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PTSD and Romantic Relationship Satisfaction: Cluster- and Symptom-Level Analyses

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Abstract

Previous studies have demonstrated bidirectional associations between posttraumatic stress disorder (PTSD) and romantic relationship dissatisfaction. Most of these studies were focused at the level of the disorder, examining the association between relationship dissatisfaction and having a diagnosis of PTSD or the total of PTSD symptoms endorsed. This disorder-level approach is problematic for trauma theorists who posit symptom-level mechanisms for these effects. In the present study, we examined the prospective, bidirectional associations between PTSD symptom clusters (e.g., reexperiencing) and relationship satisfaction using the data from 101 previously studied individuals who had had a recent motor vehicle accident. We also conducted exploratory analyses examining the prospective, bidirectional associations between individual PTSD symptoms and relationship satisfaction. Participants had completed the PTSD Checklist-Civilian Version and the Relationship Assessment Scale at 4, 10, and 16 weeks after the MVA. We performed time-lagged mixed-effects regressions to examine the effect of lagged relationship satisfaction on PTSD clusters and symptoms, and vice versa. No cluster effects were significant after controlling for a false discovery rate. Relationship satisfaction predicted prospective decreases in reliving the trauma ($d = 0.42$), emotional numbness ($d = 0.46$), and irritability ($d = 0.49$). These findings were consistent with the position that relationship satisfaction affects PTSD through symptom-level mechanisms.

Posttraumatic stress disorder (PTSD; American Psychiatric Association [APA], 2013) is associated with impairments in romantic relationship satisfaction (Lambert, Engh, Hasbun, & Holzer, 2012; Taft, Watkins, Stafford, Street, & Monson, 2011)–an effect that has been increasingly recognized as bidirectional (Monson, Fredman, & Dekel, 2010). The mechanisms, however, by which PTSD and relationship problems affect one another remain poorly understood. One reason these mechanisms remain elusive may be that many

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researchers have typically approached the question at the level of absence or presence of a diagnosis of PTSD or symptom totals (see Lambert et al., 2012; Taft et al., 2011). This method fundamentally differs from an approach that analyses relationship satisfaction using individual symptoms as the independent variables. For example, Monson et al. (2010) have posited that PTSD symptoms such as behavioral avoidance and emotional numbing negatively affect relationships via reduced engagement in pleasurable activities or reduced expression of loving feelings (p. 180). Furthermore, Monson, Taft, and Fredman (2009) have proposed that social support, such as that provided by a romantic partner, may promote recovery from trauma by ameliorating specific consequences of PTSD, such as negative trauma-related appraisals or avoidance (p. 711). Research that exclusively examines the association between relationship satisfaction and presence or absence PTSD diagnosis, or overall PTSD symptom severity, cannot address symptom-level mechanisms.

One possible reason for the discrepancy in approaches is the dominance of the disease model of mental disorders (Deacon, 2013). The disease model conceptualizes mental disorders, including PTSD, as underlying conditions that give rise to observable symptoms (see Borsboom, 2008; Borsboom & Cramer, 2013; Deacon, 2013; McNally, 2012). Just as fever and sore throat may reflect an underlying influenza infection, the disease model holds that the symptoms of mental disorders reflect an underlying psychobiological abnormality (Borsboom, 2008; Borsboom & Cramer, 2013; Deacon, 2013). From a disease model perspective it makes sense to explore the association between relationship satisfaction and either a diagnosis of PTSD or PTSD symptom severity, as these indices are thought to reflect the presence and/or severity of the underlying condition responsible for PTSD. Thus, tacit acceptance of the disease model may explain prior failure to examine associations between romantic relationship satisfaction and individual PTSD symptoms.

Our claim is that the disease model is a latent variable model, in which symptoms are presumed to co-occur because they arise from a common cause (Borsboom & Cramer, 2013). Thus, this model holds that symptoms of PTSD cohere because they reflect the presence of an underlying condition which gives rise to the diagnostic entity of PTSD (McNally, 2012). There are, however, at least two limitations to this assumption for most mental disorders, including PTSD (see Borsboom, 2008; Borsboom & Cramer, 2013; Deacon, 2013; McNally et al., 2015; Robinaugh, LeBlanc, Vuletich, & McNally, 2014). First, despite the identification of numerous PTSD risk factors (Ozer, Best, Lipsey, & Weiss, 2003), the search for a single genetic or neurobiological marker to identify all cases of PTSD has been unsuccessful (see Pitman et al., 2012; Smoller, 2016). Indeed, researchers have been increasingly arguing that mental disorders are etiologically complex and unlikely to be explained by a simple underlying cause (Kendler, 2005). Second, this model ignores an alternative explanation for the tendency of PTSD symptoms to cluster together—the possibility that there are causal associations among the symptoms themselves. For example, cognitive–behavioral theories of PTSD propose that emotional reactivity to reminders of trauma may motivate behavioral avoidance, which may in turn increase emotional reactivity to reminders (e.g., Ehlers & Clark, 2000; Foa, Steketee, & Rothbaum, 1989). These plausible causal associations potentially undermine the rationale for attributing symptom covariance to a latent condition.

Given these limitations, Borsboom and Cramer (2013) argued for a new ontological model of mental disorders, termed the network or causal systems approach. They proposed that mental disorders are best conceptualized as self-sustaining networks of causally interconnected symptoms. From this perspective, the development of PTSD occurs through the activation of symptoms (e.g., trauma → intrusive thoughts of the trauma). Symptoms then give rise to other symptoms (e.g., intrusive thoughts of the trauma → intense psychological distress → efforts to avoid thoughts about the trauma → difficulty concentrating), and ultimately the emergence of the broader PTSD symptom network. Thus, symptoms and the relationships among them constitute the disorder. From this perspective, risk factors affect disorders by activating or reinforcing symptoms, or by strengthening the causal associations between symptoms (Borsboom & Cramer, 2013; Fried, Nesse, Zivin, Guile, & Sen, 2014; Robinaugh et al., 2014). Conversely, symptoms, once activated, can have a direct and variable impact on psychosocial outcomes (Fried & Nesse, 2014). For example, if the symptom of emotional numbing is activated within an individual's PTSD symptom network, the impact on his or her relationship satisfaction might be greater than if the difficulty falling asleep symptom is activated. The network approach thereby provides the rationale for examining the symptom level mechanisms posited by Monson et al. (2009, 2010).

Some prior attempts have been made to explore these symptom-level mechanisms, typically by investigating associations between PTSD symptom clusters and relationship problems (e.g., Campbell & Renshaw, 2013; Cook, Riggs, Thompson, & Coyne, 2004; Erbes, Meis, Polusny, Compton, & Wadsworth, 2012; Riggs, Byrne, Weathers, & Litz, 1998; Taft et al., 2009). Several cluster models of PTSD symptoms have been proposed, including the Elhai et al. (2011) 5-factor model (reexperiencing, avoidance, numbing, dysphoric arousal, and anxious arousal). Using cluster-level approaches, researchers have found that patients' scores on the numbing/dysphoria cluster were most strongly associated with relationship problems (Campbell & Renshaw, 2013; Cook et al., 2004; Erbes et al., 2012; Riggs et al., 1998). There is also evidence that a reexperiencing/avoidance cluster independently predicts partner physical aggression (partial $r = .16$), and that an arousal cluster independently predicts partner psychological aggression (partial $r = .24$; Taft et al., 2009).

These cluster-level findings represent an important step forward in our understanding of how PTSD symptoms may affect relationship satisfaction and vice versa. Published studies adopting this approach, however, have suffered from several limitations. First, although the associations between the PTSD clusters and relationship problems are presumed to be bidirectional, past longitudinal studies have only examined the prospective prediction of relationship satisfaction by the PTSD clusters and not the prediction of the PTSD clusters by relationship satisfaction (Campbell & Renshaw, 2013; Erbes et al., 2012). Accordingly, analyses examining the longitudinal prediction of the PTSD clusters by relationship satisfaction in a recently traumatized sample are needed to explore if and how relationship variables may affect the development of PTSD.

A second limitation of past cluster-level studies concerns the ontology of the clusters themselves. The Elhai et al. (2011) 5-factor model was confirmed using factor analysis, which implies the existence of a latent condition to which the clustering or covariance

between symptoms is attributed (Schmittmann et al., 2013). We contend that this model merely shifts the level of the explanatory variable from latent disease to latent cluster and therefore is vulnerable to many of the same limitations as the disease model of PTSD. For example, whereas some clusters (e.g., the avoidance cluster) contain symptoms that might be plausibly attributed to a common cause, others contain symptoms with plausible causal associations that may explain their covariance. Consider the dysphoric arousal cluster. Difficulty sleeping may cause concentration problems and irritability, which may lead to worry and further sleep interference. Similarly, with regard to the anxious arousal cluster, hypervigilance may cause increased startle response. As these examples make clear, more research is needed to examine the specific elements of the PTSD symptom network, as well as to clarify the nature of the relationships among the symptoms within this network. Given the current state of limited knowledge, however, a conservative approach is to consider each of the PTSD symptoms as separate psychological phenomena with distinct risk and impairment profiles.

The aims of the present study were to address gaps in the literature on the bidirectional relationship between romantic relationship satisfaction and PTSD. We first sought to extend past cluster-level studies by examining the longitudinal, bidirectional associations between relationship satisfaction and the PTSD symptoms clusters using existing data from a recently traumatized sample. We then sought to conduct an initial inquiry into the longitudinal, bidirectional relationships between relationship satisfaction and the individual symptoms of PTSD. We expected that symptom-level analyses would afford a greater level of precision than cluster-level analyses—either by illuminating significant symptom effects that were masked by non-significant clusters, or by identifying significant versus non-significant symptom effects within significant clusters.

Method

Participants and Procedure

We analyzed archival data collected as part of a doctoral dissertation on the interrelationship between social support, romantic relationship variables, and PTSD, following a serious motor vehicle accident (MVA; Marques Miller, 2007). Participants in the previous study were individuals from the greater Buffalo, New York area who were involved in an injury-related MVA within 1 month of participation and whose response to the MVA was characterized by fear, helplessness, horror, or the perception that they could have died in the accident (Criterion A for PTSD according to the *Diagnostic and Statistical Manual of Mental Disorders,* 4th ed., *DSM-IV*; APA, 1994). In addition, participants were in monogamous, heterosexual romantic relationships of at least 4-month duration at study entry. Exclusion criteria for the larger study included (a) non-English speaking, (b) ongoing physical or emotional abuse, (c) current treatment for substance use disorders, (d) impaired cognitive functioning, (e) psychotic symptoms, (f) ongoing divorce, (g) driving while intoxicated accidents, or (h) suicidal ideation that required immediate intervention.

There were 2,373 potential participants who had been identified through public MVA records and had been mailed a letter of invitation within 2 weeks of their accident. Of those who received a letter, 199 responded (response rate = 8.4%), and 111 met full inclusion/

exclusion criteria. They completed questionnaires at the first study time point (4 weeks post-MVA). Of those, 101 completed the second study time point (10 weeks post-MVA) and 100 completed the third study time point (16 weeks post-MVA). The data from the 101 participants who completed at least the first and second time points were used in this study.

The mean age was 38.30 years (*SD* = 12.93). Participants were predominantly female (*n* = 75; 74.3%) and Caucasian (*n* = 75; 74.3%). With regard to relationship status, 46 participants were married (45.5%; relationship lengths ranged from 4 to 552 months), 9 were engaged (8.9%; 8–60 months), 9 were cohabitating (8.9%; 18–300 months), and 37 were dating (36.6%; 4–264 months).

The Institutional Review Board of the State University of New York at Buffalo approved procedures for the original study. Detailed procedures have been reported elsewhere (Marques Miller, 2007). Respondents to the initial letter completed phone screening within 3 weeks of their MVA. Informed consent was obtained during the phone screening. Eligible participants then received packets of self-report questionnaires by mail at three time points: 4, 10, and 16 weeks post-MVA. The initial packet also included an informed consent form that participants signed and returned. Participants were tracked longitudinally in a Microsoft Excel database via participant ID numbers. The dates of questionnaire packet completion were documented in this database and were used to calculate the time between questionnaire packets. Total compensation for the study was $60: $10 for the first packet, $10 for the second packet, and $40 for the third packet.

Measures

The PTSD Checklist, Civilian Version (PCL-C; Weathers, Litz, Herman, Huska, & Keane, 1993) is a 17-item self-report measure of PTSD symptom severity. Respondents rated each of the 17 *DSM-IV* PTSD symptoms over the past month on a 5-point scale with the following anchors: 1 = *not at all*, 2 = *a little bit*, 3 = *moderately*, 4 = *quite a bit*, and 5 = *extremely.* The PCL-C total score is the sum of the 17 items (range = 17 to 85). Higher scores indicate more severe PTSD symptoms. A score of > 44 on the PCL-C is one of several scores used to categorize probable PTSD in MVA survivors (Blanchard, Jones-Alexander, Buckley, & Forneris, 1996). Participants' PTSD cluster scores were calculated as the mean of the PCL-C items that comprise each cluster. The individual PCL-C items served as severity measures for the individual PTSD symptoms. Internal consistency for the PCL-C was excellent at the first (α = .94), second (α = .94), and third time point (α = .95). Internal consistency for the clusters was acceptable at the first (αs = .75 to .91), second (αs = .74 to .92), and third time point (αs = .80 to .93).

The Relationship Assessment Scale (RAS; Hendrick, 1988) is a 7-item self-report measure of romantic relationship satisfaction over the past month. To complete the RAS, respondents rated each item on a 5-point scale with variable anchors for each item. For example, one question asks, "[Over the past month] how well did your partner meet your needs?" Participants indicated their responses on the following scale: 1 = *A – poorly*, 2 = *B*, 3 = *C – average*, 4 = *D*, 5 = *E –extremely well*. The RAS score is the average of the seven items (range = 1 to 5). Higher scores indicate greater relationship satisfaction. Internal consistency was excellent at the first (α = .90), second (α = .90), and third time point (α = .92).

Data Analysis

Data were screened for missing values. One participant did not complete the third time point, but was included in all models. One participant was missing RAS data at the second time point and was therefore dropped from analyses in which lagged PTSD variables predicted RAS. For these models, $n = 100$.

Time-lagged mixed-effects regression modeling was employed to examine the prospective prediction of the PTSD clusters by relationship satisfaction and vice versa, as well as to examine the prospective prediction of the individual PTSD symptoms by relationship satisfaction and vice versa. Lagged regression models test the effect of X_{T1} on Y_{T2} while controlling for the autoregression of Y (i.e., the effect of Y_{T1} on Y_{T2}). In the present study, lagged effects represent an estimate derived from the prediction of the week 10 dependent variable (DV) by the week 4 independent variable (IV) (adjusting for the week 4 DV) and the prediction of the week 16 DV by the week 10 IV (adjusting for the week 10 DV). Autoregressions of X (i.e., the effect of X_{T1} on X_{T2}) were not adjusted for due to concerns that collinearity of contemporaneous and lagged IVs would suppress lagged effects. See Zalta et al. (2014) for similar analyses.

To examine the time-lagged effect of RAS on the PTSD clusters, we ran five mixed-effects regression models with time-lagged RAS as a predictor of each cluster at the subsequent time point, adjusting for the autoregressions of each cluster. To test the competing directional hypothesis, we ran five more mixed-effects regression models using each cluster as a time-lagged predictor of RAS adjusting for the autoregressions of RAS. To examine the time-lagged effect of RAS on the PTSD symptoms, we ran 17 mixed-effects regression models using time-lagged RAS as a predictor of each PCL-C item, adjusting for the autoregressions of each PCL-C item. We then ran 17 mixed-effects regression models using each PCL-C item as a time-lagged predictor of RAS adjusting for the autoregressions of RAS. All models included the intercept as a random effect. The linear effect of time and the random effect of slope were not included in any models due to an insufficient number of time points to model trajectories. Models were estimated with maximum likelihood estimation using the lme4 package in R (Version 3.2.1) software (R Core Team, 2015).

Cluster-level analyses resulted in 10 separate hypothesis tests, and symptom-level analyses resulted in 34 separate hypothesis tests. We therefore controlled the false discovery rate (FDR) for each group of tests via correction with the B-H procedure (Benjamini & Hochberg, 1995). FDR was set to $q^* = .05$. Given the exploratory nature of the present study, both the uncorrected and corrected results are presented.

Results

Mean PCL-C total scores for the sample were 39.21 ($SD = 15.43$, 40.6% above diagnostic cutoff of 44), 35.52 ($SD = 14.83$; 24.8% above a diagnostic cutoff of 44), and 32.58 ($SD = 14.89$; 24.8% above a diagnostic cutoff of 44), at all three time points, respectively. PCL-C total scores decreased over 4 to 10 weeks post-MVA, $t(100) = 3.20$, $p = .002$, and from 10 to 16 weeks post-MVA, $t(99) = 3.02$, $p = .003$. See Supplementary Table 1 for descriptive statistics for the cluster scores and individual PCL-C items at the three time points. Mean

RAS scores for the sample were 3.82 (*SD* = 0.88), 3.79 (*SD* = 0.92), and 3.81 (*SD* = 0.97) at all three time points, respectively. RAS scores did not change from 4 to 10 weeks post-MVA, $t(99) = 0.59$, $p = .557$, or from 10 to 16 weeks post MVA, $t(98) = 0.35$, $p = .724$.

The results of time-lagged mixed-effects regression models predicting the PTSD clusters by RAS scores are presented in Table 1. Autoregressions for the clusters were large (*ds* ranged from 1.29 to 1.95, *ps* < .001). Using uncorrected results, higher RAS scores predicted later declines in participants' cluster scores for numbing and dysphoric arousal. After correction with the B-H procedure, neither effect was significant. The results of time-lagged mixed-effects regression models predicting RAS by each of the clusters are presented in Table 2. Autoregressions for RAS were large (*d*s ranged from 2.27 to 2.39, *p*s < .001). None of the lagged effects of uncorrected or corrected on RAS were significant.

The results of time-lagged mixed-effects regression models predicting the individual PTSD symptoms by lagged RAS scores are presented in Table 3. Autoregressions for the symptoms were large (*d*s range 0.82 to 1.81, *p*s < .001). In the uncorrected results, higher RAS scores were significantly related to later decreases in six symptoms: intrusive thoughts, reliving the trauma, social disconnection, emotional numbness, difficulty sleeping, and irritability. Following correction with the B-H procedure, however, only effects were significant: reliving the trauma, emotional numbness, and irritability.

The results of time-lagged mixed-effects regression models predicting RAS scores by lagged symptoms are presented in Table 4. The autoregressions for RAS scores were large (*d*s ranged from 2.16 to 2.4, *p*s < . 001). There were no lagged effects of symptoms on RAS scores, uncorrected or corrected.

Discussion

The present study explored the longitudinal, bidirectional associations between PTSD symptoms and clusters and relationship satisfaction. In uncorrected cluster-level analyses, relationship satisfaction predicted decreases in participants' scores on two clusters: numbing and dysphoric arousal. Adjusting for experiment-wise alpha inflation rendered them nonsignificant. In uncorrected symptom-level analyses, relationship satisfaction was associated with decreases in six symptoms, three of which remained significant after controlling the FDR: reliving the trauma, emotional numbness, and irritability.

Notably, none of the PTSD variables was related to declines in relationship satisfaction during the study period. There are at least two possible explanations for these findings. First, these null results may have been statistical artifacts caused by the greater reliability of the RAS compared to the cluster and symptom measures. Internal consistency was lower for the PTSD clusters than for the RAS, though still in the acceptable range. Reliability could not be calculated for symptoms, as symptoms were assessed via individual PCL-C items. Though single-item measures do not necessarily suffer from low reliability (e.g., Zimmerman et al., 2006), reliability does increase with test length (Furr & Bacharach, 2008, p. 124). As a result, if measurement error were a factor, it likely affected the PCL-C items more than the RAS, though the extent of this effect is unknown. As an anonymous reviewer of this article

noted, greater reliability of the RAS could have resulted in greater autoregressive effects for this measure, and therefore less variance to be accounted for by cluster-level or symptom-level predictors. Indeed, autoregressive effects for the RAS were larger than those for the cluster-level or symptom-level variables. Another possibility, however, was that larger autoregressive effects for the RAS simply reflected less naturally occurring change of relationship satisfaction compared to PTSD symptoms during the study. Consistent with this hypothesis, previous studies have shown that social support predicted change in PTSD symptoms in the initial months posttrauma, whereas the deleterious effects of PTSD symptoms on social support did not emerge until symptoms had persisted for approximately 1 year (Kaniatsy & Norris, 2008). It will be important for future studies to adjudicate between these two explanations. Development of multiple-item measures of symptoms may help to resolve questions of measurement error, and the extension of these analyses to a dataset with a longer follow-up may help to clarify the effects of symptoms on relationship satisfaction over time.

As anticipated, our symptom-level analyses yielded increased precision over cluster-level analyses. Specifically, we observed a significant prospective effect of relationship satisfaction on symptoms from three distinct clusters: reliving the trauma (reexperiencing cluster), emotional numbness (numbing cluster), and irritability (dysphoric arousal cluster). Reliving the trauma was an instance of the claim we have presented: it demonstrated how a nonsignificant cluster effect could be misleading if the items were not examined individually. Emotional numbness and irritability showed stronger relationships than the corresponding cluster-level analyses, though other symptoms from the numbing and dysphoric arousal clusters were not significantly linked to relationship satisfaction. These results are consistent with network theory (Borsboom & Cramer, 2013), as researchers taking this approach have argued that risk factors should affect mental disorders not by influencing a latent entity (e.g., a latent disorder or symptom cluster), but by directly influencing individual symptoms (Fried et al., 2014; Robinaugh et al., 2014). This point has been demonstrated empirically for depression symptoms through the use of structural equation modeling (SEM; Fried et al., 2014). Specifically, researchers have demonstrated that SEMs, in which the prospective effects of risk factors on depression symptoms are free to vary, provide a better fit for the data than models in which these prospective effects are constrained to be equal across symptoms (Fried et al., 2014). Our modest sample size precluded us from undertaking a similar direct comparison of the cluster-level and symptom-level risk factor models. The present results, however, provided initial evidence that relationship satisfaction may have influence the development of some PTSD symptoms through its effect on individual symptoms (rather than symptom clusters).

Although observed symptom-level effects are not necessarily indicative of causal pathways, the present results can inform the development of testable hypotheses about potential symptom-level mechanisms. For example, relationship satisfaction was negatively related to the severity with which participants endorsed reliving the trauma. One possibility was that a supportive partner helped correct survivors' negative appraisals of reliving experiences (see Monson et al., 2009), which serve to maintain this symptom (Ehlers & Clark, 2000). Relationship satisfaction also predicted decline in emotional numbness. This result suggested that the documented association between relationship satisfaction and the

numbing cluster may have been largely driven by the symptom of emotional numbness symptom. Furthermore, this finding suggested that relationship satisfaction may have promoted recovery from emotional numbness posttrauma, perhaps through the provision and elicitation of loving feelings. Finally, we observed a negative effect of relationship satisfaction on irritability. Variation in dyadic conflict may have accounted for this effect. A top priority for future research will be to examine the causal nature of these potential symptom-level mechanisms, either through intra-individual network analyses with time-series data collected daily during the first year posttrauma or experimental manipulation of survivors' relationship satisfaction.

Our study had a number of limitations. First, the response rate of 8.4% to the initial study invitation disappointingly low, likely limiting the generalizability of the present findings. Second, we used single items to measure individual PTSD symptoms, potentially increasing measurement error and affecting results. Third, we did not collect contemporaneous data on partners' relationship satisfaction or emotional distress—data that may have further clarify PTSD etiology given that symptom networks may interact with other symptom networks (e.g., folie à deux; Borsboom & Cramer, 2013). Fourth, the causal nature of the effects in the present study was unclear. Statisticians have questioned regression-based methods of causality testing (c.f. Sekhon, 2009). Accordingly, the lagged regression modeling employed here should be considered a preliminary step toward establishing causal connections that may contribute to the development and maintenance of PTSD. Additionally, the associations between relationship satisfaction and PTSD symptoms (or clusters) observed here may have arisen due to direct predictive associations or due to shared associations among relationship satisfaction, a given PTSD symptom (or cluster), and a third variable, such as another PTSD symptom (or cluster). Our sample size of 101 precluded the use of a more comprehensive statistical model to address this limitation. Fifth, the present study had a relatively truncated assessment period. Sixth, our analyses were exploratory; therefore, replication in an independent sample is critically important.

It may be beneficial to determine if the present results could enhance treatment of a couple where one member has full or partial PTSD. Cognitive-Behavioral Conjoint Therapy (CBCT) for PTSD is a couple treatment that educates the couple about "the reciprocal influences of PTSD symptoms and relationship functioning" (Monson et al., 2012, p. 702), and has been effective in simultaneously reducing patients' PTSD symptoms and improving relationship satisfaction. The present results could be used to further inform the CBCT approach. For example, it may be useful clinicians who provide CBCT clinicians to explore whether partners' reactions to flashbacks are contributing to the negative appraisals that maintain this symptom. Clinicians may also find it helpful to have patients track symptoms such as emotional numbness and irritability, and to notice dyadic events (e.g., being ignored, receiving criticism) that typically precede an increase in symptoms. These interventions may help patients and partners develop a deeper understanding of the connection between their own relationship satisfaction and PTSD symptoms.

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References

American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 4. Washington, DC: Author; 1994.

American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 5. Arlington, VA: Author; 2013.

Benjamini Y, Hochberg Y. Controlling the false discovery rate: A practical and powerful approach to multiple testing. Journal of the Royal Statistical Society: Series B (Methodological). 1995; 57:289–300. http://www.jstor.org/stable/2346101.

Blanchard EB, Jones-Alexander J, Buckley TC, Forneris CA. Psychometric properties of the PTSD Checklist (PCL). Behaviour Research and Therapy. 1996; 34:669–673. DOI: 10.1016/0005-7967(96)00033-2 [PubMed: 8870294]

Borsboom D. Psychometric perspectives on diagnostic systems. Journal of Clinical Psychology. 2008; 64:1089–1108. DOI: 10.1002/jclp.20503 [PubMed: 18683856]

Borsboom D, Cramer AOJ. Network analysis: An integrative approach to the structure of psychopathology. Annual Review of Clinical Psychology. 2013; 9:91–121. DOI: 10.1146/annurev-clinpsy-050212-185608

Campbell SB, Renshaw KD. PTSD symptoms, disclosure, and relationship distress: Explorations of mediation and associations over time. Journal of Anxiety Disorders. 2013; 27:494–502. DOI: 10.1016/j.janxdis.2013.06.007 [PubMed: 23917126]

Cook JM, Riggs DS, Thompson R, Coyne JC. Posttraumatic stress disorder and current relationship functioning among World War II ex-prisoners of war. Journal of Family Psychology. 2004; 18:36–45. DOI: 10.1037/0893-3200.18.1.36 [PubMed: 14992608]

Deacon BJ. The biomedical model of mental disorders: A critical analysis of its validity, utility, and effects on psychotherapy research. Clinical Psychology Review. 2013; 33:846–861. DOI: 10.1016/j.cpr.2012.09.007 [PubMed: 23664634]

Ehlers A, Clark DM. A cognitive model of posttraumatic stress disorder. Behaviour Research and Therapy. 2000; 38:319–345. DOI: 10.1016/S0005-7967(99)00123-0 [PubMed: 10761279]

Elhai JD, Biehn TL, Armour C, Klopper JJ, Frueh BC, Palmieri PA. Evidence for a unique PTSD construct represented by PTSD's D1–D3 symptoms. Journal of Anxiety Disorders. 2011; 25:340–345. DOI: 10.1016/j.janxdis.2010.10.007 [PubMed: 21094021]

Erbes CR, Meis LA, Polusny MA, Compton JS, Wadsworth SM. An examination of PTSD symptoms and relationship functioning in U.S. soldiers of the Iraq war over time. Journal of Traumatic Stress. 2012; 25:187–190. DOI: 10.1002/jts.21689 [PubMed: 22522733]

Foa EB, Steketee G, Rothbaum BO. Behavioral/cognitive conceptualizations of post-traumatic stress disorder. Behavior Therapy. 1989; 20:155–176. DOI: 10.1016/S0005-7894(89)80067-X

Fried EI, Nesse RM. The impact of individual depressive symptoms on impairment of psychosocial functioning. PLoS ONE. 2014; 9(2):e90311.doi: 10.1371/journal.pone.0090311 [PubMed: 24587318]

Fried EI, Nesse RM, Zivin K, Guile C, Sen S. Depression is more than the sum of its parts: Individual DSM symptoms have different risk factors. Psychological Medicine. 2014; 44:2067–2076. DOI: 10.1017/S0033291713002900 [PubMed: 24289852]

Furr, RM., Bacharach, VR. Psychometrics: An introduction. Los Angeles, CA: Sage; 2008.

Hendrick SS. A generic measure of relationship satisfaction. Journal of Marriage and Family. 1988; 50:93–98. DOI: 10.2307/352430

Kaniatsy K, Norris FH. Longitudinal linkages between perceived social support and posttraumatic stress symptoms: Sequential roles of social causation and social selection. Journal of Traumatic Stress. 2008; 21:274–281. DOI: 10.1002/jts.20334 [PubMed: 18553415]

Kendler KS. Toward a philophical structure for psychiatry. American Journal of Psychiatry. 2005; 162:433–440. DOI: 10.1176/appi.ajp.162.3.433 [PubMed: 15741457]

Lambert JE, Engh R, Hasbun A, Holzer J. Impact of posttraumatic stress disorder on the relationship quality and psychological distress of intimate partners: A meta-analytic review. Journal of Family Psychology. 2012; 26:729–737. DOI: 10.1037/a0029341 [PubMed: 22924422]

Marques Miller, L. Unpublished doctoral dissertation. The State University of New York; Buffalo, Buffalo, NY: 2007. The interrelationship between social support and post-trauma symptoms in the aftermath of a severe motor vehicle accident.

McNally RJ. The ontology of posttraumatic stress disorder: Natural kind, social construction, or causal system? Clinical Psychology Science and Practice. 2012; 19:220–228. DOI: 10.1111/cpsp.12001

McNally RJ, Robinaugh DJ, Wu WYW, Wang L, Deserno M, Borsboom D. Mental disorders as causal system: A network approach to posttraumatic stress disorder. Clinical Psychological Science. 2015; 3:836–849. DOI: 10.1177/2167702614553230

Monson, CM., Fredman, SJ., Dekel, R. Posttraumatic stress disorder in an interpersonal context. In: Beck, JG., editor. Interpersonal processes in the anxiety disorders. Washington, DC: American Psychological Association; 2010.

Monson CM, Fredman SJ, Macdonald A, Pukay-Martin ND, Resick PA, Schnurr PP. Effects of cognitive-behavioral couple therapy for PTSD: A randomized controlled trial. Journal of the American Medical Association. 2012; 308:700–709. DOI: 10.1001/jama.2012.9307 [PubMed: 22893167]

Monson CM, Taft CT, Fredman SJ. Military-related PTSD and intimate relationships: From description to theory-driven research and intervention development. Clinical Psychology Review. 2009; 29:707–714. DOI: 10.1016/j.cpr.2009.09.002 [PubMed: 19781836]

Ozer EJ, Best SR, Lipsey TL, Weiss DS. Predictors of posttraumatic stress disorder and symptoms in adults: A meta-analysis. Psychological Bulletin. 2003; 129:52–73. DOI: 10.1037/0033-2909.129.1.52 [PubMed: 12555794]

Pitman RK, Rasmussan AM, Koenen KC, Shin LS, Orr SC, Gilbertson MW, … Liberzon I. Biological studies of posttraumatic stress disorder. Nature Reviews Neuroscience. 2012; 13:769–787. DOI: 10.1038/nrn3339 [PubMed: 23047775]

R Core, Team. R: A language and environment for statistical computing. R Foundation for Statistical Computing; Vienna, Austria: 2015. https://www.R-project.org/

Riggs DS, Byrne CA, Weathers FW, Litz B. The quality of intimate relationships of male Vietnam veterans: Problems associated with posttraumatic stress disorder. Journal of Traumatic Stress. 1998; 11:87–101. DOI: 10.1023/A:1024409200155 [PubMed: 9479678]

Robinaugh DJ, LeBlanc NJ, Vuletich HA, McNally RJ. Network analysis of persistent complex bereavement disorder in conjugally bereaved adults. Journal of Abnormal Psychology. 2014; 123:510–522. DOI: 10.1037/abn0000002 [PubMed: 24933281]

Schmittmann VD, Cramer AOJ, Waldorp LJ, Epskamp S, Kievit RA, Borsboom D. Deconstructing the construct: A network perspective on psychological phenomena. New Ideas in Psychology. 2013; 31:45–53. DOI: 10.1016/j.newideapsych.2011.02.007

Sekhon JS. Opiates for the matches: Matching methods for causal inference. Annual Review of Political Science. 2009; 12:487–508. DOI: 10.1146/annurev.polisci.11.060606.135444

Smoller JW. The genetics of stress-related disorders: PTSD, depression, and anxiety disorders. Neuropsychopharmacology Reviews. 2016; 41:297–319. [PubMed: 26321314]

Taft CT, Watkins LE, Stafford J, Street AE, Monson CM. Posttraumatic stress disorder and intimate relationship problems: A meta-analysis. Journal of Consulting and Clinical Psychology. 2011; 79:22–33. DOI: 10.1037/a0022196 [PubMed: 21261431]

Taft CT, Weatherill RP, Woodward HE, Pinto LA, Miller MW, Dekel R. Intimate partner and general aggression perpetration among combat veterans presenting to a posttraumatic stress disorder clinic. American Journal of Orthopsychiatry. 2009; 79:461–468. DOI: 10.1037/a0016657 [PubMed: 20099937]

Weathers, FW., Litz, B., Herman, DS., Huska, JA., Keane, TM. The PTSD Checklist (PCL): Reliability, validity, and diagnostic utility. Paper presented at the 9th Annual Conference of the International Society for Traumatic Stress Studies; San Antonio, TX. 1993 Oct.

Zalta AK, Gilihan SJ, Fisher AJ, Mintz AJ, McLean CP, Yehuda R, Foa EB. Change in negative cognitions associated with PTSD predicts symptom reduction in prolonged exposure. Journal of Consulting and Clinical Psychology. 2014; 82:171–175. DOI: 10.1037/a0034735 [PubMed: 24188512]

Zimmerman M, Ruggero CJ, Chelminski I, Young D, Posternak MA, Friedman M, … Attiullah N. Developing brief scales for use in clinical practice: The reliability and validity of single-item self-report measures of depression symptom severity, psychosocial impairment due to depression, and quality of life. Journal of Clinical Psychiatry. 2006; 67:1536–1541. DOI: 10.4088/JCP.v67n1007 [PubMed: 17107244]

Table 1

Time-Lagged Mixed-Effects Regressions Predicting PTSD Symptom Clusters by Lagged Relationship Satisfaction

Variable	*B*	*SE*	*d*
Reexperiencing on RAS			
Intercept	0.96	0.27	0.50**
Reexperiencing autoregression	0.67	0.05	1.95***
Lagged RAS	−0.11	0.06	0.28
Avoidance on RAS			
Intercept	0.93	0.32	0.41**
Avoidance autoregression	0.58	0.06	1.43***
Lagged RAS	−0.04	0.07	0.09
Numbing on RAS			
Intercept	1.04	0.27	0.55***
Numbing autoregression	0.61	0.05	1.56***
Lagged RAS	−0.11	0.06	0.29*
Dysphoric arousal on RAS			
Intercept	1.27	0.29	0.62***
Dysphoric arousal autoregression	0.64	0.05	1.92***
Lagged RAS	−0.16	0.06	0.37*
Anxious arousal on RAS			
Intercept	1.14	0.34	0.48**
Anxious arousal autoregression	0.52	0.06	1.29***
Lagged RAS	−0.06	0.08	0.11

Note. $N = 101$. Cohen's $d = t * \sqrt{(2/N)}$. PTSD = posttraumatic stress disorder; RAS = Relationship Assessment Scale.

* $p < .05$.

** $p < .01$.

*** $p < .001$.

Table 2

Time-Lagged Mixed-Effects Regressions Predicting Relationship Satisfaction by Lagged PTSD Symptom Clusters

Variable	*B*	*SE*	*d*
RAS on reexperiencing			
Intercept	0.74	0.23	0.47**
RAS autoregression	0.80	0.05	2.35***
Lagged reexperiencing	−0.00	0.04	0.01
RAS on avoidance			
Intercept	0.78	0.21	0.51***
RAS autoregression	0.80	0.05	2.37***
Lagged avoidance	−0.02	0.04	0.06
RAS on numbing			
Intercept	0.70	0.24	0.40**
RAS autoregression	0.81	0.05	2.27***
Lagged numbing	0.01	0.05	0.03
RAS on dysphoric arousal			
Intercept	0.60	0.23	0.36*
RAS autoregression	0.82	0.05	2.35***
Lagged dysphoric arousal	0.04	0.04	0.13
RAS on anxious arousal			
Intercept	0.69	0.21	0.46**
RAS autoregression	0.81	0.05	2.39***
Lagged anxious arousal	0.01	0.04	0.05

Note. $n = 100$. Cohen's $d = t * \sqrt{(2/N)}$. PTSD = posttraumatic stress disorder; RAS = Relationship Assessment Scale.

* $p < .05$.

** $p < .01$.

*** $p < .001$.

Table 3

Time-Lagged Mixed-Effects Regressions Predicting Individual PTSD Symptoms by Lagged Relationship Satisfaction

Variable	*B*	*SE*	*d*
PCL-C1 on RAS			
Intercept	1.61	0.35	0.65***
PCL-C1 autoregression	0.49	0.06	1.25***
Lagged RAS	−0.17	0.08	0.31*
PCL-C2 on RAS			
Intercept	0.94	0.31	0.43**
PCL-C2 autoregression	0.57	0.05	1.63***
Lagged RAS	−0.07	0.07	0.13
PCL-C3 on RAS			
Intercept	1.61	0.31	0.72***
PCL-C3 autoregression	0.52	0.05	1.34***
Lagged RAS	−0.21[a]	0.07	0.42**
PCL-C4 on RAS			
Intercept	1.34	0.36	0.52***
PCL-C4 autoregression	0.60	0.06	1.51***
Lagged RAS	−0.15	0.08	0.25
PCL-C5 on RAS			
Intercept	1.12	0.32	0.48**
PCL-C5 autoregression	0.58	0.05	1.56***
Lagged RAS	−0.11	0.07	0.21
PCL-C6 on RAS			
Intercept	0.98	0.37	0.37*
PCL-C6 autoregression	0.52	0.06	1.22***
Lagged RAS	−0.02	0.08	0.03
PCL-C7 on RAS			
Intercept	1.23	0.34	0.51**
PCL-C7 autoregression	0.51	0.06	1.23***
Lagged RAS	−0.09	0.08	0.15
PCL-C8 on RAS			
Intercept	1.42	0.31	0.64***
PCL-C8 autoregression	0.43	0.06	1.08***
Lagged RAS	−0.13	0.07	0.24
PCL-C9 on RAS			
Intercept	1.21	0.33	0.51***
PCL-C9 autoregression	0.58	0.05	1.54***

Variable	*B*	*SE*	*d*
Lagged RAS	−0.14	0.07	0.26
PCL-C10 on RAS			
Intercept	1.34	0.33	0.57***
PCL-C10 autoregression	0.64	0.05	1.70***
Lagged RAS	−0.19	0.07	0.37*
PCL-C11 on RAS			
Intercept	1.84	0.36	0.73***
PCL-C11 autoregression	0.42	0.06	0.94***
Lagged RAS	−0.25[a]	0.08	0.46**
PCL-C12 on RAS			
Intercept	1.18	0.31	0.53***
PCL-C12 autoregression	0.35	0.06	0.82***
Lagged RAS	−0.04	0.07	0.09
PCL-C13 on RAS			
Intercept	1.45	0.36	0.56***
PCL-C13 autoregression	0.63	0.05	1.81***
Lagged RAS	−0.16	0.08	0.28*
PCL-C14 on RAS			
Intercept	1.73	0.33	0.74***
PCL-C14 autoregression	0.54	0.05	1.42***
Lagged RAS	−0.25[a]	0.07	0.49**
PCL-C15 on RAS			
Intercept	1.39	0.38	0.51***
PCL-C15 autoregression	0.55	0.06	1.39***
Lagged RAS	−0.15	0.08	0.25
PCL-C16 on RAS			
Intercept	1.46	0.39	0.52***
PCL-C16 autoregression	0.38	0.06	0.86***
Lagged RAS	−0.05	0.09	0.08
PCL-C17 on RAS			
Intercept	1.02	0.34	0.42**
PCL-C17 autoregression	0.58	0.05	1.52***
Lagged RAS	−0.07	0.08	0.13

Note. $N = 101$. Cohen's $d = t * \sqrt{(2/N)}$. PTSD = posttraumatic stress disorder; *SE* = standard error; RAS = Relationship Assessment Scale; PCL-C = PTSD Checklist, Civilian Version; PCL-C1 = intrusive thoughts; PCL-C2 = disturbing dreams; PCL-C3 = reliving the trauma; PCL-C4 = emotional reactivity; PCL-C5 = physical reactivity; PCL-C6 = avoiding thoughts; PCL-C7 = avoiding activities; PCL-C8 = traumatic amnesia; PCL-C9 = loss of interest; PCL-C10 = social disconnection; PCL-C11 = emotional numbness; PCL-C12 = foreshortened future; PCL-C13 = difficulty sleeping, PCL-C14 = irritability; PCL-C15 = difficulty concentrating; PCL-C16 = hypervigilance; PCL-C17 = hyperarousal.

[a]Denotes that the lagged effect remained significant when the false discovery rate is controlled at $q^* = 0.05$, per the Benjamini and Hochberg (1995) procedure.

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*$p < .05$.

**$p < .01$.

***$p < .001$.

Table 4

Time-Lagged Mixed-Effects Regressions Predicting Relationship Satisfaction by Lagged Individual PTSD Symptoms

Variable	*B*	*SE*	*d*
RAS on PCL-C1			
Intercept	0.85	0.22	0.54***
RAS autoregression	0.80	0.05	2.35***
Lagged PCL-C1	−0.03	0.04	0.14
RAS on PCL-C2			
Intercept	0.65	0.22	0.43**
RAS autoregression	0.81	0.05	2.38***
Lagged PCL-C2	0.03	0.03	0.10
RAS on PCL-C3			
Intercept	0.79	0.22	0.51***
RAS autoregression	0.80	0.05	2.33***
Lagged PCL-C3	−0.02	0.04	0.07
RAS on PCL-C4			
Intercept	0.73	0.22	0.47**
RAS autoregression	0.81	0.05	2.36***
Lagged PCL-C 4	0.001	0.03	0.00
RAS on PCL-C5			
Intercept	0.71	0.21	0.48**
RAS autoregression	0.81	0.05	2.38***
Lagged PCL-C5	0.01	0.03	0.03
RAS on PCL-C6			
Intercept	0.77	0.21	0.52***
RAS autoregression	0.80	0.05	2.37***
Lagged PCL-C6	−0.01	0.03	0.06
RAS on PCL-C7			
Intercept	0.77	0.21	0.52***
RAS autoregression	0.80	0.05	2.37***
Lagged PCL-C7	−0.01	0.04	0.06
RAS on PCL-C8			
Intercept	0.78	0.21	0.53***
RAS autoregression	0.80	0.05	2.36***
Lagged PCL-C8	−0.02	0.04	0.08
RAS on PCL-C9			
Intercept	0.77	0.22	0.49**
RAS autoregression	0.80	0.05	2.31***

Variable	*B*	*SE*	*d*
Lagged PCL-C9	−0.01	0.04	0.04
RAS on PCL-C10			
Intercept	0.66	0.23	0.40**
RAS autoregression	0.81	0.05	2.29***
Lagged PCL-C10	0.02	0.04	0.08
RAS on PCL-C11			
Intercept	0.75	0.25	0.43**
RAS autoregression	0.80	0.05	2.16***
Lagged PCL-C11	−0.01	0.04	0.02
RAS on PCL-C12			
Intercept	0.60	0.21	0.41**
RAS autoregression	0.82	0.05	2.40***
Lagged PCL-C12	0.05	0.04	0.19
RAS on PCL-C13			
Intercept	0.63	0.21	0.42**
RAS autoregression	0.81	0.05	2.40***
Lagged PCL-C13	0.03	0.03	0.14
RAS on PCL-C14			
Intercept	0.62	0.23	0.39**
RS autoregression	0.82	0.05	2.35***
Lagged PCL-C14	0.03	0.04	0.12
RAS on PCL-C15			
Intercept	0.66	0.23	0.40**
RAS autoregression	0.81	0.05	2.30***
Lagged PCL-C15	0.02	0.03	0.07
RAS on PCL-C16			
Intercept	0.72	0.21	0.49**
RS autoregression	0.81	0.05	2.39***
Lagged PCL-C16	0.01	0.03	0.03
RAS on PCL-C17			
Intercept	0.68	0.21	0.45**
RAS autoregression	0.81	0.05	2.38***
Lagged PCL-C17	0.02	0.03	0.07

Note. $n = 100$. Cohen's $d = t * \sqrt{(2/N)}$. PTSD = posttraumatic stress disorder; *SE* = standard error; RAS = Relationship Assessment Scale; PCL-C = PTSD Checklist, Civilian Version; PCL-C1 = intrusive thoughts; PCL-C2 = disturbing dreams; PCL-C3 = reliving the trauma; PCL-C4 = emotional reactivity; PCL-C5 = physical reactivity; PCL-C6 = avoiding thoughts; PCL-C7 = avoiding activities; PCL-C8 = traumatic amnesia; PCL-C9 = loss of interest; PCL-C10 = social disconnection; PCL-C11 = emotional numbness; PCL-C12 = foreshortened future; PCL-C13 = difficulty sleeping; PCL-C14 = irritability; PCL-C15 = difficulty concentrating; PCL-C16 = hypervigilance; PCL-C17 = hyperarousal.

*$p < .05$.

**$p < .01$.

*** $p < .001$.