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Couple Treatment for PTSD: Partner Outcomes and Partner and Patient Influences on Treatment Outcomes

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COUPLE TREATMENT FOR PTSD: PARTNER OUTCOMES AND PARTNER AND
PATIENT INFLUENCES ON TREATMENT OUTCOMES

by

Philippe Shnaider

B.A., York University, June 2010

A thesis

presented to Ryerson University

in partial fulfillment of the
requirements for the degree of

Master of Arts

in the Program of

Psychology

Toronto, Ontario, Canada, 2012

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Couple Treatment for PTSD: Partner Outcomes and Partner and Patient Influences on Treatment

Outcomes

Master of Arts 2012

Philippe Shnaider

Psychology

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Abstract

A growing body of literature has documented interpersonal factors associated with the occurrence and treatment of posttraumatic stress disorder (PTSD). Among these factors, intimate partners' psychological functioning has consistently been found to be associated with patients' PTSD severity. The present study investigated intimate partners' psychological functioning outcomes in a sample of 40 partners of individuals with PTSD within a randomized waitlist controlled trial of cognitive-behavioural conjoint therapy for PTSD. In addition, models of influence from partners to patients and vice versa were examined. More specifically, these models investigated the influence of pretreatment functioning and symptom change on treatment outcomes. There were no significant differences between active treatment and waitlist in intimate partners' psychological functioning at posttreatment. Furthermore, neither partners' psychological functioning, nor patients' PTSD symptoms, influenced the others' treatment outcomes. Findings are discussed with a focus on guiding future research on partners' psychological functioning in the context of PTSD.

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Couple Treatment for PTSD: Partner Outcomes and Partner and Patient Influences on Treatment Outcomes

In North America, approximately 75% of the population has been exposed to a traumatic event and about 10% of the population is diagnosed with posttraumatic stress disorder (PTSD) at some point in their lifetime as a result (Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995; Van Ameringen, Mancini, Patterson, & Michael, 2008). Posttraumatic stress disorder is a condition known to be highly associated with intimate relationship discord, as well as general psychological distress, defined as elevations in psychological symptomatology, in romantic partners of individuals living with this condition (for reviews, see Renshaw, Blais, & Caska, 2011; Taft, Watkins, Stafford, Street, & Monson, 2011). Descriptive studies have consistently documented an association between PTSD symptoms in one partner and general psychological distress in their significant other (e.g., Beckham, Lytle, & Feldman, 1996; for a review see Renshaw et al., 2011). Furthermore, a few studies have documented that social support, and interpersonal relationship and family functioning affects individual PTSD treatment outcomes (Monson, Rodriguez, & Warner, 2005; Price, Gros, Strachan, Ruggiero, & Acierno, 2011; Tarrrier, Sommerfield, & Pilgrim, 1999). Yet, the effects of PTSD treatments on partners' well-being and the potential influence of partners' psychological functioning on patients' PTSD treatment outcomes, and vice versa, are relatively unknown. Thus, the purpose of this study was to examine whether partners' psychological functioning improved in a conjoint therapy for PTSD, and to examine the potential influence of partners' and patients' respective psychological functioning and PTSD symptoms on each others' treatment outcomes.

Interpersonal Nature of PTSD

Although PTSD has traditionally been conceptualized as an individual disorder with intrapersonal causes and effects, a growing body of literature has demonstrated that the suspected risk factors and consequences of PTSD extend to interpersonal factors as well. For example, two meta-analyses examining correlates of PTSD found that lack of social support (Brewin, Andrews, & Valentine, 2000) and perceived social support (Ozer, Best, Lipsey, Weiss, 2003) were among the strongest associated factors, such that individuals who reported greater PTSD symptomatology tended to report poorer rates of social support. Moreover, a recent meta-analysis examining the association between PTSD and intimate relationship problems concluded that the average strength of the association between PTSD symptoms and relationship problems was of medium size. These problems were present across both sexes, military and civilian samples, as well as across different countries (Taft et al., 2011). More specifically, a number of studies have documented interpersonal problems associated with PTSD to include increased interpersonal violence and hostility, and decreased sexual intimacy, sociability, expressiveness, and familial cohesion (e.g., Beckham, Feldman, Kirby, Hertzberg, & Moore, 1997; Beckham, Roodman, et al., 1996; Roberts et al., 1982; Solomon, Mikulincer, Fried, & Wosner, 1987).

Significant Others' Psychological Distress

Building from the research on interpersonal functioning and PTSD, studies have begun to more closely examine the psychological functioning of romantic partners of individuals with PTSD and the relationship of this functioning to PTSD symptomatology (e.g., Calhoun, Beckham, & Boswarth, 2002; Dekel, Solomon, & Blicch, 2005; Renshaw & Campbell, 2011). Calhoun et al. (2002) found that romantic partners of Vietnam combat veterans with PTSD reported higher levels of depressive, anxiety, and obsessive-compulsive symptoms compared with partners of Vietnam combat veterans without PTSD, a finding that has been supported by

several others (Dekel, 2007; Manguno-Mire et al., 2007; Mikulincer, Florian, & Solomon, 1995; Westerink & Giarratano, 1999). In addition, these individuals have been shown to exhibit greater levels of hostility, higher rates of somatic complaints and sleep problems, lower levels of happiness and life satisfaction, higher levels of loneliness and social dysfunction, as well as a greater degree of general emotional distress in comparison with partners of individuals without PTSD (Calhoun et al., 2002; Dekel, et al., 2005; Dirkzwager, Bramsen, Adèr, & van der Ploeg, 2005; Jordan et al., 1992; Mikulincer et al., 1995; Waysman, Mikulincer, Solomon, & Weisenberg, 1993; Westerink & Giarratano, 1999). Beyond the presence of greater psychological distress among partners of individuals with PTSD, research has supported that the degree of partners' psychological distress is positively associated with the severity of their partners' PTSD symptoms (Beckham, Lytle et al., 1996; Dekel, 2007; Gallagher, Riggs, Byrne, & Weathers, 1998; Glenn et al., 2002). For example, in a cross-sectional study of male prisoners of war and their female partners, the prisoners of war's PTSD symptoms significantly predicted partners' distress in a positive direction (Dekel, 2007). Similarly, using correlational analyses in a sample of male Vietnam veterans and their female partners, Gallagher et al. (1998) found significant cross-sectional associations between reports of Veterans and partners' own PTSD symptoms.

Most of the extant literature examining the association between partners' psychological functioning and patients' PTSD symptoms has been cross-sectional in nature. However, one longitudinal study provides evidence that these variables are associated over time. In a sample of male Vietnam veterans with PTSD and their partners, Beckham, Lytle et al. (1996) demonstrated that veterans' PTSD symptoms were positively associated with partners' psychological distress across an 8-month time interval. Specifically, they found significant associations between

veterans' PTSD symptoms and partners' overall distress and depressive symptomatology over time. However, researchers in this study did not test the effect of partners' psychological distress on patients' symptomatology, thereby conceptualizing the association between these variables as unidirectional. Although it is possible for partners' psychological distress to result from patients' PTSD, no studies have conclusively established the directionality of this association.

The Role of Significant Others in PTSD Treatment

In their review of the impact of PTSD on veterans and their families, Galovski and Lyons (2004) indicated that research to date had not identified the reduction of partners' psychological distress as a treatment goal in PTSD treatments. Thus, PTSD treatments that simultaneously aim to reduce partners' distress and patients' PTSD symptoms would represent a unique category of treatments in the existing literature. Of the seven prior uncontrolled (Cahoon, 1984; Devilly, 2002; MacIntosh & Johnson, 2008; Monson et al., 2011; Monson, Schnurr, Stevens, & Guthrie, 2004; Sautter, Glynn, Thompson, Franklin, & Han, 2009; Schumm, Fredman, & Monson, 2011) and two controlled (Glynn et al., 1999; Sweany, 1987) treatment trials that have investigated conjoint treatments for PTSD, only a small subset have examined changes in partners' psychological functioning.

In Devilly's (2002) uncontrolled study of a 5-day residential treatment program that included female partners of male Australian veterans, there were statistically significant, small-to-medium, within group effect size improvements in partners' depression and anxiety scores at posttreatment, 3-month, and 6-month follow-up. Similarly, in Monson et al.'s (2004) uncontrolled trial of cognitive-behavioural conjoint therapy (CBCT) for PTSD (Monson & Fredman, 2012), Monson, Stevens, and Schnurr (2005) found significant and large within group effects on general anxiety ($d = 1.29$) and social functioning ($d = 1.61$) in a sample of wives of

male Vietnam veterans. Although improvements in wives' depression scores were not statistically significant, potentially due to the small sample size ($n = 7$), a medium effect size improvement was found ($d = .66$). However, Monson and colleagues' (2011) uncontrolled trial of CBCT, in a community sample, did not detect partner improvements. Interestingly, partners evidenced significant and large increases in anger expression across treatment ($d = -1.64$). The authors suggested that this increase in anger expression may have signaled that partners were more open and able to express their negative feelings as result of treatment. In a sample of 14 combat exposed Vietnam veterans and their partners, Sweany's (1987) controlled trial of behavioural couple therapy did not find statistically significant differences between the treatment and control conditions on significant others' depression scores at posttreatment.

In addition to the studies examining partners' treatment outcomes, a few studies have begun to examine the influence of significant others on patients' treatment gains. Prior research has found that poor social support, family functioning, and intimate relationship functioning were associated with less improvement in PTSD and secondary treatment outcomes (i.e., depressive symptoms, anxiety, violence) in both individual and group treatment formats (Monson, Rodriguez et al., 2005; Price et al., 2011; Tarrier et al., 1999). For example, Tarrier et al. (1999) examined the role of expressed emotion, a measure of the quality of the relationship between a patient and their relatives (Leff & Vaughn, 1985), in PTSD symptom change in a randomized controlled trial comparing individual imaginal exposure and cognitive therapy. Expressed emotion was assessed by interviewing a patient's key relative before randomization. Results indicated that higher levels of expressed emotion, denoting poorer family functioning, were associated with less individual improvement in PTSD symptoms. Specifically, elevations on the criticism and hostility scales of the expressed emotion interview tended to have the

greatest influence on PTSD outcomes. In an effectiveness study by Monson, Rodriguez et al. (2005), comparing a skills-focused and trauma-focused group treatment for PTSD, a strong negative relationship was found between pretreatment intimate relationship functioning and violence outcomes for the trauma-focused group. Individuals in the trauma-focused group with higher pretreatment intimate relationship functioning had lower rates of violence at posttreatment, after controlling for pretreatment violence. Together, these studies illustrate the influence that relationships and interactions with significant others have on PTSD treatment outcomes. Yet, there is scant research on the effects of PTSD treatments on partners' mental health outcomes or the reciprocal influence of either partner's change across treatment on the other's treatment outcomes.

Although there are a few studies documenting changes in partners' mental health within dyadic interventions for PTSD, the extent to which these treatments benefit partners' psychological functioning, and the degree to which partners mutually influence each others' symptoms, is not yet known. No studies to date have examined the potential bidirectional influence of individuals' pretreatment functioning, nor the reciprocal influence of changes in individuals' symptomatology across treatment, on their significant others' treatment outcomes. Researchers have, for some time now, indicated that a greater understanding of the association between partners' mental health outcomes and PTSD recovery is sorely needed (Manguno-Mire et al., 2007; Beckham, Lytle et al., 1996). With the documented presence of partners' distress among romantic partners of individuals with PTSD, understanding the role of partners' psychological functioning in PTSD is essential to promoting a fuller understanding of posttrauma recovery. This information can help to further uncover the interpersonal nature of PTSD and can

provide suggestions on how to approach treatment and prevention efforts that are mutually beneficial to the patient, his/her partner, and the relationship they share.

Theoretical Advancements

In tandem with the surge in interventional and naturalistic investigation of significant others' psychological distress and its association to their partners' PTSD, theoretical contributions have recently been made accounting for these relationships, both at the individual and relational level, within romantic dyads. However, only two existing theories have conceptualized the relationship between these individual and relational factors at the same time and as reciprocally related. Cognitive-behavioural interpersonal theory (C-BIT) of PTSD (Monson, Fredman, & Dekel, 2010; Monson, Stevens et al., 2005) is one of these. Rather than construing PTSD as a disorder that develops per se, C-BIT depicts PTSD as a disorder of impeded recovery within a systemic model in which individual level factors within each partner, as well as relational level factors, interact to influence recovery. Similarly, the couple adaption to traumatic stress (CATS) model (Nelson-Goff & Smith, 2005) maintains that individual and relational factors interact to influence reactions to a traumatic event. Given that the current study examines influences of partners within a dyad in the context of CBCT, a therapy developed on the basis of C-BIT, the current focus is directed toward this model.

According to C-BIT, there are cognitive, behavioural, and emotional factors that operate within each individual that can lead to maladaptive coping patterns. In essence, the thoughts, behaviours, and emotions that a traumatized individual has and engages in influence his/her recovery. For example, if a traumatized individual behaviourally avoids reminders of the traumatic event, this can result in the negative reinforcement of trauma-related avoidance behaviour, which consequently hinders the recovery process. In the case of cognitive factors,

beliefs about the trauma and its sequelae, such as believing that a traumatic event happened as punishment for one's wrongdoings, or that one could have changed the outcome of an event if they had only responded differently, can lead to distorted thinking and make it difficult to overcome and move past a traumatic experience.

Similarly, there are cognitive, behavioural, and emotional factors that characterize a traumatized individual's romantic partner that also influence the traumatized individual's recovery. These factors can result from being in relation to the traumatized partner or can be a product of experiences independent of the influence of the partner with PTSD (e.g., preexisting psychopathology, other psychosocial stressors). That is, a partner's thoughts, behaviours, and emotions may result from their traumatized partner's experience of a traumatic event or from preexisting maladaptive patterns, such as those resulting from previous or current mental health issues. An example of the former is when a significant other takes on all driving duties after his/her partner has been in an automobile accident; the latter being a case where a partner suffering from social anxiety does not engage in social situations, thus potentially contributing to the traumatized partner's avoidance in tandem with his or her own avoidance.

In addition, C-BIT purports that each partner's cognitions, behaviours, and emotions interact to affect the relationship milieu shared by the dyad, which in turn creates relational factors that also influence the traumatized individual's recovery and the significant other's mental health functioning. Finally, relational level factors feed back and influence, as well as interact with, individual level factors. These relational factors also influence individual functioning, including PTSD recovery. For example, expressed hostility by one partner toward the other (an individual factor) creates an atmosphere that challenges safety and trust in the relationship (a relational factor), which influences each partner's mental health by confirming or

altering views about the world, oneself, or others (an individual factor). Taken as a whole, C-BIT proposes that each partner's individual cognitive-behavioural-emotional system and his or her shared relational system interact to influence an individual's recovery from PTSD, his/her partner's psychological functioning, and their relationship adjustment.

The Present Study

Although put forth theoretically (Monson et al., 2010; Monson, Stevens et al., 2005), the notion of partners' psychological functioning changing as a result of a PTSD-specific dyadic treatment or influencing PTSD patients' recovery has received little empirical attention in the literature. Consistent with C-BIT, partners' psychological distress may be a significant factor that affects patients' PTSD symptoms and vice versa. The current study sought to investigate partners' psychological functioning outcomes (i.e., depressive, anxiety, and anger symptoms) in the treatment of PTSD within a randomized controlled trial of CBCT, as well as the influence of partners' psychological functioning and patients' PTSD symptoms on one another during treatment.

This research was designed to answer two primary questions: 1) Does partners' psychological functioning improve by virtue of being in a conjoint treatment simultaneously focused on ameliorating PTSD and enhancing relationships? and 2) Do partners' and patients' respective psychological functioning and PTSD symptoms influence one another? The following specific hypotheses were offered in light of the existing literature: 1) Compared with partners waiting for treatment, partners in the CBCT condition will display greater improvements in psychological functioning; 2) Partners' and patients' respective pretreatment psychological functioning and PTSD symptoms would predict the others' symptom change (*cross paths* model; see Figure 1); 3) Partners' and patients' change in psychological functioning and PTSD

symptoms would be reciprocally related to one another (*reciprocal paths* model; see Figure 2);

4) As a further test of hypothesis three, it was predicted that models in which the paths from patient-to-partner or partner-to-patient were constrained to zero (statistically equivalent to eliminating them from the model), would be a worse fit to the data than the reciprocal paths model. Constraining the paths to zero allowed for a test of the notion that the reciprocal paths model best explains the data.

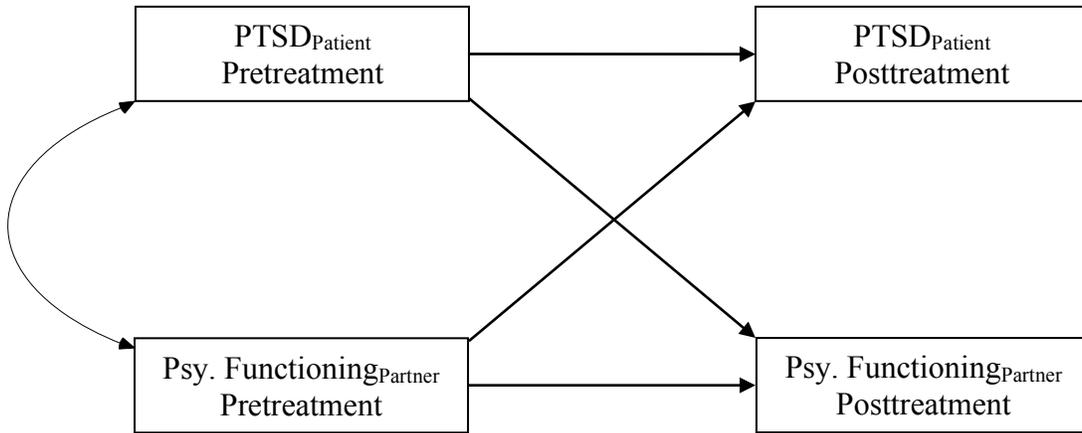


Figure 1. Cross paths model. Pretreatment functioning as predictors of symptom response with treatment. PTSD = Posttraumatic stress disorder; Psy. = Psychological.

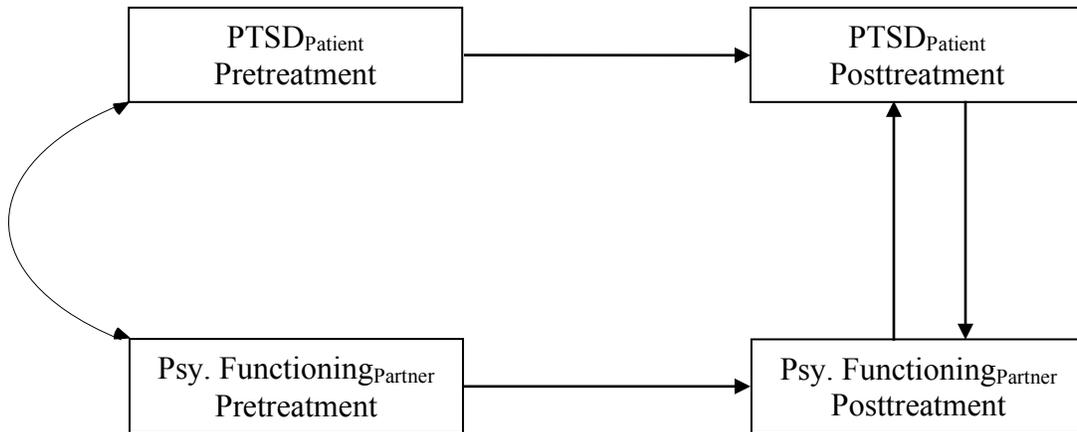


Figure 2. Reciprocal paths model. Symptom changes as predictors of partners' symptom response with treatment. PTSD = Posttraumatic stress disorder; Psy. = Psychological.

Method

Participants

The sample consisted of 40 romantic dyads in which one partner reported symptoms meeting *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision (DSM-IV-TR*; American Psychiatric Association, 2000) diagnostic criteria for PTSD and was recruited from two study sites (a university in Toronto, ON, and an outpatient mental health clinic in Boston, MA). To be eligible to participate, dyads had to be between the ages of 18 and 75 years, with one partner meeting PTSD diagnostic criteria as determined by the Clinician-Administered PTSD Scale (CAPS; Blake et al., 1995) at least 3 months posttrauma. If the patient was prescribed psychoactive medications, the medication regimen had to be stable for a 2-month period prior to participation, and could not be changed throughout the course of the study. All dyads consented to being audiorecorded or videorecorded, as well as agreed to not receive conjoint or individual treatments for PTSD during the course of the study. In cases in which participants were in psychotherapy at study entry, study personnel consulted with the participant's treating clinician to ensure that they were not receiving an evidence-based treatment for PTSD (e.g., prolonged exposure, cognitive-processing therapy, eye movement desensitization and reprocessing); however, participants could receive evidence-based treatments for other disorders. Exclusion criteria included current substance dependence in either member of the dyad, as determined by the Structured Clinical Interview for DSM-IV-Patient Version (SCID-P; First, Spitzer, Gibbon, & Williams, 1995); a current PTSD diagnosis in the partner, as determined by the CAPS; the presence of current uncontrolled psychotic or bipolar disorder, imminent suicidality or homicidality, self-injurious behaviour within the past 6 months in either partner; and severe cognitive impairments in either partner. In addition, dyads were excluded if

there was any severe physical or sexual aggression in the relationship within the past 12 months, as determined by the Revised Conflict Tactics Scales (CTS-2; Straus, Hamby, McCoy, & Sugarman, 1996). Furthermore, partners were not required to be in the distressed range on measures of psychological functioning, nor were patients and partners required to have relationship adjustment scores in the distressed ranged.

Characteristics of the sample can be found in Table 1. The sample consisted of 37 heterosexual couples and three same-sex female couples. The three same-sex female couples were randomized to the CBCT condition. Thus, there were fewer male partners who were randomized to CBCT compared with waitlist. Overall, there were few significant baseline differences between the sites. The Boston site had more partners with a lifetime history of substance use disorders [partners: Boston, $n = 11$ (64.7%); Toronto, $n = 6$ (26.1%); $\chi^2(1) = 5.97$, $p = .02$] more partners with a lifetime history of an anxiety disorder [Boston, $n = 10$ (58.8%); Toronto, $n = 1$ (4.3%); $\chi^2(1) = 14.55$, $p < .001$], and more partners with concurrent comorbid anxiety disorders [Boston, $n = 5$ (29.4%); Toronto, $n = 0$ (0.0%), $\chi^2(1) = 7.73$, $p = .01$]. The Boston site also had more patients currently receiving any additional mental healthcare [Boston, $n = 11$ (64.7%); Toronto, $n = 7$ (30.4%); $\chi^2(1) = 4.64$, $p = .03$].

Table 1

Baseline Characteristics by Condition and Partner.

Characteristic	Patients			Partners		
	CBCT	WL	Total	CBCT	WL	Total
	<i>n</i> = 20 <i>M</i> or <i>no.</i> (<i>SD</i> or %)	<i>n</i> = 20 <i>M</i> or <i>no.</i> (<i>SD</i> or %)	<i>N</i> = 40 <i>M</i> or <i>no.</i> (<i>SD</i> or %)	<i>n</i> = 20 <i>M</i> or <i>no.</i> (<i>SD</i> or %)	<i>n</i> = 20 <i>M</i> or <i>no.</i> (<i>SD</i> or %)	<i>N</i> = 40 <i>M</i> or <i>no.</i> (<i>SD</i> or %)
Age	40.40 (11.27)	33.80 (10.50)	37.10 (11.26)	40.70 (12.52)	34.85 (9.96)	37.78 (11.55)
Male	7 (35.0%)	3 (15.0%)	10 (25.0%)	10 (50.0%) ^a	17 (85.0%) ^b	27 (67.5%)
Non-Caucasian	5 (25.0%)	6 (30.0%)	11 (27.5%)	4 (20.0%)	4 (20.0%)	8 (20.0%)
Married	8 (40.0%)	6 (30.0%)	14 (35.0%)	8 (40.0%)	6 (25.0%)	14 (32.5%)
Military veteran status	6 (30.0%)	3 (7.5%)	9 (22.5%)	1 (5.0%)	0 (0.0%)	1 (2.5%)
Employment (at least part-time)	12 (60.0%)	12 (60.0%)	24 (60.0%)	13 (65.0%)	13 (65.0%)	26 (65.0%)
Relationship Length	8.13 (8.67)	5.43 (5.84)	6.85 (7.50)	8.18 (9.03)	5.37 (5.75)	6.78 (7.61)
Index trauma						
Adult sexual trauma	4 (20.0%)	4 (20.0%)	8 (20.0%)			
Child sexual trauma	3 (15.0%)	8 (40.0%)	11 (27.5%)			
Noncombat physical assault	4 (20.0%)	2 (10.0%)	6 (15.0%)			
Motor vehicle accident	1 (5.0%)	2 (10.0%)	3 (7.5%)			
Witnessing/learning about death/illness	2 (10.0%)	3 (15.0%)	5 (12.5%)			
Combat	2 (10.0%)	0 (0.0%)	2 (5.0%)			
Other	4 (20.0%)	1 (5.0%)	5 (12.5%)			
Years since trauma	16.95 (14.09)	13.48 (13.10)	15.17 (13.52)			
Current Other Axis I Disorder	11 (55.0%)	14 (70.0%)	25 (62.5%)	3 (15.0%)	7 (35.0%)	10 (25.0%)

Mood disorder	7 (35.0%)	9 (45.0%)	16 (40.0%)	1 (5.0%)	2 (10.0%)	3 (7.5%)
Other anxiety disorder	5 (25.0%)	7 (35.0%)	12 (30.0%)	2 (10.0%)	3 (15.0%)	5 (12.5%)
Substance abuse	0 (0.0%)	1 (5.0%)	1 (2.5%)	0 (0.0%)	1 (5.0%)	1 (2.5%)
Other	2 (10.0%)	2 (10.0%)	4 (10.0%)	0 (0.0%)	2 (10.0%)	2 (5.0%)
Lifetime Other Axis I Disorder	19 (95.0%)	20 (100.0%)	39 (97.5%)	15 (75.0%)	13 (65.0%)	28 (70.0%)
Mood disorder	18 (90.0%)	17 (85.0%)	35 (87.5%)	12 (60.0%)	9 (45.0%)	21 (52.5%)
Other anxiety disorder	9 (45.0%)	10 (50.0%)	19 (47.5%)	6 (30.0%)	5 (25.0%)	11 (27.5%)
Substance abuse or dependence	9 (45.0%)	8 (40.0%)	17 (42.5%)	10 (50.0%)	7 (35.0%)	17 (42.5%)
Other	5 (25.0%)	3 (15.0%)	8 (20.0%)	1 (5.0%)	2 (10.0%)	3 (7.5%)
Any mental healthcare utilization	12 (60.0%)	11 (55.0%)	23 (57.5%)	10 (50.0%)	5 (25.0%)	15 (37.5%)
Stable psychotropic medications	7 (35.0%)	11 (55.0%)	18 (45.0%)	7 (35.0%)	2 (10.0%)	9 (22.5%)
Concurrent psychotherapy	8 (40.0%)	7 (35.0%)	15 (37.5%)	7 (35.0%)	5 (25.0%)	12 (30.0%)
CAPS	69.5 (12.7)	74.8 (15.0)	72.1 (14.0)			
BDI-II	23.00 (11.87)	22.89 (10.46)	22.94 (11.02)	10.80 (9.08)	8.50 (10.34)	9.65 (9.68)
STAI						
State	48.84 (12.61)	51.20 (11.96)	50.05 (12.18)	35.37 (10.58)	34.70 (14.16)	35.03 (12.39)
Trait	51.47 (11.48)	53.94 (10.09)	52.74 (10.72)	38.74 (11.05)	37.76 (11.54)	38.23 (11.16)
STAXI						
State	12.74 (4.38)	13.05 (4.94)	12.90 (4.62)	10.21 (.71)	12.55 (6.82)	11.41 (4.99)
Trait	23.68 (8.51)	22.70 (5.67)	23.18 (7.11)	16.68 (2.65)	17.15 (6.61)	16.92 (5.02)
Anger Expression	35.21 (13.00)	37.12 (7.88)	36.19 (10.58)	21.57 (8.64)	24.00 (8.71)	22.82 (8.64)

Note. Means or proportions that do not share the same subscript were significantly different at $p < .05$. CBCT = Cognitive-Behavioural Conjoint Therapy; WL = Waitlist; CAPS = Clinician-Administered PTSD Scale; BDI-II = Beck Depression Inventory-II; STAI = State Trait Anxiety Inventory; STAXI = State Trait Anger Expression Inventory.

Measures

Clinician-interview and self-report measures administered as part of the parent CBCT waitlisted controlled trial were used. Relevant to the current study, patients' PTSD symptoms and partners' depressive, anxiety, and anger symptoms, as well as all individuals' intimate relationship violence and mental healthcare utilization were examined.

Clinician-Administered PTSD Scale. The CAPS (Blake et al., 1995) is a semistructured clinical interview that is used to assess PTSD diagnostic criteria and symptom severity consistent with the DSM-IV-TR (American Psychiatric Association, 2000). For a symptom to count toward a PTSD diagnosis it must receive a minimum rating of one on the frequency scale and two on the intensity scale. The CAPS was used as the primary measure of PTSD symptoms. Psychometric research has consistently shown that the CAPS has excellent interrater reliability and internal consistency on the three symptom clusters and on the 17-core items. In addition, test-retest reliability has been reported in the high range for the three symptom clusters and the 17 core items. The CAPS has also been shown to have strong convergent validity with other measures of PTSD (for a review see Weathers, Keane, & Davidson, 2001). Using 10% of the completed CAPS assessments, interrater reliability between study assessors and an independent assessor was calculated. Reliability was excellent for the CAPS total score ($ICC = .99$). In addition, internal consistency was excellent for the CAPS 17-core items across the four assessments (α ranging from .79-.95).

Structured Clinical Interview for DSM-IV-Patient Version. The SCID-P (First et al., 1995) is a semistructured clinical interview that assesses for diagnostic criteria of a variety of mental disorders in line with DSM-IV-TR criteria. The SCID-P was used to identify exclusion criteria and to describe the sample. There is support for the interrater reliability of the SCID-P,

with kappa values ranging from .60 to .94 across Axis one disorders (Lobbestael, Lurgans, & Arntz, 2011). Interrater reliability calculations on 10% of the assessments indicated that the reliability for current and lifetime SCID-P diagnoses was excellent (κ s ranging from .71-1.00) across all disorders except mood disorders, which was moderate ($\kappa = .60$).

Beck Depression Inventory-II. The Beck Depression Inventory-II (BDI-II; Beck, Steer, & Brown, 1996) is a 21-item, self-report measure that assesses the severity of depressive symptoms. The BDI-II was given to all individuals and was used as a primary outcome measure in partners and as a measure of comorbidity in patients. Internal consistency has been reported at .91 (Beck, Steer, Ball, & Ranieri, 1996) and test-retest reliability has been reported at .93 (Beck, Steer, & Brown, 1996). Research has also supported convergent validity for the BDI-II, with correlations between the BDI-II and the Hamilton Psychiatric Rating Scale for Depression reported at .71 (Beck, Steer, & Brown, 1996). Internal consistency was excellent across the four assessments (α s ranging from .93-.94).

State-Trait Anxiety Inventory. The State-Trait Anxiety Inventory (STAI; Spielberger, 1983) is a 40-item measure made up of two 20-item scales examining state anxiety (STAI-S) and trait anxiety (STAI-T). The STAI was given to all individuals and was used as a primary outcome measure in partners and as a measure of comorbidity in patients. Internal consistency has been estimated at .89 and test-retest reliability ranging from .73-.86, with lower estimates for the state scale compared with the trait scale (Barnes, Harp, & Jung, 2002; Spielberger, 1983). Although previous studies have documented problems with the validity of the STAI as a measure of anxiety specifically (Bieling, Antony, & Swinson, 1998; Grös, Antony, Simms, & McCabe, 2007), it has been used in previous studies examining CBCT (e.g., Monson et al., 2011) and does seem to measure general psychological distress; consequently, it was used in the current study

in order to compare findings to previous work and as a measure of partners' general psychological distress. Internal consistency was excellent for both the state and trait scales across the four assessments, with α s ranging from .95-.96 and .94-.96 respectively.

State-Trait Anger Expression Inventory. The State-Trait Anger Expression Inventory (STAXI; Spielberger, 1988) is a 44-item measure made up six scales and two subscales: 1) state anger; 2) trait anger, 2a) angry temperament, 2b) angry reaction; 3) anger-in; 4) anger-out; 5) anger control; and 6) anger expression. Items are rated on a 4-point scale. The STAXI was given to all individuals and was used as a primary outcome measure in partners and as a measure of comorbidity in patients. Internal consistency for the state and trait scales has been estimated between .82 and .93 (Spielberger, 1988). Internal consistency was excellent for both the state and trait scales across the four assessments, with α s ranging from .77-.95 and .85-.90 respectively. However, internal consistency was poor for the anger expression scale across the four assessments (α s = .13-.52). Consistent with previous research in this area (e.g., Monson et al., 2011), only the state, trait, and anger expression scales were examined.

Revised Conflict Tactics Scales. The CTS-2 (Straus et al., 1996) is a 39-item measure that assesses physical and psychological conflict within a romantic relationship. Individuals respond to each item according to their perpetration and their partner's perpetration of said acts. The CTS-2 contains five subscales examining physical assault, psychological aggression, negotiation, injury, and sexual coercion, with subscale internal consistency ranging from .79 to .95 and support for convergent and divergent validity of the subscales (Strauss et al., 1996).

Mental Healthcare Utilization. The Mental Healthcare Utilization (MHU) measure is a clinician-administered interview that examines current utilization of psychosocial and psychopharmacological treatments. Items inquire about the type and frequency of treatments

currently being utilized, as well as the type of provider and methods used. Although no psychometric research exists on the MHU, a similar measure has been used in related research (Schnurr et al., 2007). The MHU was given to all individuals to account for treatments external to the study.

Procedure

Participants were recruited using flyers in the community and online, and referrals from partnering hospitals and mental health service providers in the Toronto, ON, and Boston, MA, metropolitan areas, as well as through the Veterans Affairs hospital in Boston, MA. Couples who contacted study personnel were screened via telephone for preliminary inclusion criteria and were informed of the details of participation. Those who screened positive for the inclusion criteria were invited for an informed consent meeting during which they received extensive information regarding participation. After obtaining informed consent, the CAPS, SCID-P, and MHU were administered to the participants by doctoral- and master's-level trained clinicians. Individuals were then given self-report measures assessing a variety of different outcomes (e.g., anxiety, depression, substance use, PTSD) that were used as pretreatment measures of psychological functioning. Pending the presence of PTSD in one partner, as determined by the CAPS, and no exclusionary criteria being met, dyads were randomized into the treatment trial, either to receive CBCT immediately or to a waitlist condition.

Treatments

Cognitive-Behavioural Conjoint Therapy for PTSD. Dyads randomized into the treatment immediately condition received the CBCT treatment protocol (Monson & Fredman, 2012), administered by doctoral- or master-level trained therapists. CBCT is a 15-session conjoint treatment protocol aimed at reducing PTSD symptoms while simultaneously enhancing

relationship satisfaction and dyadic functioning. The treatment consists of three phases and was designed to be delivered over a 12-week period, with sessions in phases one and two being delivered twice weekly and sessions in phase three being delivered weekly. Phase one (sessions 1-2) is aimed at introducing the treatment protocol, explaining the rationale for treatment, delivering psychoeducation about PTSD and relationships, and establishing safety in the relationship. Phase two (sessions 3-7) utilizes a series of dyadic interventions directed at increasing communication in order to enhance relationship functioning and satisfaction. In addition, this phase has an emphasis on reducing avoidance in a dyadic context. Phase three (sessions 8-15) has a focus on making meaning of the traumatic event and ending therapy. During this phase, cognitive change strategies are used to address maladaptive trauma-related cognitions. In addition, this segment of treatment aims to highlight treatment gains and reinforce the dyad's commitment to ongoing improvement. All therapists were supervised by the primary developer of CBCT, Candice Monson, PhD. In addition to their baseline/pre-treatment assessment, dyads randomized to this condition were assessed at mid- (after session 7) and post-treatment, as well as at a 3-month follow-up.

Waitlist. Dyads randomized into the waitlist condition received CBCT after 12 weeks of waiting. In addition to their baseline assessment, dyads in this condition were assessed at time points equivalent to the CBCT condition's mid- (after 4 weeks of waiting) and post-treatment (after 12 weeks of waiting) assessments. In addition, dyads in the waitlist condition received a final assessment after completing treatment. Thus, their 12-week assessment and post-treatment assessment served as an uncontrolled replication of CBCT effects (see Figures 3 and 4).

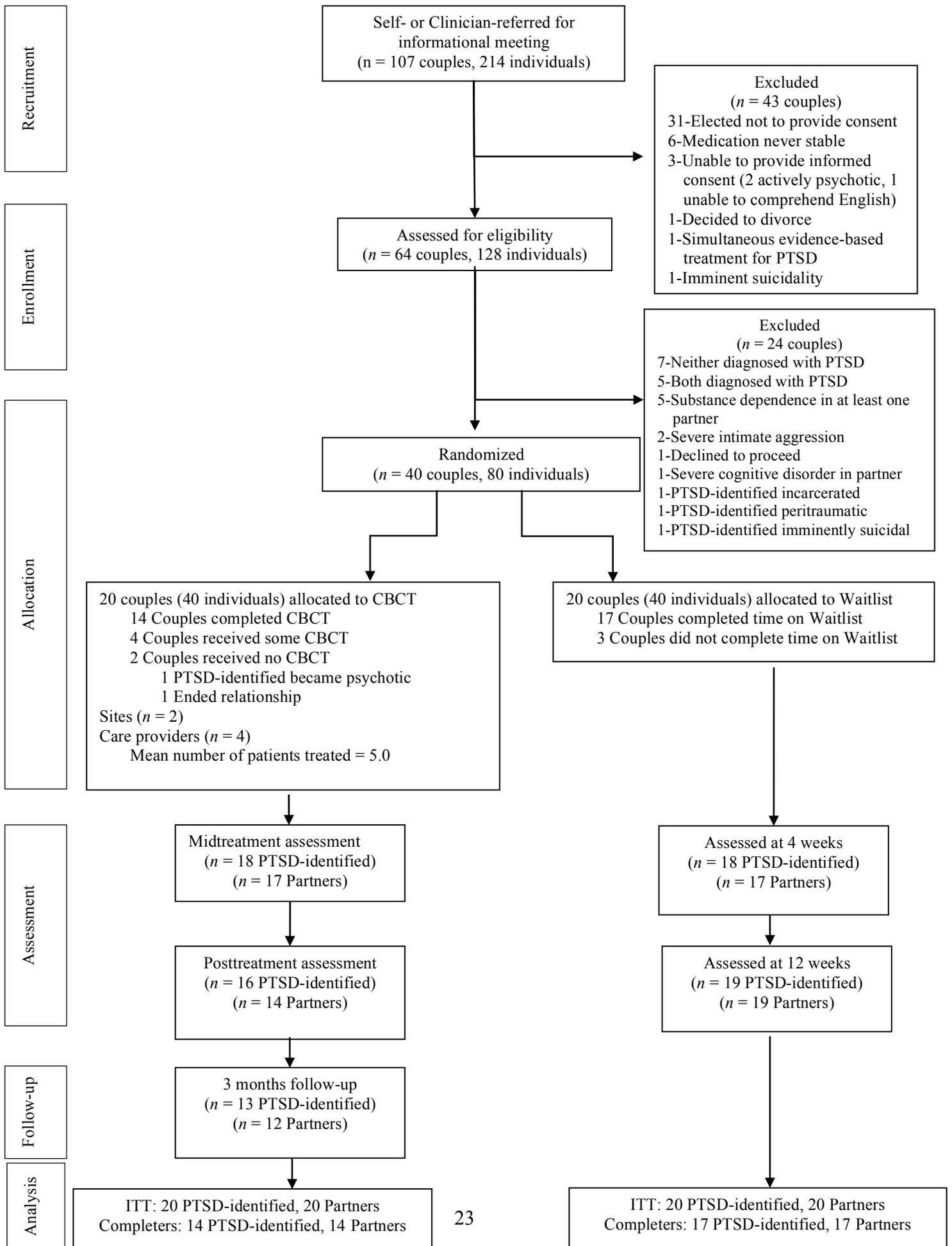


Figure 3. Flow chart of participants in the trial for the growth curve modeling analyses. ITT = intention-to-treat.

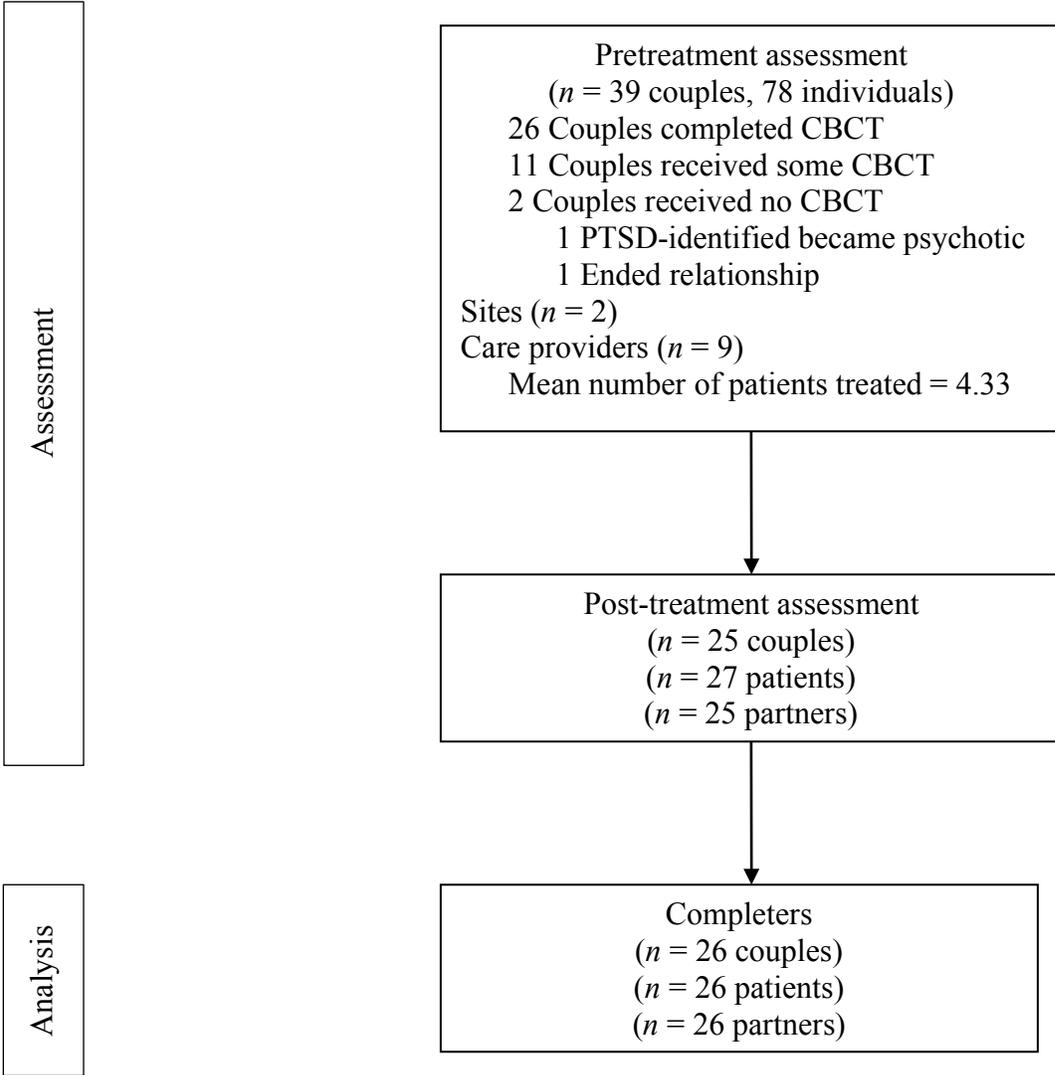


Figure 4. Flow chart of participants in the trial for the path analyses.

Data Analytic Strategy

Given the specific hypotheses that were put forth, two different types of analyses were conducted. Specifically, growth curve modeling and path analysis, a type of structural equation modeling, were used to analyze the data.

Growth curve modeling. For analyses comparing the CBCT and waitlist conditions, growth curve modeling was used. Growth curve modeling, also referred to as hierarchical linear modeling, is a statistical technique for analyzing data in which there is nested data, meaning data points that exist within a larger contextual variable (in this case time points are nested within an individual). This technique takes into account systematic shared variance between data points, compensating for the violation of the assumption of independence of observations normally required for other statistical analyses (Tabachnick & Fidell, 2007).

The current study utilized a growth curve model with a 2-level hierarchical structure [time points (level 1) nested within individuals (level 2)]. The models compared partners' BDI-II, STAI (state and trait scales), and STAXI (state, trait, and anger expression scales) across time and conditions, using the CBCT and waitlist conditions' pre/baseline, mid-/4-week, and posttreatment/12-week assessments. Furthermore, models can have fixed or random intercepts and effects, indicating that the intercept and/or slope of the model is held stable across all participants or is permitted to fluctuate. For growth curve modeling analyses within the current paper, random intercepts, random effects models were utilized. This approach was selected in order to account for individual differences in baseline psychological functioning and change across treatment.

Growth curve modeling has the advantage of allowing time to be treated as either a continuous or categorical variable, allowing for unstructured time data (i.e., data that has

deviated from the data collection schedule) to be analyzed (Raudenbush & Bryk, 2002). Thus, time was treated as a continuous variable in the growth curve models, allowing for time unstructured data to be analyzed. An elapsed time variable was calculated that detailed the number of weeks that had elapsed since the baseline assessment.

Hypothesis one. Analyses for hypothesis one were conducted in accordance with intention-to-treat principles ($n = 40$) and on the completer sample ($n = 31$). Completer status was based on a dyad's completion of their respective condition (i.e., completion of the 15 sessions of CBCT for the CBCT condition and completion of the 4- and 12-week assessments for the waitlist condition). To determine whether partners in the CBCT condition had significantly greater improvements in measures of psychological functioning compared with partners in the waitlist condition, a 2-level (random intercepts, random effects) growth curve model was constructed with condition assignment (level-2) and time (level-1) as predictors of significant others' psychological functioning (BDI-II, STAI: state and trait, and STAXI: state, trait, and anger expression; level-1). Separate growth curve models were constructed for each outcome variable.

Path analysis. For analyses examining the influence of partners' and patients' symptoms on one another, path analysis was used. Path analysis, a type of structural equation modeling, is a statistical technique used to examine dependencies among a number of variables and their fit to a data set (Tabachnick & Fidell, 2007). In addition, it can test theoretical models as well as reciprocal relationships between variables. Path analysis can also accommodate missing data by utilizing available data on a given variable rather than deleting cases because of data omission. Full maximum likelihood estimation was used, allowing for the estimation of missing data points from existing data (Blunch, 2008; Tabachnick & Fidell, 2007).

In addition to evaluating significant paths between variables, path analysis models were evaluated using fit indices to assess how well the proposed models explained the data. That is, the fit indices assess the degree to which a proposed model's variance-covariance matrix matches the original variance-covariance matrix that existed between variables. As each fit index has individual strengths and biases (Blunch, 2008; Tabachnick & Fidell, 2007), a number of fit indices were examined in assessing the fit of the proposed models. Indices that were examined included the model chi-square statistic (χ^2), the normed chi square statistic (χ^2/df), the normed fit index (NFI), the comparative fit index (CFI), the Tucker-Lewis coefficient (TLI), the parsimony-adjusted CFI (PCFI), and the root mean square error of approximation (RMSEA).

As mentioned, each of these indices has strengths and weaknesses. The chi-square statistic assesses absolute fit of a model. It examines how well the proposed model, with imposed associations between variables, can recreate the variance-covariance matrix to that of no model at all. This test operates under the null hypothesis that the proposed model fits the data. Thus, large p values are indicative of good model fit. Taking the chi-square statistic and dividing it by the degrees of freedom offers another index of absolute fit. If this value is less than two, it is suggestive of good model fit (Tabachnick & Fidell, 2007). Although easily interpretable, the chi-square statistic is sensitive to sample size. This results in a tendency to reject the model in large sample sizes and retain it in small sample sizes. Thus, other fit indices should be reported to supplement the interpretation of the model fit. Relative fit indices provide another way by which models can be evaluated. These statistics use a saturated model (i.e., a model with the maximum number of parameters) and an independence model (i.e., a model with no correlations among variables) as boundaries that confine the model being examined. Essentially, the saturated model provides the best possible fit to the data, and the independence model provides the worst possible

fit to the data. As a result, relative fit indices indicate the degree to which the proposed model has travelled between these parameters. In addition to providing boundaries by which to assess the proposed model, these indices allow for comparison across different models (Blunch, 2008). The relative fit indices include: the NFI (which tends to underestimate the fit in small sample sizes; Bentler & Bonnett, 1980), the CFI (which takes into account degrees of freedom and thus is more appropriate for small samples; Bentler, 1990), and the TLI (Tucker & Lewis, 1973). Given that relative fit indices examine the distance travelled between the parameters set out by the saturated and independence models, values greater than .95 are an indication of the proposed model having a good fit to the data (Blunch, 2008). In addition, the relative fit indices can be used as comparative indices for nested models (i.e., models that can be derived by constraining paths of parent models), with the NFI and TLI being statistically tested. In these comparisons, the null hypothesis is that the reduced model does not result in a significant worse fit to the data compared to the full model. Parsimony adjusted measures are a series of indices that introduce a penalty for complex models (i.e., models having increased parameters with the intention of increasing model fit). The PCFI is akin to the CFI with a bias correction for number of parameters. Larger values indicate a greater fit, with values greater than .60 being accepted as a good fit (Blunch, 2008). In addition, the RMSEA (Steiger & Lind, 1980) was used with its significance test for closeness of fit (PCLOSE). Values of less than .05 on the RMSEA is a sign of a good fit, with a PCLOSE value of greater than .05 as an indication that the null hypothesis is met (Blunch, 2008).

Collapsing the sample. As noted previously, the waitlist condition received treatment after their 12-week assessment and then received a final assessment after completing a course of CBCT. Thus, in order to increase sample size, the remaining hypotheses utilized the waitlist

condition's 12-week assessment as a measure of pretreatment functioning and collapsed across conditions. Posttreatment assessments were also collapsed across conditions. Essentially, this provided pre- and posttreatment assessments for each condition. To most accurately represent significant others' influence on one another across treatment, only treatment completers were retained ($n = 26$). In this case, completers consisted of the dyads initially randomized to CBCT who achieved completer status ($n = 14$), as well as dyads randomized to the waitlist who completed a course of CBCT after their 12-week assessment ($n = 12$). This subsample, consisting of treatment completers collapsed across condition, was referred to as the *collapsed sample* and was the subject of the remaining analyses.

In order to identify variables in which there was sufficient pre- to posttreatment variation, a series of repeated measures *t*-tests were conducted. Only variables that significantly improved from pre- to posttreatment in the collapsed sample were further examined using path analysis.

Hypothesis two. To determine if partners' baseline psychological functioning and patients' baseline PTSD symptoms predicted their significant others' symptom change, a path analysis was conducted examining the relationships specified in Figure 1 (i.e., cross paths model).

Hypothesis three. To determine if partners' change in psychological functioning and patients' PTSD symptom change predicted one another, a path analysis was conducted examining the relationships specified in Figure 2 (i.e., reciprocal paths model).

Hypothesis four. To determine if the reciprocal paths model, specified in hypothesis three (see Figure 2), was a good fit to the data, the paths from patient-to-partner and partner-to-patient were constrained to zero, in turn. Essentially, this was statistically equivalent to

eliminating the path from the model (i.e., indicating that there was no relationship between these variables). This process tested whether the reciprocal paths model best explained the data.

Results

Overall, there were few significant findings across all of the analyses conducted. Neither the growth curve modeling analyses examining partner outcomes, nor path analyses resulted in significant findings.

Growth Curve Modeling

Hypothesis one. Least square means, pre-post effect sizes, and between-condition effect sizes for the outcome variables of interest are presented in Table 2 for both the intention-to-treat and completer samples. Contrary to hypothesis one, growth curve modeling analyses did not reveal a significant effect of time or a significant time by treatment condition interaction for any of the partner outcome measures for the intention-to-treat sample. Similar findings were obtained when using data from only those individuals who achieved completer status.

Table 2

Partners' Outcomes as a Function of Condition

Outcome Measure	Pre-Post g^*		Between Group g^*	Midtreatment/ 4-Week Waitlist Least Square Means		Posttreatment/ 12-Week Waitlist Least Square Means	
	CBCT	WL		CBCT	WL	CBCT	WL
Intention-to-Treat Sample				$n = 20$	$n = 20$	$n = 20$	$n = 20$
BDI-II	.06	.02	-.33	10.84	7.70	10.52	7.60
STAI							
State	-.01	-.17	.15	35.42	35.80	35.51	37.49
Trait	.03	.14	-.23	38.89	37.30	38.66	36.19
STAXI							
State	-.39	.02	.25	10.64	12.04	10.97	11.96
Trait	.10	.07	-.06	17.09	16.87	16.89	16.61
Anger Expression	.05	-.13	.45	21.42	24.22	21.17	24.94
Completer Sample				$n = 14$	$n = 17$	$n = 14$	$n = 17$
BDI-II	.03	.03	-.09	9.37	8.60	9.22	8.42
STAI							
State	-.03	-.22	.48	33.63	37.57	33.78	39.74
Trait	-.01	.10	.03	37.91	39.12	38.00	38.36
STAXI							
State	-.16	.03	.45	10.23	12.28	10.28	12.18
Trait	.20	.04	-.10	17.13	16.47	16.72	16.33
Anger Expression	.07	-.17	.64	20.44	24.41	20.04	25.24

Note. Positive effect sizes represent improvements in the expected direction. Effect sizes were calculated using Hedge's g with bias correction. Least square means were calculated from multilevel models. CBCT, Cognitive-Behavioral Conjoint Therapy; WL = Waitlist; BDI-II = Beck Depression Inventory-II; STAI = State-Trait Anxiety Inventory; STAXI = State Trait Anger Expression Inventory.

Path Analyses

Table 3 includes the Pearson's correlation matrix for variables used in the repeated measures *t*-tests and the path analyses. Results of the repeated measures *t*-tests can be found in Table 4. Overall, there were few significant correlations between measures of partners' psychological functioning and patients' PTSD. In addition, only partners' depressive symptomatology and patients' PTSD significantly improved with treatment.

Table 3
Pearson Correlation Matrix for Repeated Measures t-tests and Path Variables

Variable	Correlations												
	1	2	3	4	5	6	7	8	9	10	11	12	13
1. CAPS Pre ^a (<i>n</i> = 26)	-												
2. CAPS Post ^a (<i>n</i> = 24)	.48*	-											
3. BDI-II Pre (<i>n</i> = 26)	.38	.17	-										
4. BDI-II Post (<i>n</i> = 22)	.16	-.01	.78***	-									
5. STAI-S Pre (<i>n</i> = 26)	.35	.10	.56**	.29	-								
6. STAI-S Post (<i>n</i> = 23)	-.09	-.05	.12	.31	.31	-							
7. STAI-T Pre (<i>n</i> = 26)	-.01	.16	.68***	.74***	.49*	.28	-						
8. STAI-T Post (<i>n</i> = 23)	-.02	.13	.55**	.80***	.37	.50*	.79***	-					
9. STAXI-S Pre (<i>n</i> = 26)	.24	.04	.23	-.23	.54**	-.12	.10	-.01	-				
10. STAXI-S Post (<i>n</i> = 23)	-.18	-.22	-.05	-.08	.19	.53*	.07	.14	.27	-			
11. STAXI-T Pre (<i>n</i> = 26)	.10	-.01	.06	-.08	.02	-.29	-.06	-.18	.43*	-.18	-		
12. STAXI-T Post (<i>n</i> = 23)	.09	-.02	-.09	-.17	.07	-.21	-.04	-.07	.41	.04	.64**	-	
13. STAXI-E Pre (<i>n</i> = 26)	.21	.29	.13	-.28	.40*	.09	.19	-.06	.38	.17	.32	.49*	-
14. STAXI-E Post (<i>n</i> = 23)	.13	.06	.07	-.02	.32	.07	.30	.16	.24	.02	.28	.46*	.67***

Note. Variables without a superscript pertain to partners. CAPS = Clinician-Administered PTSD Scale; Pre = Pretreatment; Post = Posttreatment; BDI-II = Beck Depression Inventory-II; STAI-S = State Trait Anxiety Inventory-State; STAI-T = State Trait Anxiety Inventory-Trait; STAXI-S = State Trait Anger Expression Inventory-State; STAXI-T = State Trait Anger Expression Inventory-Trait; STAXI-E = State Trait Anger Expression Inventory-Anger Expression.

^aCAPS were calculated using the patients' scores.

* $p < .05$. ** $p < .01$. *** $p < .001$.

Table 4

Partners' and Patients' Treatment Outcomes in the Collapsed Sample

Variables	Collapsed Sample			
	Pre-Tx <i>M (SD)</i>	Post-Tx <i>M (SD)</i>	<i>t (df)</i>	<i>g*</i>
CAPS ^a	66.21 (19.23)	29.25 (21.53)	8.70 ^{***} (23)	1.78
BDI-II	9.14 (7.25)	6.83 (6.23)	2.38 [*] (21)	.34
STAI-S	36.70 (9.43)	34.09 (6.87)	1.28 (22)	.31
STAI-T	38.96 (9.17)	37.52 (7.73)	1.28 (22)	.17
STAXI-S	10.70 (1.40)	10.43 (.73)	.90 (22)	.23
STAXI-T	16.30 (3.40)	16.00 (3.36)	.51 (22)	.09
STAXI-E	24.22 (8.13)	21.74 (6.50)	1.94 (22)	.33

Note. Variables without a superscript pertain to partners. Positive effect sizes represent improvements in the expected direction. Effect sizes were calculated using Hedge's *g* with bias correction. Pre-Tx = Pretreatment; Post-Tx = Posttreatment; CAPS = Clinician-Administered PTSD Scale; BDI-II = Beck Depression Inventory-II; STAI-S = State Trait Anxiety Inventory-State; STAI-T = State Trait Anxiety Inventory-Trait; STAXI-S = State Trait Anger Expression Inventory-State; STAXI-T = State Trait Anger Expression Inventory-Trait; STAXI-E = State Trait Anger Expression Inventory- Anger Expression.

^aCAPS were calculated using the patients' scores.

* $p < .05$. *** $p < .001$.

Hypothesis two. Contrary to hypothesis two, partners' pretreatment BDI-II scores were not significantly associated with patients' posttreatment CAPS scores. Similarly, patients' pretreatment CAPS scores were not significantly associated with partners' posttreatment BDI-II scores (see Figure 5). Overall, the cross paths model resulted in a moderate-good fit to the data, with exception of the PCFI.

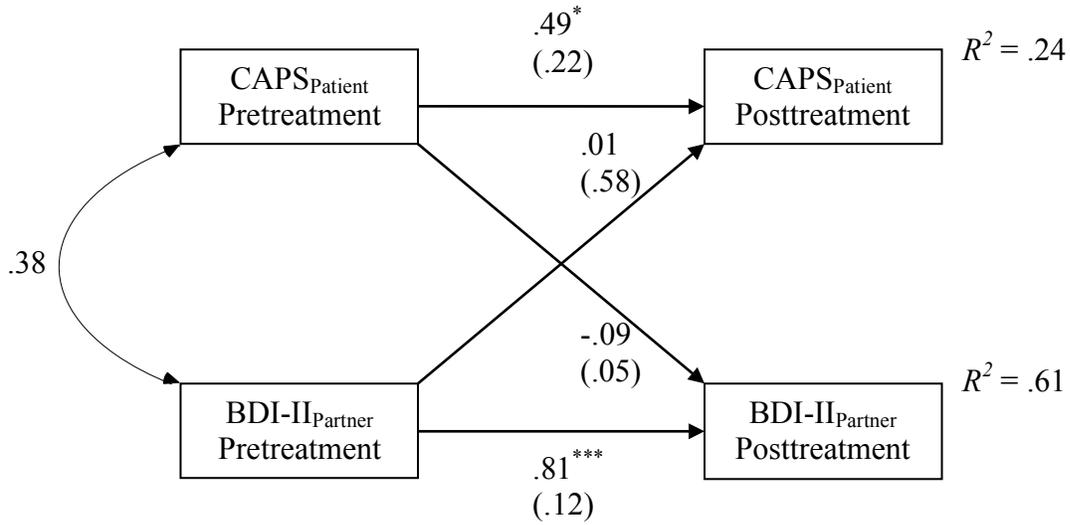


Figure 5. Cross paths model. Pretreatment functioning as predictors of symptom response with treatment. CAPS = Clinician-Administered PTSD Scale; BDI-II = Beck Depression Inventory-II. $\chi^2(1) = .37, p = .54, \chi^2/df = .37, NFI = .99, CFI = 1.00, TLI = 1.31, RMSEA = .00, PCLOSE = .56, PCFI = .10. *p < .05. ***p < .001.$

Hypotheses three and four. Contrary to hypothesis three, partners' pre- to posttreatment change on the BDI-II was not significantly associated with patients' pre- to posttreatment change on the CAPS. Similarly, patients' pre- to posttreatment change on the CAPS was not significantly associated with partners' pre- to posttreatment change on the BDI-II (see Figure 6). Overall, the reciprocal paths model resulted in a moderate-good fit to the data, with exception of the PCFI.

When the model was reconstructed with the path from patients' posttreatment CAPS to partners' posttreatment BDI-II constrained to zero, the association between partners' pre- to posttreatment change on the BDI-II to patients' pre- to posttreatment change on the CAPS remained nonsignificant (see Figure 7). Overall, this model resulted in a moderate-good fit to the data, with the exception of the PCFI. In addition, compared with the reciprocal paths model, the constrained model did not result in a significantly worse fit to the data (see Table 5).

When the reciprocal path was constrained, the association between patients' pre- to posttreatment change on the CAPS to partners' pre- to posttreatment change on the BDI-II remained nonsignificant (see Figure 8). This model resulted in a moderate-good fit to the data, with the exception of the PCFI. In addition, compared with the reciprocal paths model, the constrained model did not result in a significantly worse fit to the data (see Table 5).

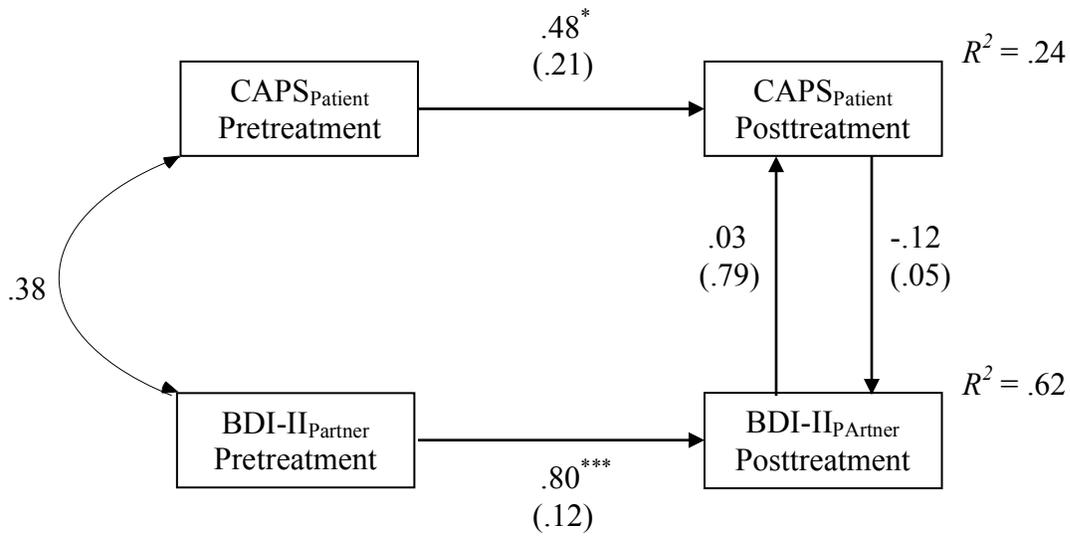


Figure 6. Reciprocal paths model. Symptom changes as predictors of partners' symptom response with treatment. CAPS = Clinician-Administered PTSD Scale; BDI-II = Beck Depression Inventory-II. $\chi^2(1) = .45, p = .83, \chi^2/df = .45, NFI = 1.00, CFI = 1.00, TLI = 1.46, RMSEA = .00, PCLOSE = .84, PCFI = .10.$ * $p < .05.$ *** $p < .001.$

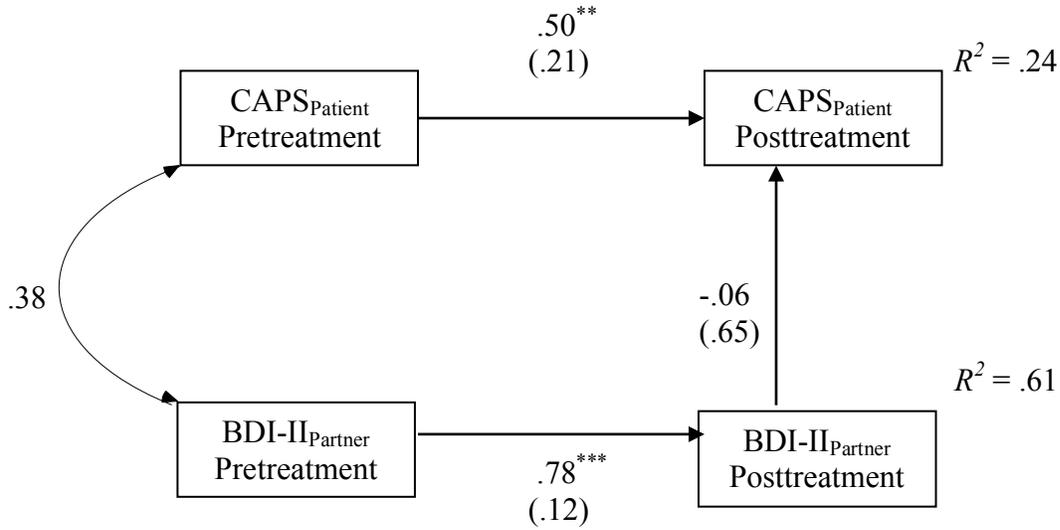


Figure 7. Reciprocal paths model with CAPS to BDI-II constrained. Symptom changes as predictors of partners' symptom response with treatment, with CAPS to BDI-II constrained to zero. CAPS = Clinician-Administered PTSD Scale; BDI-II = Beck Depression Inventory-II.

$\chi^2(2) = .63, p = .73, \chi^2/df = .32, NFI = .98, CFI = 1.00, TLI = 1.33, RMSEA = .00, PCLOSE = .75, PCFI = .20. ** p < .01. *** p < .001.$

Table 5

Comparative Fit Indices for the BDI-II-CAPS Constrained Paths Models,

Assuming the BDI-II-CAPS Reciprocal Paths Model to be Correct.

Models	NFI	TLI	<i>p</i>
CAPS to BDI-II Constrained	.02	.13	.45
BDI-II to CAPS Constrained	.00	-.01	.88

Note. CAPS = Clinician-Administered PTSD Scale; BDI-II = Beck

Depression Inventory-II.

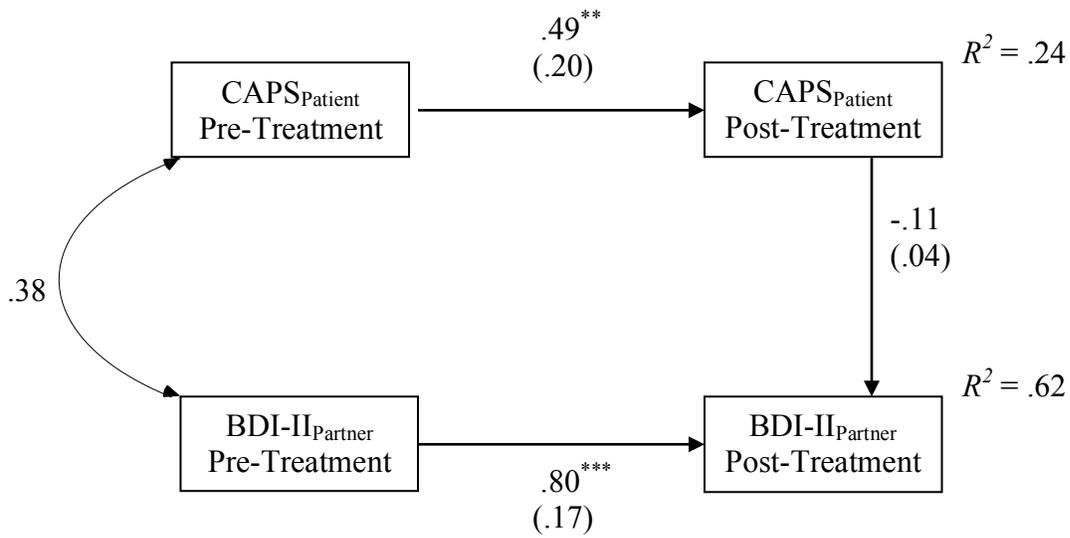


Figure 8. Reciprocal paths model with BDI-II to CAPS constrained. Symptom changes as predictors of partners' symptom response, with treatment with BDI-II to CAPS constrained to zero. CAPS = Clinician-Administered PTSD Scale; BDI-II = Beck Depression Inventory-II.

$\chi^2(2) = .07$ $p = .97$, $\chi^2/df = .03$, NFI = 1.00, CFI = 1.00, TLI = 1.47, RMSEA = .00, PCLOSE = .97, PCFI = .20. $^{**}p < .01$. $^{***}p < .001$.

Discussion

The current study examined intimate partners involved in a randomized controlled trial of a conjoint therapy for PTSD. Specifically, this study investigated changes in partners' psychological functioning resulting from treatment, and whether their psychological functioning changed as a function of patients' PTSD symptomatology at pretreatment and across treatment, and vice versa. Based on the existing literature on PTSD and its associated interpersonal factors, a number of specific hypotheses were put forth. Contrary to expectation, findings of the current study did not support the hypotheses; however, there are some important theoretical and methodological considerations that advance understanding of partners' psychological functioning and its association to patients' posttraumatic recovery.

Partners' Psychological Functioning After Treatment

A primary goal of this study was to examine if partners randomized to CBCT had greater improvements in psychological functioning compared with those awaiting treatment. Results revealed no differences in any domain of psychological functioning (i.e., depressive, anxiety, and anger symptoms) between CBCT and waitlist conditions at posttreatment. Previous PTSD treatment studies have revealed variable results with regard to partner effects of treatment. Specifically, the one controlled (Sweany, 1987) and three uncontrolled studies (Devilley, 2002; Monson et al., 2011; Monson, Stevens et al., 2005) previously reporting on partners' symptom outcomes did not find consistent improvements across measures. Specific to CBCT, one trial with Vietnam veterans' wives found improvements in some domains of psychological functioning (Monson, Stevens et al., 2005) and the other with a community sample found increased anger expression following treatment (Monson et al., 2011).

The current study is the first to test the effects of a conjoint treatment for PTSD on partners' symptom outcomes using a controlled trial methodology. Prior studies have only utilized uncontrolled trial designs to determine treatment efficacy (Devilley, 2002; Monson, Stevens et al., 2005). Although uncontrolled studies are helpful in identifying new phenomena and piloting interventions, controlled treatment trials offer a more rigorous methodological approach to determine treatment effects. The waitlist design allows for control of issues such as the natural course of recovery and the monitoring of worsening of participants in the waitlist condition (O'Leary & Borkovec, 1978).

A possible methodological reason for the null findings of this study relates to measurement of the outcomes. Partners involved in the current study and prior treatment studies have not generally been in clinically distressed ranges on the outcome measures of interest (Devilley, 2002; Monson et al., 2011; Monson, Stevens et al., 2005; Sweany, 1987). Although clinical levels of psychological symptoms have consistently been found among partners of individuals with PTSD relative to partners of trauma exposed individuals without PTSD and community controls in naturalistic or observational studies (e.g., Beckham, Lytle, et al., 1996; Manguno-Mire et al., 2007; for a review see Renshaw et al., 2011), the distress that partners in treatment trials have exhibited does not appear to be as severe. According to normative data, normal scores on the BDI-II, STAI, and STAXI for reference samples that best approximate that of the current study are reported below 13 for the BDI-II, 36 for the STAI-state, 35 for the STAI-trait, 13 for the STAXI-state, 20 for the STAXI-trait, and 20 for the STAXI-anger expression (Beck, Steer, & Brown, 1996; Spielberger, 1983; Spielberger, 1988). Partners' symptoms in the current sample tended to be lower than the normative sample or, in a few cases, slightly above the mean (see Table 1). This indicates that the current sample does not appear to have elevated

levels of distress relative to a normative population. As a result, it is more difficult to change levels of distress when there are minimal/normal levels of existing distress. It is also important to note which measures of symptom outcomes were used in this and prior trials. To more fully understand the potential effects of conjoint therapy on partners, positive domains of functioning, such as quality of life or well-being, should also be assessed. Finally, there has been discussion in the literature regarding secondary or vicarious traumatization, which has led to concern about the inclusion of partners in trauma-focused treatments (for a review see Figley, 1989). Findings of the current study do not support this notion, as partners did not appear to evidence any iatrogenic effects of treatment.

Another methodological consideration is that treatment trials are often underpowered to detect secondary outcomes, such as those investigating partners' outcomes, which are likely smaller than the expected primary outcomes on which these studies are powered (Kraemer, Wilson, Fairburn, & Agras, 2002). Although it seems unlikely that power was an issue in the current study given the small effect size estimates for the outcomes, this is an important issue that should be taken into consideration when conducting future research in this area. Similarly, as was done in the current study, mean scores have typically been used to assess partners' treatment outcomes. Given the low levels of distress in this population, a mean difference or nomothetic approach to the analyses may obscure the potential benefits to partners. Future research should take a more ideographic approach by investigating changes in subsets of partner samples presenting with elevated or clinical levels of distress, or consider using clinically significant distress cut offs and clinically significant change analyses, as well as loss of diagnoses, when reporting on partners' treatment outcomes.

Finally, partners' concurrent mental healthcare utilization should be taken into account as a possible explanation of the null findings. In the current study, a relatively large percentage (37.5%) of partners in the sample reported current use of mental healthcare services. This is approximately 10% higher than an estimate reported for partners of a veteran sample with PTSD (Sherman et al., 2005). Although the frequencies and exact types of interventions utilized by partners in the current study are unknown, there is the possibility that their ongoing mental healthcare utilization partially accounted for the stability of their symptoms across the duration of the study. Further research is necessary to establish the association between partners' individual mental healthcare use and changes in their psychological functioning in dyadic interventions to rule out this possible confound.

Influences on Treatment Effects

In addition to examining partners' treatment outcomes, the current study aimed to investigate different models by which partners and patients may influence one another's outcomes in treatment. Interestingly, in contrast to the growth curve modeling analyses, results of the collapsed sample analyses indicated that partners' depressive symptoms and patients' PTSD symptoms significantly reduced across treatment. No other domain of partners' psychological functioning resulted in significant improvements in these analyses.

A possible explanation for the significant results in partners' depressive symptoms in the collapsed sample is that the individuals who received treatment after completing the waitlist condition had a different treatment response than those originally randomized to CBCT. It may be that they had more persistence or motivation for the therapy if they completed the waitlist and then the treatment, resulting in better treatment effects. Future research should consider examining differential responding in conditions prior to collapsing across them in order to

identify between group differences in treatment response. Exploratory posthoc analyses revealed that the current sample did, in fact, have differential responses to treatment as a function of condition, such that when conditions were examined in isolation those who received treatment after completing the waitlist condition significantly improved, whereas those randomized to CBCT immediately did not.¹

With a few exceptions, the fit indices from the path analyses, designed to investigate different models of influence, indicated that all of the models put forth were generally a good fit to the data. The poor fit suggested by the PCFI across models was likely a result of the ratio of number of parameters to number of observed variables, because PCFI penalizes models with increased parameters as they tend to increase model fit. Despite the general goodness of fit across models, evaluation of the individual paths suggested that the majority of predictive power resulted from pretreatment scores predicting their corresponding posttreatment scores (e.g., BDI-II_{partner} pretreatment predicting BDI-II_{partner} posttreatment). Thus, there was little to no additional value of including the cross and reciprocal paths in the various models. In fact, paths depicting influence from one partner to the other were nonsignificant across all models. Furthermore, the regression coefficients associated with these paths were small, suggesting that the null findings were likely not due to a small sample and resulting power issue ($|\beta_s| < .12$). This explanation is consistent with the good model fit suggested by fit indices other than the PCFI, as they do not account for unnecessary or excessive paths. In sum, although many of the fit indices suggested that the models fit the data well, partner and patient influences on one another did not appear to be contributing to their overall predictive power.

The current study does not support the notion that significant others influence each other's treatment outcomes in spite of prior studies documenting an association between different

facets of interpersonal functioning at pretreatment and PTSD treatment outcomes (Monson, Rodriguez, et al., 2005; Price et al., 2011; Tarrrier et al., 1999). There may be important differences between the current and previous research that explain these contrary findings. One difference is that the constructs under investigation varied across studies. Prior studies used interpersonal- or dyadic-level predictor variables (i.e., quality of relationships, social support, intimate relationship functioning), whereas the current study explored the influence of intrapersonal variables (i.e., partners' psychological functioning and patients' PTSD symptomatology). It may be that interpersonal-level variables have more shared method variance (i.e., reporting on a shared phenomenon) and thus more consistently evidence an association with patients' treatment response. Intrapersonal variables on the other hand, such as partners' psychological functioning, may not be as closely related to these other constructs and thus may not have as close an association with patients' treatment outcomes.

The hypothesis that interpersonal variables rather than intrapersonal variables may be of greater importance in individual treatment outcomes offers a possible explanation of the lack of findings in the current study. Although this hypothesis provides an explanation for the null findings with respect to the paths denoting influence from one partner to the other, it does not explain the absence of significant associations between partners' pretreatment psychological functioning and patients' pretreatment PTSD. Inspection of the strength of the pretreatment correlations in the current study suggests that the lack of statistically significant findings in this study may be a result of low power related to small sample size. Previous studies have consistently found small to moderate associations between patients' and partners' symptomatology ($r = .18-.45$), with many reporting correlations in the $r = .20$ range (e.g., Dekel et al., 2005; Manguno-Mire et al., 2007; Renshaw & Campbell, 2011). With the exception of the

trait scales of STAI and STAXI, pretreatment correlations between measures of partners' psychological distress and patients' PTSD ranged from $r = .21$ to $.38$ in the current study; yet, none were statistically significant in this sample of 26 dyads.

To further document the influence of power in detecting statistically significant associations, a posthoc power analysis was conducted using the largest correlation between patient and partner pretreatment variables. Results of this analysis suggested that 56 dyads would have been necessary to statistically detect this effect with 80% confidence and an alpha of $.05$. Given that previous research in this area has relied almost exclusively on military and veteran samples to establish the association between partners' psychological distress and patients' PTSD (e.g., Beckham, Lytle, et al., 1996; Manguno-Mire et al., 2007; Renshaw & Campbell, 2011), this study adds to the existing literature by demonstrating the potential for these findings to generalize to non-military/non-veteran samples with PTSD. Although not statistically significant, the size of these correlations suggests that these variables are related cross-sectionally prior to intervention.

Theoretical Implications

The findings of the current study assist in informing and outlining the bounds of current theory. Specifically, C-BIT (Monson et al., 2010; Monson, Stevens, et al. 2005) was used to inform the hypotheses and the models of influence explored in the current study. Given the results, it is necessary to consider factors that can explain the discrepancy between the associations suggested by this theory and the lack of findings that were yielded from this study.

One possibility for this discrepancy is that only some of the associations indicated in C-BIT were explored in the current study. Although partner and patient symptomatology was explored, each element of an individual's system (i.e., cognitions, behaviours, emotions) and the

relational system more generally (i.e., relationship functioning/adjustment) were not explored. Thus, it may be that these associations do not present in isolation, but are a part of a more complex set of associations that become evident when all elements are considered, as postulated by the systemic nature of C-BIT.

Another possible explanation of the discrepancy between the associations suggested in C-BIT and the results of the current study is that the relational system is relatively more responsible for the associations between individuals' symptomatology. In other words, relationship-level variables may mediate the influence from partners-to-patients and vice versa across treatment. Thus, without simultaneous consideration of relationship variables in the current sample, one may be missing critical information. This is consistent with the earlier suggestion that these models of influence may be more pertinent if relationship-level variables are considered (e.g., Monson, Rodriguez et al., 2005; Tarrrier et al., 1999). Further investigation of the theory in its entirety by more thoroughly testing all elements of the model is warranted before more definitive conclusions can be made.

Limitations and Future Directions

There are a number of limitations of the current study that should be taken into account in future research. First, the current study had a small sample size for both the growth curve models and path analyses. Small sample sizes in path analyses are particularly problematic as the findings can be difficult to replicate due to large standard errors, thus questioning the validity of the overall model and associations between variables within it (Norman, & Streiner, 2003). It is important to replicate the current work in a larger sample to further establish the consistency of the findings. In addition, a larger sample size would likely lead to significant pretreatment

correlations, which would represent an important extension of the association between partners' psychological functioning and patients' PTSD symptoms in nonmilitary/nonveteran populations.

This study is also limited by the measures used to assess partners' psychological functioning. Previous research in the field has used the term *general psychological distress* to connote elevations in partners' symptomatology in various domains (e.g., anxiety, depression, sleep problems). In the current study, efforts were made to use the term *psychological functioning* to convey that these symptoms were not in a clinical range. In an attempt to address this issue, Renshaw and colleagues have begun to develop a measure of general psychological distress to be used with partners of those with mental health conditions (Renshaw, Rodebaugh, & Rodrigues, 2010). It is suggested that future work in this line of research include a focus on being more exhaustive in its evaluation of partners' psychological functioning. Among a more exhaustive and standard set of assessments, it is recommended that positive domains of functioning, such as quality of life, also be investigated to better capture the experience of partners. Currently, the lack of standardization across studies has resulted in the term *general psychological distress* being used when referring to variety of symptom outcomes. By standardizing a battery of measures used to assess psychological functioning in partners and referring to specific validated constructs, the overall validity of these findings would increase by allowing for more accurate comparisons of results across studies.

The present study also did not include other measures that may be necessary for understanding how significant others influence each other during treatment. As mentioned earlier, this study could have benefitted from dyadic-level measures, such as relationship adjustment or satisfaction. In addition, more accurately capturing all aspects of the individual-level systems identified in C-BIT (i.e., cognitions, emotions, and behaviours) could have

provided a clearer picture of the way in which significant others influence each other. Future studies should consider the inclusion of both of these classes of variables in order to present a fuller picture of the model specified in C-BIT.

Conclusion

Overall, though the present study has a number of limitations, the integration of its findings into the existing literature provides important future directions to advance the field. The number of future directions that stem from this line of work range from measure development and psychometric analyses to replication and expansion of the current study, all of which would continue to extend and make tremendous contributions to the existing knowledge about the role of partners' psychological functioning as it relates to patients with PTSD and the influence between partners and patients in dyadic treatments more generally.

The results of the current study suggest that, on average, partners of individuals with PTSD do not evidence improvements in their psychological functioning after receiving a dyadic treatment for PTSD and relationship enhancement. Given the relatively low levels of distress in this population, it is suggested that an overall mean difference approach is not the best method by which to examine research in this area. Instead, it is encouraged that more descriptive, categorical, and clinical significance analyses be conducted to inform potential partner benefits in future research. Further, more closely monitoring and understanding partners' mental healthcare utilization may assist in understanding any additional partner benefits resulting from dyadic treatments.

In addition, the current results suggest that significant others' symptomatology do not mutually influence each other's treatment response. Although contrary to the associations suggested in C-BIT and in previous work, it is hypothesized that the lack of findings are likely

due to a failure to consider relationship-level variables in the current analyses. Nevertheless, the current study is among the first to suggest that the association between partners' psychological distress and patients' PTSD may be present in populations outside of military and veteran populations. Although underpowered, this represents the possible extension of this association to new populations, further demonstrating one of many interpersonal factors associated with PTSD.

As the interpersonal factors associated with PTSD continue to be investigated, the current study is particularly useful in suggesting variables that should be considered in future work. Primarily, the inclusion of relationship-level factors are likely essential to more fully understanding the role of significant others in PTSD treatments. Given the preliminary nature of this research, the results of the current study should be subjected to replication. With continued investigation of the interpersonal factors associated with PTSD, efforts can continue to be refined to ensure prevention, assessment, and treatment of PTSD extends not only to patients, but to their significant others, and the relationships they share.

Footnote

¹When examined independently by condition, exploratory posthoc analyses of partners' depressive symptoms in the collapsed sample significantly improved for partners who were initially randomized to waitlist ($M_{Diff} = 3.99$, $SD = 5.05$, $t(9) = 2.50$, $p < .05$), but not for those initially randomized to CBCT ($M_{Diff} = .92$, $SD = 3.78$, $t(11) = .84$, $p > .05$).

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