPT-C. To continue to develop this essential line of research, future studies are needed to examine specific mechanisms of change in dissociation during various forms of psychotherapy, including but not limited to the interventions examined in this study. The differential treatment findings also support the possibility of a dissociative subtype of PTSD patients who respond to therapy differently than those with lower levels or no dissociation.

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