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Heterogeneity of posttraumatic stress symptomatology and social connectedness in treatment-seeking military veterans: a longitudinal examination

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ABSTRACT

Elucidating whether PTSD symptoms predict poorer social connectedness over time (i.e. social erosion) and/or that poor social connectedness contributes to maintenance of PTSD (i.e. social causation) has implications for PTSD treatment and relapse prevention. Most extant research has been cross-sectional and examined overall PTSD symptoms. Evidence of longitudinal associations among heterogeneous PTSD symptom clusters and social connectedness could provide insight into more nuanced targets for intervention. Using data from 1,491 U.S. military veterans in residential treatment for PTSD at 35 Department of Veterans Affairs facilities, we evaluated a two-wave cross-lagged panel model including a five-factor model of PTSD and two aspects of social connectedness. PTSD, quality of connectedness (i.e. degree of distress related to interpersonal conflict), and structural social support (i.e. number of days of contact with supportive loved ones) in the past 30 days were assessed at baseline and 4 months after discharge. The largest effect was greater severity of PTSD dysphoric arousal symptoms (i.e. irritability/anger, poor concentration, and sleep problems) at baseline predicting more conflict-related distress at follow-up ($\beta = 0.43$). Post-hoc symptom-level analyses indicated that irritability/anger drove this association. In addition, conflict-related distress predicted greater PTSD symptom severity across all five clusters ($\beta$S = 0.10 to 0.14, $p$s < 0.01). More days of contact predicted lower severity of avoidance and numbing symptoms ($\beta$S = −0.05 and −0.07, $p$s < 0.01), along with individual symptoms within these clusters, plus flashbacks. Results support both social erosion and social causation models. Engaging loved ones in veterans’ treatment and targeting dysphoric arousal symptoms, particularly anger and irritability, may improve long-term PTSD and relationship outcomes, respectively.

HIGHLIGHTS

- Sample: 1,491 military veterans in residential treatment for PTSD.
- Examined 5-factor model of PTSD and social connectedness measured twatreatment baseline and 4 months after discharge.
- Baseline dysphoric arousal (anger, sleep problems, poor concentration) predicted distress related to interpersonal conflict; anger drove this association.
- Conflict-related distress at baseline predicted symptoms in all clusters at follow-up.
- More days of contact with supportive loved ones at baseline predicted less avoidance and numbing.

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Posttraumatic stress disorder; social connectedness; social support; dysphoric arousal; veterans; cross-lagged panel analysis

PALABRAS CLAVE

trastorno de estrés postraumático; vinculación social; apoyo social; activación disfórica; veteranos; análisis de panel de retardo cruzado

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Supplemental data for this article can be accessed here.

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Posttraumatic stress disorder (PTSD), a mental health condition that can develop following exposure to trauma, is highly prevalent among military veterans (Fulton et al., 2015; Marmar et al., 2015). PTSD has been increasingly recognized as a disorder that develops, and may be mitigated, in the context of social connections (Bryant, 2016; Wagner, Monson, & Hart, 2016). PTSD is robustly correlated with poor social support (Brewin, Andrews, & Valentine, 2000; Ozer, Best, Lipsey, & Weiss, 2003; Steenkamp et al., 2017), with this association generalizing across veterans with heterogeneous demographic characteristics (Sripada, Lamp, Defever, Venners, & Rauch, 2016) and across sources of support (Wilcox, 2010). Veterans seeking PTSD treatment report poorer social functioning than other treatment-seeking veterans (Tsai, Harpaz-Rotem, Pietrzak, & Southwick, 2012) and frequently identify interpersonal difficulties as treatment targets (Rosen, Adler, & Tiet, 2013).

PTSD and poor social connectedness are theorized to be causally associated via social erosion (i.e. PTSD symptoms eroding social connectedness over time, also termed ‘social selection’) and social causation (i.e. poor social connectedness conferring risk for development and maintenance of PTSD; Kaniasty & Norris, 2008). Studies in military samples that tested both models support the social erosion model (King, Taft, King, Hammond, & Stone, 2006) or both models (Ren, Skinner, Lee, & Kazis, 1999; Shallcross, Arbisi, Polusny, Kramer, & Erbes, 2016; Woodward et al., 2018). Data from non-military samples support social causation (Dworkin, Ullman, Stappenbeck, Brill, & Kayser, 2018), social erosion (Nickerson et al., 2017), or both models (Platt, Lowe, Galea, Norris, & Koenen, 2016).

Inconsistent findings may be due to heterogeneities in study design, such as duration of time since participants’ trauma exposure, prevalence of PTSD in a given sample, and variability in assessment methods (Woodward et al., 2018).

Social connectedness is a multidimensional construct that includes distinct structural, functional, and quality components (Holt-Lunstad, 2018). Aspects of social connectedness relevant to individuals with PTSD include high levels of conflict and difficulties getting along with others, both (poor) quality components, and availability of sources of social support in terms of the presence and availability of existing relationships, a structural component. These aspects of social connectedness are often not highly correlated (Holt-Lunstad, 2018); for example, one may have many high-conflict relationships, or few relationships that are highly supportive. Meta-analytic findings have indicated that PTSD is associated with relationship discord, marital conflict, and perpetration of aggression, with these associations stronger in military than civilian samples (Taft, Watkins, Stafford, Street, & Monson, 2011). Individuals with PTSD also report less structural social support, namely smaller social network size and complexity, as well as lower frequency of contact with loved ones within these networks (King, King, Keane, Foy, & Fairbank, 1999; Schnurr, Lunney, & Sengupta, 2004). We previously found that the majority of veterans starting residential treatment for PTSD reported experiencing at least moderate distress related to conflict with others, as well as less contact with supportive loved ones in the month before starting treatment (Sippel, Watkins, Pietrzak, Hoff, & Harpaz-Rotem, 2018).
Because the relationship between PTSD and social connectedness varies over the course of the disorder (Kaniasty & Norris, 2008), and in order to inform treatment recommendations, it is important to examine longitudinal associations among PTSD symptoms and distinct aspects of social connectedness among veterans who have already developed PTSD. Additionally, data from veterans seeking treatment across different settings, such as residential settings, are needed for generalization of findings. Veterans referred to residential treatment are often more highly symptomatic and may have unique life circumstances (Sripada et al., 2019). To date, only one longitudinal study of PTSD and social connectedness in veterans who had already completed residential treatment for PTSD has been conducted (Laffaye, Cavella, Drescher, & Rosen, 2008). Results supported the social erosion model; over 6 months, more severe PTSD predicted lower perceived support from non-veteran friends. Perceived support did not predict later PTSD symptoms. Extension of this work in a larger, more representative sample of veterans who are entering residential treatment can help to inform the development of prevention and intervention strategies that target and/or utilize social connectedness, a key mechanism of recovery from mental illness (Leamy, Bird, Le Boutillier, Williams, & Slade, 2011).

Specific PTSD symptom clusters and symptoms may be uniquely longitudinally associated with the quality and degree of social connectedness. Emotional numbing symptoms have been associated with relationship distress (Erbes, Meis, Polusny, Compton, & Wadsworth, 2012) and perceptions that relationships do not meet specific needs (Van Voorhees et al., 2012). Hyperarousal symptoms relate to increased likelihood of aggressive behaviour toward intimate partners (Savarese, Suvak, King, & King, 2001; Taft et al., 2007). Shallcross et al. (2016) conducted the only longitudinal study of PTSD symptom clusters and social support in a military sample. Among National Guard soldiers assessed three times over 27 months, symptoms in the dysphoric cluster of the four-factor Dysphoric model (i.e. numbing, irritability/anger, difficulty sleeping, and poor concentration) predicted reductions in perceived support, consistent with the social erosion model. Regarding social causation, lower levels of perceived support predicted increases in re-experiencing, avoidance, and arousal symptoms. Taken together, these results suggest that more general trauma-related distress may erode social connectedness, while poorer social connectedness may lead to or exacerbate more distinctive PTSD symptoms.

The 5-factor Dysphoric Arousal model of DSM-IV PTSD symptoms has shown better fit than 3- and 4-factor models of PTSD (Armour, Carragher, & Elhai, 2013; Armour, Müllerová, & Elhai, 2016; Pietrzak, Tsai, Harpaz-Rotem, Whealin, & Southwick, 2012), including in a sample of over 320,000 veterans (Harpaz-Rotem, Tsai, Pietrzak, & Hoff, 2014). This model differs from the 4-factor Dysphoria model in that it splits dysphoria symptoms into numbing and dysphoric arousal (Elhai et al., 2011). Our previous cross-sectional findings in treatment-seeking veterans with PTSD revealed that more severe numbing and dysphoric arousal symptoms were associated with poorer quality of social connectedness and that numbing symptoms were associated with less contact with loved ones (Sippel et al., 2018). And while hyperarousal symptoms have previously been found to be associated with aggression (Savarese et al., 2001; Taft et al., 2007), a recent study showed that only dysphoric arousal, not anxious arousal, symptoms were associated with aggression (Watkins, Sippel, Pietrzak, Hoff, & Harpaz-Rotem, 2017). An examination of the 5-factor Dysphoric Arousal model of PTSD and relationship satisfaction (a quality component) among motor vehicle accident (MVA) survivors who were assessed 4, 10, and 16 weeks post-MVA revealed that PTSD symptom clusters did not predict relationship satisfaction, nor did relationship satisfaction predict PTSD symptom clusters (LeBlanc et al., 2016). However, analyses of individual symptoms showed that relationship satisfaction predicted decreases in reliving the trauma, emotional numbness, and irritability/anger; no individual PTSD symptoms predicted relationship satisfaction.

Examination of social connectedness with respect to the 5-factor Dysphoric Arousal model of PTSD and individual PTSD symptoms among treatment-seeking veterans may thus reveal novel associations that could add to our understanding of the social context of recovery from PTSD in this population.

The objective of the current study was to use cross-lagged panel analysis to examine longitudinal associations between the 5-factor Dysphoric Arousal model PTSD symptom clusters and two distinct aspects of social connectedness in a large cohort of veterans seeking residential treatment for PTSD using two time-points: treatment baseline and 4 months after discharge. In light of previous research indicating that quality of social support and structural social support are often uncorrelated (Holt-Lunstad, 2018), we expected unique associations between these aspects of social connectedness and PTSD symptom clusters. Hypotheses were largely based on a longitudinal study among National Guard soldiers in which numbing, irritability/anger, difficulty sleeping, and poor concentration predicted reductions in
perceived support (Shallcross et al., 2016). Per the social erosion model, we predicted that greater severity of baseline PTSD dysphoric arousal symptoms would predict greater conflict-related distress at follow-up and that more severe emotional numbing symptoms would predict fewer days of contact with supportive loved ones. Per the social causation model, we expected that greater baseline conflict-related distress would predict more severe re-experiencing, avoidance, and anxious arousal symptoms, the latter corresponding to the 4-factor model arousal symptoms in Shallcross et al. (2016). Per LeBlanc et al.’s study of MVA survivors (2016), we also conducted exploratory follow-up analyses examining longitudinal associations among social connectedness and individual PTSD symptoms.

1. Methods

1.1. Participants and procedures

Participants were 1,491 U.S. military veterans engaged in residential treatment for PTSD across 35 VA facilities between 2012 and 2014. Veterans were asked to complete self-report assessments of demographic variables, psychiatric symptoms including PTSD, substance use, combat status, and other outcomes at the beginning of treatment as part of the VA program evaluation protocol. This study was approved by the local institutional review board. All veterans provided written informed consent. A total of 4,296 veterans entered treatment during this period; 3,789 (88.2%) completed the survey upon entry. Four months after discharge, 2,277 (60.1%) returned a follow-up questionnaire packet. Because veterans who had been administered a PTSD Checklist (PCL; Weathers, Litz, Herman, Huska, & Keane, 1993), a self-report PTSD scale, as part of their treatment in VA within 90 days of intake had their last total PCL score recorded in the assessment instead of their completing a new PCL, symptom-level data that could be used to calculate symptom cluster scores were not available for these veterans \((n = 694; 30.5\%)\). Veterans were included in the study if they completed the follow-up survey and if they had all PTSD symptom-level data available (40.7% of the original sample including veterans with no item-level data).

Veterans who did not return the follow-up questionnaire were younger (42.0 versus 50.0 years; \(t = -17.23, p < .001\)), had shorter treatment length (42.7 days versus 44.8 days; \(t = -2.85, p < .001\)), were less likely to have served in combat \((\chi^2 = 26.76, p < .001)\), and were less likely to have served in combat \((\chi^2 = 6.62, p < .05)\) than veterans who returned the questionnaire. Veterans did not differ with respect to sex, PTSD severity, or social connectedness. Veterans who did not have item-level data (i.e. had completed a PCL within the previous 90 days) were more likely to be female \((\chi^2 = 8.5, p < .01)\) and less likely to have served in combat \((\chi^2 = 14.74, p < .01)\). Veterans with and without item-level PCL data did not differ on age, treatment length, PTSD symptom severity, or social connectedness.

1.2. Assessments

Demographic data were derived from a self-report intake form. Identification of alcohol or drug use disorder (illegal or prescription drug abuse/dependence) needing treatment during the current episode, the types of treatment in which veterans engaged while in residential treatment, and the condition of each veteran’s discharge were reported by the veteran’s provider.

1.2.1. PTSD symptoms

PTSD symptoms were measured at baseline and follow-up with the PCL for DSM-IV (Weathers et al., 1993), which assesses the extent to which veterans were bothered by each symptom over the past month on a 5-point scale ranging from ‘Not at all’ to ‘Extremely.’ Veterans were asked to answer questions in response to events that led to their PTSD. Cronbach’s alphas were 0.89 at baseline and 0.95 at follow-up.

1.2.2. Social connectedness

Social connectedness was assessed at both timepoints using two items from the Brief Addiction Monitor (BAM; Cacciola et al., 2013), each with a 5-point Likert response scale: ‘In the past 30 days, how much have you been bothered by arguments or problems getting along with any family members or friends?’ \((1 = \text{not at all}, 5 = \text{extremely})\) and ‘In the past 30 days, how many days were you in contact or spent time with any family members or friends who are supportive of your recovery?’ \((0, 1–3, 4–8, 9–15, \text{or} 16–30)\).

1.3. Data analysis

We first used confirmatory factor analysis to examine the measurement models for PTSD at each time point. We then used structural equation modelling (SEM) to examine a cross-lagged panel model including PTSD symptom clusters and social connectedness at each timepoint. The cross-lagged panel model included autoregression pathways and pathways from PTSD symptom clusters to social connectedness and vice versa. PTSD symptom clusters and social connectedness variables were allowed to correlate within each time point. All models were estimated with Mplus v. 6 (Muthén and Muthén, 1998–2011). To correct for skewed distributions, maximum likelihood estimation with robust
Lastly, we conducted an exploratory cross-lagged path analysis to examine PTSD symptom-level relationships with social connectedness, adjusting for multiple comparisons with the B-H procedure, false discovery rate set to q* = .05 (Benjamini & Hochberg, 1995). These symptom-level models included all covariates. Results of ML estimation with 16 predictors are stable with sample sizes greater than 400 (Savalei & Bentler, 2005); we therefore expected our item-level path analysis (27 predictors) to be stable in our sample of 1,491 participants.

2. Results

2.1. Descriptive statistics

Descriptive statistics for the final sample are presented in Table 1. Most veterans were white, unmarried, unemployed, and had experienced combat. Mean PCL scores at baseline (67.19) were above the cut-point of 50 for PTSD in military populations, with 94.4% of veterans exceeding the threshold of 50.

2.2. Measurement model

In nearly the same sample, Watkins et al. (2017) confirmatory factor analysis (CFA) of PTSD symptoms indicated that the five-factor Dysphoric Arousal model (Elhai et al., 2011) had better fit (χ² = 952.19, df = 109, CFI = .97, RMSEA = .045) than the three-factor DSM-IV [χ²(7) = 727.47, p < 0.0001], four-factor Emotional Numbing [χ²(4) = 239.08, p < 0.0001; King, Leskin, King, & Weathers, 1998], and four-factor Dysphoria (ΔBIC = −281.06; Simms, Watson, & Doebbell, 2002) models of PTSD symptom structure. We confirmed model fit in the current sample, for which the five-factor Dysphoric Arousal model had acceptable fit at baseline, χ² (109) = 622.01, p < .001, CFI = .93, RMSEA = .055, and good fit at follow-up, χ² (109) = 448.75, p < .001, CFI = .97, RMSEA = .045. See Table 2 for a summary of the fit of the CFA and SEM models.

We also tested measurement invariance of PTSD across the two time points. We found configural errors were applied. We examined model fit with chi-square (χ²), comparative fit index (CFI), and root-mean-square error of approximation (RMSEA). Good fit of a model is generally indicated by CFI values ≥0.95 and RMSEA values ≤0.06 (Hu & Bentler, 1999) and acceptable model fit is indicated by RMSEA values ≤0.08, and CFI values ≥0.90 (Kline, 2011).

Our first SEM model included covariates. We modelled demographic variables that have been previously been associated with PTSD and/or social connectedness (i.e. age, sex, combat status, marital status), SUD (present vs. absent) to account for potential differences in interpretations of the word ‘recovery’ in the BAM item, and treatment site (given sources of variability across the 35 facilities) with PTSD and social connectedness at both timepoints. Length and completion of treatment were modelled only with follow-up variables. The total number of parameters estimated in the covariate model was 286. In a second SEM model, non-significant covariate pathways were removed to yield a more parsimonious model, leaving 222 parameters to be estimated. Our sample size of 1,491 is appropriate for these analyses as it exceeds the estimated samples needed to detect regression coefficients equaling .25-.50 (Wolf, Harrington, Clark, & Miller, 2013), the minimum sample size recommended for robust ML (>400; Savalei & Bentler, 2005), and the 5:1 ratio of cases to parameters needed for unbiased estimates (Bentler & Chou, 1987).

Table 1. Demographic and clinical characteristics.

<table>
<thead>
<tr>
<th>Variable</th>
<th>M (SD) or N (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>49.26 (14.07)</td>
</tr>
<tr>
<td>Male</td>
<td>1453 (94.2%)</td>
</tr>
<tr>
<td>Combat veteran</td>
<td>1284 (83.7%)</td>
</tr>
<tr>
<td>White race</td>
<td>816 (52.9%)</td>
</tr>
<tr>
<td>War era</td>
<td></td>
</tr>
<tr>
<td>Iraq and Afghanistan</td>
<td>638 (41.4%)</td>
</tr>
<tr>
<td>Vietnam</td>
<td>565 (36.6%)</td>
</tr>
<tr>
<td>Persian Gulf</td>
<td>350 (22.7%)</td>
</tr>
<tr>
<td>Between Vietnam and Persian Gulf</td>
<td>265 (17.2%)</td>
</tr>
<tr>
<td>Korean War</td>
<td>4 (0.3%)</td>
</tr>
<tr>
<td>Between Korea and Vietnam</td>
<td>3 (0.2%)</td>
</tr>
<tr>
<td>Married</td>
<td>701 (45.5%)</td>
</tr>
<tr>
<td>Currently unemployed</td>
<td>1321 (85.7%)</td>
</tr>
<tr>
<td>T1 PCL ≥ 50</td>
<td>1456 (94.4%)</td>
</tr>
<tr>
<td>T2 PCL ≥ 50</td>
<td>1188 (77.0%)</td>
</tr>
<tr>
<td>T1 ≤ 15 days of contact with loved ones in past month</td>
<td>905 (58.7%)</td>
</tr>
<tr>
<td>T2 ≤ 15 days of contact with loved ones in past month</td>
<td>822 (53.3%)</td>
</tr>
<tr>
<td>T1 moderate to severe conflict-related distress</td>
<td>975 (63.2%)</td>
</tr>
<tr>
<td>T2 moderate to severe conflict-related distress</td>
<td>879 (57.0%)</td>
</tr>
<tr>
<td>Substance use disorder</td>
<td>714 (46.5%)</td>
</tr>
<tr>
<td>Days of residential treatment delivered</td>
<td>48.50 (20.81)</td>
</tr>
<tr>
<td>Treatments most frequently engaged in Seeking Safety</td>
<td>1258 (81.6%)</td>
</tr>
<tr>
<td>Pharmacotherapy</td>
<td>1236 (80.2%)</td>
</tr>
<tr>
<td>Cognitive behavior therapy</td>
<td>1031 (66.9%)</td>
</tr>
<tr>
<td>Cognitive Processing Therapy</td>
<td>952 (61.7%)</td>
</tr>
<tr>
<td>Treatment completion</td>
<td>1334 (86.5%)</td>
</tr>
</tbody>
</table>

Note. War era categories not mutually exclusive. PCL = PTSD Checklist. T1 = baseline. T2 = 4 months post-discharge.

Lastly, we conducted an exploratory cross-lagged path analysis to examine PTSD symptom-level relationships with social connectedness, adjusting for multiple comparisons with the B-H procedure, false discovery rate set to q* = .05 (Benjamini & Hochberg, 1995). These symptom-level models included all covariates. Results of ML estimation with 16 predictors are stable with sample sizes greater than 400 (Savalei & Bentler, 2005); we therefore expected our item-level path analysis (27 predictors) to be stable in our sample of 1,491 participants.

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Table 2. Assessment of model fit.

<table>
<thead>
<tr>
<th>Model</th>
<th>Number of Parameters</th>
<th>χ²</th>
<th>df</th>
<th>CFI</th>
<th>RMSEA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline CFA</td>
<td>61</td>
<td>622.01</td>
<td>109</td>
<td>.93</td>
<td>.055</td>
</tr>
<tr>
<td>Follow-up CFA</td>
<td>61</td>
<td>448.75</td>
<td>109</td>
<td>.97</td>
<td>.045</td>
</tr>
<tr>
<td>SEM model with covariates</td>
<td>286</td>
<td>2146.56</td>
<td>797</td>
<td>.95</td>
<td>.033</td>
</tr>
<tr>
<td>SEM model with non-significant covariates removed</td>
<td>222</td>
<td>2192.54</td>
<td>823</td>
<td>.95</td>
<td>.033</td>
</tr>
</tbody>
</table>

Note. CFA = confirmatory factor analysis. SEM = structural equation modelling.
invariance \( \chi^2 (465) = 1375.92, \text{CFI} = 0.96, \text{TLI} = 0.96, \text{RMSEA} = .04 \), indicating that the factor model measures the PTSD structure similarly across time points. Based on the Likelihood Ratio Test the metric invariance model fit worse than the configural invariance model, \(-2\Delta LL (12) = 42.81, p < .01\). However, differences in \( \chi^2 \) are dependent on sample size. Based on critical values of change in Goodness of Fit Indexes (\( \Delta \text{GFIs} \)) recommended by Cheung and Rensvold (2002), the null hypothesis of invariance should not be rejected (\( \Delta \text{CFI} = -0.001 \), which is smaller than the recommended cutoff of \(-0.01\)).

### 2.3. Cross-lagged panel SEM model and symptom-level path analyses

The final cross-lagged panel model with non-significant covariates removed fit the data well, \( \chi^2 (823) = 2192.54, p < .001, \text{CFI} = .95, \text{RMSEA} = .033 \). All autoregressive pathways were significant (see Figure 1 note and SI Table 1), indicating that each variable predicted itself at follow-up. All cross-sectional associations were significant (see Table 3), with the exception of associations between number of days of contact and both avoidance and anxious arousal symptoms at baseline.

#### 2.3.1. PTSD symptoms predicting social connectedness

Several cross-lagged relationships between PTSD symptom clusters and social connectedness variables were significant (see Figure 1). Results concerning covariates in the first model are displayed in SI Tables 2 and 3. The removal of non-significant pathways of covariates in the second model did not impact the results. All pathways that were significant in the first full model remained significant in the second model; similarly, all pathways that were not significant in the first model remained non-significant in the second model.

The strongest association was between more severe dysphoric arousal symptoms at baseline and more

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**Table 3. Cross-lagged model cross-sectional associations.**

<table>
<thead>
<tr>
<th>Variable</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
</tr>
</thead>
<tbody>
<tr>
<td>Re-experiencing</td>
<td>-</td>
<td>.30**</td>
<td>.16**</td>
<td>.35**</td>
<td>.29**</td>
<td>.19**</td>
<td>-.05*</td>
</tr>
<tr>
<td>Avoidance</td>
<td>.49**</td>
<td>-</td>
<td>.17**</td>
<td>.28**</td>
<td>.24**</td>
<td>.17**</td>
<td>-.04</td>
</tr>
<tr>
<td>Emotional numbing</td>
<td>.28**</td>
<td>.31**</td>
<td>-</td>
<td>.22**</td>
<td>.17**</td>
<td>.17**</td>
<td>-.08**</td>
</tr>
<tr>
<td>Dysphoric arousal</td>
<td>.39**</td>
<td>.41**</td>
<td>.26**</td>
<td>-</td>
<td>.35**</td>
<td>.37**</td>
<td>-.07**</td>
</tr>
<tr>
<td>Anxious arousal</td>
<td>.49**</td>
<td>.55**</td>
<td>.31**</td>
<td>.46**</td>
<td>-</td>
<td>.19**</td>
<td>-.01</td>
</tr>
<tr>
<td>Conflict-related distress</td>
<td>.34**</td>
<td>.33**</td>
<td>.25**</td>
<td>.38**</td>
<td>.38**</td>
<td>-</td>
<td>-.12**</td>
</tr>
<tr>
<td>Days of contact</td>
<td>-.19**</td>
<td>-.17**</td>
<td>-.14**</td>
<td>-.18**</td>
<td>-.18**</td>
<td>-</td>
<td>-.20**</td>
</tr>
</tbody>
</table>

Cross-sectional associations for baseline data are above the diagonal and associations for follow-up data are below the diagonal. ** \( p < .001 \), * \( p < .05 \)
conflict-related distress at follow-up ($\beta = .43, p < .01$). All other path coefficients were between $\beta = -.15$ and $\beta = .14$. None of the baseline PTSD symptom clusters predicted days of past-month contact at follow-up. Supplemental symptom-level path analysis results (see SI Tables 4 and 5) showed that more severe irritability and angry outbursts (PCL-14) predicted more conflict-related distress at follow-up. Though the re-experiencing and anxious arousal clusters at baseline were associated with less conflict-related distress at follow-up in the five-factor model, the symptom-level analysis revealed that no individual symptoms in these clusters predicted conflict-related distress.

2.3.2. Social connectedness predicting PTSD symptoms

Greater conflict-related distress at baseline was associated with more severe PTSD symptoms within each PTSD cluster at follow-up. Symptom-level path analyses showed that greater conflict-related distress at baseline predicted all PTSD symptoms at follow-up except intrusive memories (PCL-1), amnesia (PCL-8), and sleep difficulties (PCL-13). More days of past-month contact at baseline were associated with lower severity of symptoms in the avoidance and emotional numbing clusters at follow-up, and according to the symptom-level model, less severe flashbacks (PCL-3), behavioral avoidance (PCL-7), loss of interest (PCL-9), estrangement from others (PCL-10), and emotional numbness (PCL-11).

3. Discussion

In a large sample of veterans who participated in residential treatment for PTSD, a cross-lagged panel analysis revealed unique longitudinal associations among PTSD symptom clusters and both quality and structural aspects of social connectedness. Consistent with the social erosion model, which posits that psychological distress leads to a decline in social connectedness over time, the largest effect was that more severe PTSD dysphoric arousal symptoms at baseline predicted poorer quality of social connectedness at follow-up. Post-hoc analyses revealed that more severe irritability and angry outbursts drove this association. Consistent with the social causation model, in which poor social connectedness confers risk for development and maintenance of PTSD, we observed a more general effect of poorer quality of social connectedness at baseline; more distress related to interpersonal conflict predicted more severe symptoms in all PTSD symptom clusters at follow-up. Post-hoc symptom-level path analyses indicated that greater conflict-related distress at baseline predicted all PTSD symptoms at follow-up except intrusive memories, amnesia, and sleep difficulties. Additionally, more days of contact at treatment baseline predicted less severe symptoms in the avoidance and numbing clusters at follow-up. At the symptom level, more contact predicted less severe flashbacks, behavioural avoidance, loss of interest, estrangement, and numbing. These findings build on those of a previous study of the association between PTSD and social support conducted in a small sample of veterans who had completed residential PTSD treatment (Laffaye et al., 2008), and complement results found in active-duty service members (Shallcross et al., 2016), outpatients with PTSD (Price et al., 2018), and MVA survivors (LeBlanc et al., 2016). Overall, this study suggests that multiple forms of social connectedness may help enhance recovery from PTSD, consistent with a broader literature supporting the role of connectedness in recovery from mental illness (Leamy et al., 2011). Additionally, greater severity of PTSD dysphoric arousal symptoms, particularly anger/irritability, may erode quality of connectedness.

The finding that baseline dysphoric arousal symptoms, specifically anger, predicted poorer quality of social connectedness (i.e. more distress related to interpersonal conflict) is in line with previous research showing that PTSD dysphoric symptoms predicted reductions in perceived social support (Shallcross et al., 2016). Findings are also consistent with a previously replicated association between three-factor DSM-IV hyperarousal symptoms and aggressive behaviour (Savage et al., 2001; Taft et al., 2007), which typically occurs in the context of interpersonal conflict. However, findings diverge from those among MVA survivors (LeBlanc et al., 2016), among whom dysphoric arousal symptoms did not predict relationship distress over time, which may have been due to less change in relationships satisfaction compared to PTSD symptoms over the time period of measurement (12 weeks). The negative effects of PTSD symptoms on social connectedness have been observed later in the course of PTSD (Kaniasty & Norris, 2008), and similarly, our sample had chronic PTSD as evidenced by high rates of PTSD symptoms exceeding diagnostic thresholds even after treatment. Additionally, anger has been shown to be more severe among military samples with PTSD than in civilian samples (Orth & Wieland, 2006), suggesting that it may have stronger effects on relationships in this population (Taft et al., 2011).

Our findings parse the dysphoric arousal symptoms from anxious arousal (i.e. hypervigilance and exaggerated startle). Interestingly, though anxious arousal and re-experiencing symptoms were associated with less conflict-related distress at follow-up in our primary five-factor model, our exploratory symptom-level analyses in which only anger/irritability symptoms predicted conflict-related distress
suggest that these unexpected positive associations may have been due to a suppressor effect once variance attributable to other symptom clusters was accounted for (Tzelgov & Henik, 1991).

Consistent with the social causation model and a previous study showing that relationship satisfaction predicted PTSD symptoms across the five factors (LeBlanc et al., 2016), poorer quality of social connectedness operationalized as conflict-related distress at baseline predicted greater severity of all PTSD symptom clusters at follow-up. Additionally, greater structural support (i.e. more contact with supportive loved ones) at baseline was associated with less severe symptoms in the avoidance and numbing clusters (as well as individual symptoms within these clusters – behavioural avoidance, loss of interest, estrangement from others, and emotional numbness, plus flashbacks) at follow-up. Related findings have been observed in depression; for example, loneliness, a perception of and emotional response to social isolation, has been found to predict increases in depressive symptoms, but not vice versa (Cacioppo, Hawkley, & Thisted, 2010). Our findings may differ from those of Laffaye et al. (2008) because of differences in measurement of social connectedness; Laffaye et al. measured ‘interpersonal resources’ with items assessing helpful aspects of relationships. This construct falls under the category of functional connectedness, which is distinct from both structure and quality of social connectedness (Holt-Lunstad, 2018), and may only serve as a consequence rather than contributor to PTSD over time (Laffaye et al., 2008). Taken together, these findings support the premise that the existence of available sources of support with whom there is minimal conflict may be a key contributor to psychological and physical health (Hawkley & Cacioppo, 2010; Holt-Lunstad, 2018).

Results of this study have several clinical implications. Overall, elucidating the longitudinal associations among PTSD symptom clusters and social connectedness may help inform veteran-centred treatment and discharge plans that most effectively target presenting problems and support long-term recovery. For example, improving the quality and frequency of social connection may facilitate veterans’ engagement in PTSD treatment and response to treatment. This premise is supported by previous research showing that family support for treatment increases veterans’ likelihood of staying in treatment (Meis et al., 2019) and that veterans with more social support were less likely to endorse the belief that it is up to them to handle their problems and more likely to seek treatment (Graziano & Elbogen, 2017). Further, family involvement in PTSD care was associated with more PTSD symptom reduction (Laws, Glynn, McCutcheon, Schmitz, & Hoff, 2017), and greater social support predicts better PTSD treatment response (Price, Gros, Strachan, Ruggiero, & Acieno, 2013; Price et al., 2018; Shnaider, Sijercic, Wanklyn, Suvak, & Monson, 2017). Notably, in line with the social causation model, Price et al. (2018) also found that social support moderated PTSD outcome but that PTSD did not moderate increases in social support during treatment. Future studies can assess whether interventions for increasing social connectedness improve treatment engagement and outcome among veterans seeking intensive treatment for PTSD.

Finally, in line with the social erosion model, treatment approaches that target PTSD dysphoric arousal symptoms, particularly anger and irritability, could have downstream effects on the propensity to engage in interpersonal conflict or help veterans manage conflicts more effectively. This premise is supported by evidence showing that cognitive-behavioural anger interventions improve interpersonal relationships among veterans (Shea, Lambert, & Reddy, 2013). Future research examining the effects of evidence-based PTSD treatments and reductions in trauma-related anger and irritability on various aspects of social connectedness, including the quality and frequency of social interactions, is warranted.

Taken together, our work and extant research suggest the potential utility of assessing veterans’ degree and quality of social connectedness before they start residential treatment and, when needed, intervening to increase social support for treatment. Additionally, follow-up assessment of veterans’ social connectedness after residential treatment may help to identify whether veterans’ social environments adequately support sustained recovery from PTSD or put them at risk for relapse.

Limitations include our having only two timepoints with relatively short duration in between, which precludes more fine-grained temporal specificity and causal conclusions regarding interrelationships between social connectedness and PTSD symptom clusters. We did not have information pertaining to pre-trauma or pre-PTSD social functioning, rendering us unable to test the social causation hypothesis that poor social connectedness contributed to the onset of PTSD. We necessarily used only two items assessing social connectedness that were pre-selected for program evaluation purposes. These two items map on to two distinct aspects of social connectedness but it is not clear how closely linked they are to the higher-order construct of social connectedness (Holt-Lunstad, 2018), nor to perceived positive and negative perceived social support, which have distinct effects across the trauma recovery course (Wagner et al., 2016). Namely, promoting
positive social factors after trauma could mitigate trauma’s negative effects (social causation) and not stress social support networks (social erosion/selection). Future studies should assess the frequency and nature of interpersonal conflict, whether contact with loved ones was perceived as supportive or intrusive, and the number of specific sources of support over longer periods of time. Ecological momentary assessment approaches could be useful in this regard. Similarly, PTSD was measured by self-report rather than a structured diagnostic interview; however, the PCL is well validated and very commonly used in clinical settings. These program evaluation data were collected prior to the release of DSM-5 and the corresponding PCL-5. DSM-5 includes two externalizing behaviour symptoms which form a unique factor, with evidence of a more optimal fit of a 7-factor hybrid model compared to DSM-5’s four-factor model. Further work evaluating how this 7-factor model of DSM-5 PTSD symptoms relates to social connectedness, and vice versa, over time is needed (Armour et al., 2015).

It is worth reiterating that we leveraged clinical administrative data collected at treatment baseline and 4 months after discharge from veterans who had engaged in heterogenous treatment plans during their residential programs, leading to our findings having stronger external validity but poorer internal validity than findings from randomized controlled trials. Without a matched control group, we could not directly test the effects of residential treatment for PTSD on social connectedness. Per the social erosion model, residential treatment effectiveness for PTSD symptoms would improve social connectedness over time, though it is possible that the effects would not be observable upon treatment completion but manifest later in time. Unfortunately, the lack of an assessment point at treatment completion meant that we could not examine the precise contributions of pre-post-treatment response to social connectedness. We also lacked detailed information about veterans’ specific treatment doses and modalities (which vary across facilities) that would inform which components of residential treatment may improve social connectedness. The residential treatment setting itself may have influenced results, because in these settings veterans are in frequent contact with veteran peers and treatment providers. Some veterans may have been able to sustain these relationships and generalize their social skills to other relationships after treatment ended. Future studies could compare the magnitude and sustainability of improvements in social connectedness across outpatient and residential treatments. Finally, given the unique nature of our sample of largely male combat veterans seeking residential treatment for PTSD, findings may have limited generalizability to women veterans and individuals seeking outpatient treatment.

4. Conclusion

Results from the current study support both the social causation and erosion models of PTSD and social connectedness, with unique roles of dysphoric arousal, specifically anger and irritability, in predicting social connectedness, broader effects of poor quality of social connectedness on PTSD symptom clusters and individual PTSD symptoms, and effects of contact with supportive loved ones on avoidance and numbing symptoms. Findings suggest the possibility that veterans with better social connectedness when entering treatment have more promising prognoses and that targeting anger and irritability may meaningfully reduce interpersonal conflict. Further research is needed to determine the effects of utilization of social connectedness on the likelihood of developing of PTSD, treatment engagement, treatment outcome, and long-term psychological health.

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