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Treatment Outcomes for Military Veterans With Posttraumatic Stress Disorder: Response Trajectories by Symptom Cluster

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Abstract

Although effective posttraumatic stress disorder (PTSD) treatments are available, outcomes for veterans with PTSD are relatively modest. Previous researchers have identified subgroups of veterans with different response trajectories but have not investigated whether PTSD symptom clusters (based on a four-factor model) have different patterns of response to treatment. The importance of this lies in the potential to increase treatment focus on less responsive symptoms. We investigated treatment outcomes by symptom cluster for 2,685 Australian veterans with PTSD. We used Posttraumatic Stress Disorder Checklist scores obtained at treatment intake, posttreatment, and 3- and 9-month follow-ups to define change

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across symptom clusters. Repeated measures effect sizes indicated that arousal and numbing symptoms exhibited the largest changes between intake and posttreatment, d_{RM} = -0.61 and d_{RM} = -0.52, respectively, whereas avoidance and intrusion symptoms showed more modest reductions, d_{RM} = -0.36 and d_{RM} = -0.30, respectively. However, unlike the other symptom clusters, the intrusions cluster continued to show significant changes between posttreatment and 3-month follow-up, d_{RM} = -0.21. Intrusion and arousal symptoms also showed continued changes between 3- and 9-month follow-up although these effects were very small, d_{RM} = -0.09. Growth curve model analyses produced consistent findings and indicated modest initial changes in intrusion symptoms that continued posttreatment. These findings may reflect the longer time required for emotional processing, relative to behavioral changes in avoidance, numbing, and arousal, during the program; they also reinforce the importance of prioritizing individual trauma-focused therapy directly targeting intrusions as the core component of programmatic treatment.

Treatment Outcomes for Military Veterans With Posttraumatic Stress Disorder: Response Trajectories by Symptom Cluster

Posttraumatic stress disorder (PTSD) is a severe, disabling condition that can develop in the aftermath of trauma. Although effective treatments are available (Forbes et al., 2010), outcomes for military veterans have been consistently found to be more modest than those for other trauma populations (Steenkamp, Litz, Hoge, & Marmar, 2015). Unfortunately, up to two-thirds of veterans retain their PTSD diagnosis after treatment (Bradley, Greene, Russ, Dutra, & Westen, 2005; Steenkamp et al., 2015). Researchers have identified subgroups of veterans with different response trajectories, including responders with high symptom change or low symptom change and nonresponders (Elliott, Biddle, Hawthorne, Forbes, & Creamer, 2005; Phelps et al., 2018). Phelps and colleagues (2018) used conditional latent growth

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mixture modelling to identify five classes of response trajectories, with depression and guilt serving as significant predictors of class membership. Overall, they found treatment gains to be modest, with the largest class showing a 6-point drop on the Posttraumatic Stress Disorder Checklist (PCL; Weathers, Litz, Herman, Huska, & Keane, 1993). A 10-point drop is considered clinically important. However, this research addressed overall PTSD symptom change and the authors did not investigate the multidimensional nature of PTSD and how it relates to recovery. Symptom clusters (intrusions, avoidance/numbing, and arousal) may have heterogeneous trajectory patterns in response to treatment, but when the focus is on overall outcomes, variability in symptom cluster response is not identified. It may be the case that some symptom clusters do respond more strongly to treatment than others, but that these are masked by the symptom clusters that fail to respond.

The importance of considering the symptom clusters of PTSD independently, rather than the severity of PTSD overall, is supported by research in military and veteran populations that has found PTSD symptom clusters to be differentially associated with suicidal ideation, depression, anxiety, aggression, quality of life, social functioning, alcohol misuse, and cannabis misuse (e.g., Bonn-Miller, Boden, Vujanovic, & Drescher, 2013; Hellmuth, Stappenbeck, Hoerster, & Jakupcak, 2012; Jakupcak et al., 2010; Lunney & Schnurr, 2007; Monson et al., 2012). Previous research has investigated how PTSD symptom trajectories vary over time (e.g., Bryant et al., 2015; O'Donnell, Elliott, Lau, & Creamer, 2007) and how the initial severity of particular symptom clusters predicts treatment outcome (Bae, Kim, & Park, 2016; Taylor et al., 2001). However, the comparative trajectories of symptom clusters following treatment has not been examined (Maples-Keller, Price, Rauch, Gerardi, & Rothbaum, 2017). The importance of this lies in increasing our understanding of the specific effects of standard evidence-based treatments and guiding treatment adaptations

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to better target those symptoms that are less responsive to standard treatment (Elhai & Palmieri, 2011).

The aim of this study was to investigate the patterns of change in different PTSD symptom clusters following standard treatment for PTSD in Australian veterans. As the data were collected between 2002 and 2014, our research was based on diagnostic criteria given in the fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM-IV*; APA, 1994), which included three symptom clusters: intrusions, avoidance and numbing, and arousal. To make our research relevant for the fifth edition of the *DSM* (*DSM-5*; APA, 2013), we used the four-factor model of PTSD described by King, Leskin, King, and Weathers (1998), which separates avoidance and numbing into two clusters. King and colleagues' (1998) four-factor model underpins the structure of PTSD in *DSM-5*, which includes four symptom clusters (reexperiencing, active avoidance, negative alterations in cognition and mood, and increased arousal) and has received extensive empirical support (see Palmieri, Weathers, Difede, & King, 2007, for a list of previous studies supporting this model).

Method

Participants

Participants were 2,685 Australian veterans who participated in an accredited PTSD outpatient treatment program funded by the Australian Department of Veterans' Affairs (DVA) between 2002 and 2014. The majority (98.8%) of participants were male. Most had served in the Australian Army (76.2%), with smaller numbers from the Navy (15.9%) and Air Force (7.9%). The most common war-like deployment was to Vietnam (n = 1,760), followed by Malaya (n = 102), the Middle East (n = 85), World War II (n = 61), the Persian Gulf (n = 44), and Korea (n = 25). A number of participants (n = 398) served in peacekeeping missions in Cambodia, Somalia, Rwanda, Timor, and Bougainville. A small

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number of participants (n = 38) did not participate in an overseas deployment. A diagnosis of PTSD was established using the Clinician Administered PTSD Scale (CAPS IV; Blake et al., 1995). Participants were required to have experienced the reported Criterion A event or events during the course of their military service but they were not limited to reporting events that occurred during deployment.

Procedure

Participants were involved in accredited group treatment programs. These programs incorporated 20 to 30 treatment days over a 3-month period; during this time, six to 10 participants per group received a combination of individual and group therapy. The program accreditation guidelines specified the following components of treatment: (a) psychoeducation (the nature of PTSD, rationale for treatment approach), (b) symptom management (eight sessions each on anxiety, anger, and depression), (c) trauma-focused therapy (generally through individual therapy), (d) graded in vivo exposure (individual or group based), (e) substance use issues (four to six sessions), (f) interpersonal skills (six to eight sessions), (g) physical health and lifestyle issues (four to six sessions), (h) relapse prevention, and (i) individual therapy (minimum of eight sessions). Within these parameters, there was variability between programs and across time in the treatment provided. There was also variability in the nature of the trauma-focused therapy provided; it is likely to have included prolonged exposure, cognitive processing therapy, eye movement and desensitization reprocessing (EMDR), and more general trauma-focused cognitive therapy. Unfortunately, specific information about the treatment approach was not collected, and the potential differential impact of these treatment approaches on individual PTSD symptom clusters is acknowledged. Exclusion criteria included being currently psychotic, actively suicidal, having acute substance use disorder, or being currently involved in a major life

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crisis. Participants completed questionnaires (described in detail in the Measures section) at intake, posttreatment, and 3-month and 9-month postdischarge follow-ups as part of the program evaluation process. The Department of Veterans' Affairs Human Research Ethics Committee approved the study.

Measures

PTSD. We measured PTSD severity over time using the PTSD Checklist (PCL; Weathers, Litz, Herman, Huska, & Keane, 1993), a 17-item self-report scale that measures *DSM-IV* PTSD symptoms experienced in the past month (range: 17–85 Cronbach's αs = .94–.97). Participants were asked to respond to items on the PCL in relation to their most traumatic military experience. The 17 items were separated into four symptom clusters based on King and colleagues' (1998) four-factor model. We measured intrusions with mean scores for Items 1 to 5, avoidance with mean scores for Items 6 and 7, numbing with mean scores for Items 8 to 12, and arousal with mean scores for Items 13 to 17. Confirmatory factor analysis (CFA) models indicated that this four-factor model provided adequate fit to the data, $\chi^2(113, N = 2{,}510) = 1169.04$, $p < .001$, comparative fit index (CFI) = .93, root mean square error of approximation (RMSEA) = .06, standardized root mean square residual (SRMR) = .04, which was comparable to alternative four-factor models. For example, Simms, Watson, and Doebbelling (2002) grouped items reflecting intrusion (Items 1 to 5), avoidance (Items 6 and 7), dysphoria (Items 8 to 15), and hyperarousal (Items 16 and 17) symptoms, and this provided similar fit to the current data, $\chi^2(113, N = 2{,}510) = 1113.62$, $p < .001$, CFI = .93, RMSEA = .06, SRMR = .04. We retained the King et al. (1998) model because it was closest to the *DSM-5* conceptualization of PTSD. The internal reliability for all clusters at intake was above Cronbach's α = .70 (intrusion, Cronbach's α = .88; numbing, Cronbach's α = .73; arousal, Cronbach's α =.76; and avoidance, Cronbach's α =.80).

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Demographic information. We collected demographic data, including age at intake, pension status, compensation-seeking status, and employment status, from veterans. This information was included in the questionnaires collected at intake.

Alcohol misuse. We assessed alcohol misuse with the Alcohol Use Disorders Identification Test (AUDIT; Babor, de la Fuente, Saunders, & Grant, 1989). A cutoff score of 8 on the AUDIT was used to indicate a clinically significant disorder (Creamer, Elliot, Forbes, Biddle, & Hawthorne, 2006).

Anxiety and depression. We used the Hospital Anxiety and Depression Scale (HADS; Zigmond & Snaith, 1983) to assess anxiety and depression. Cutoff scores of 11 on both the Depression and Anxiety subscales of the HADS were used to indicate clinically significant disorder (Creamer et al., 2006). Cronbach's alpha values were .77 and .83 for the Depression and Anxiety subscales, respectively, at treatment intake.

Data Analyses

We used SPSS (Version 23) for data preparation and conducted substantive analyses in Program R (Version 3.4.3) and MPlus (Version 7.4). Descriptive analyses were conducted to summarize the pattern and extent of missing data, and we estimated logistic regression models (specifying a binary indicator of missing data at posttreatment and 9-month follow-up as the endogenous variables) to indicate whether "missingness" was associated with key baseline variables. Further descriptive analyses comprised means and standard deviations, which were produced for the symptom cluster total scores across assessments. We used repeated measures effect size estimates (d_{RM}) with 95% confidence intervals (CIs) to quantify the magnitude of change in each symptom cluster over time. The latter were based on formulas for the single-group pretest–posttest design, which standardizes the sample mean

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change by variability in change scores (Morris & DeShon, 2002) and were produced in Program R using the Package "effsize" (Torchiano, 2016).

We then estimated a series of growth curve models (GCMs) to provide additional summaries of the patterns of change in PTSD symptom clusters over time. A GCM takes the form of a structural equation model (SEM), which can specify a latent variable to represent the intercept (i.e., intake score) and slope (i.e., change in score over time) of an outcome measure. In this study, the intercept and slope for each symptom cluster were derived from participants' scores at the four time points (intake, posttreatment, 3-month follow-up, and 9-month follow-up), with time thus specified initially in linear models as 0, 1, 2, and 4. Alternative models for change in each symptom cluster were then specified in subsequent models using fixed nonlinear time scores to represent square root (0.00, 1.00, 1.41, 2.00) and fourth root (0.00, 1.00, 1.19, 1.41) models of change. The specification of the nonlinear functions were based on inspection of the means for each cluster. All showed a relatively rapid change in the first half of the 12 months from intake to 9-month follow-up. Where a linear shape exists, the value for time is raised to a power of 1.00 (no change), and this function serves as the comparator. Where there is more change in the first half, the value of time is raised to a power less than 1.00 in order to match the function to the shape of the observed data. In theory, there is an infinite number of possible functions that could be tested. We chose to raise the value of time to 0.50 (square root) and 0.25 (fourth root), as these have been successfully used in previous research (Elliot et al., 2005; Murphy et al., 2008). Model interpretation was facilitated by comparing intercepts (predicted score at intake) and slopes (predicted change over time) and by observing trajectories presented graphically.

We based selection of the preferred model for each symptom cluster on a range of statistical criteria, including approximate and comparative fit indices. Approximate fit indices

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included the CFI (Bentler, 1990), the Tucker-Lewis Index of fit (TLI), and the RMSEA (Steiger, 1989) with 90% CI. The CFI and TLI range from 0 to 1, with values above .90 indicating acceptable model fit and values above .95 indicating good model fit (Hu & Bentler, 1999). A RMSEA value less than .06 supports the model as fitting the data well (Hu & Bentler, 1998). Comparative fit indices included the Akaike's Information Criterion (AIC; Akaike, 1973) and the Bayesian Information Criterion (BIC; Schwarz, 1978) as well as the sample size–adjusted BIC (SS-BIC; Sclove, 1987). These indices facilitate comparisons across nonnested models and those for which smaller values indicate superior model fit. All GCM analyses were conducted in Mplus (Muthén & Muthén, 2013) using robust maximum likelihood (MLR) estimation and full information maximum likelihood (FIML) estimation to utilize all available data. Although intraclass correlation coefficients (ICCs) indicated clustering of data attributed to patients nested within treatment services (ICCs ranged from 0.007 to 0.030, with a mean of 0.013), this clustering could not be accommodated in analyses given the small number of such services (which would comprise the Level 2 units in a multilevel or hierarchical framework), $k = 14$. We used standard formulas to estimate the "design effect" (Kish, 1965) associated with clustering, given (a) the highest ICC and (b) an average cluster size of 184 participants, and these suggested an effective sample size in this study of 414 participants.

Results

Missing Data Analysis

We observed missing data across all PCL items for 412 patients at posttreatment, 585 patients at 3-month follow-up, and 761 patients at 9-month follow-up. Importantly, these figures represent noncompliance with data collection rather than treatment dropout. Thus, there were data available from 2,273 patients at posttreatment, 2,100 patients at 3-month

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follow-up, and 1,924 patients at 9-month follow-up. Exploration of data indicated various patterns of missing data, which included 1,519 patients with complete data, 312 patients with missing data at 9-month follow-up only, 193 patients with missing data at both 3-month and 9-month follow-ups, and several other patterns of intermittent data (e.g., 143 participants had missing data at 3-month follow-up only). We conducted a series of logistic regression analyses to examine the associations between missing data (at posttreatment and 9-month follow-up) and baseline measures including age, relationship status, employment, PTSD symptoms, and number of comorbidities at intake. These indicated that being single was significantly associated with increased odds of missing data at posttreatment, odds ratio (*OR*) = 1.6, but not at 9-month follow-up, *OR* = 1.13, whereas greater numbers of comorbid conditions were associated with decreased odds of missing data at posttreatment, *OR* = 0.71, but not at 9-month follow-up, *OR* = 1.02. Age was associated with decreased odds of missing data at both posttreatment and 9-month follow-up, but the effect sizes were small, *OR* = 0.98 and *OR* = 0.96, respectively, whereas there were no significant associations between baseline PCL scores or employment and odds of missing data at posttreatment. There were small numbers of cases ($n < 10$) with item-level missing data at each time point.

Descriptive Analyses

Participant demographics are presented in Table 1. Scores on self-report measures of alcohol use, anxiety, and depression (AUDIT and HADS) indicated rates of clinically significant disorder of 36.9% for alcohol misuse, 44.4% for depression, and 23.6% for anxiety among participants. Table 2 provides descriptive statistics for total scores across the intrusion, avoidance, numbing, and arousal items of the PCL at intake, discharge, 3-month and 9-month assessments. Table 2 presents means and standard deviations for each symptom cluster across time as well as the repeated–measures effect size estimates (d_{RM}), which

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quantify the magnitude of within-group change for each symptom cluster across adjacent assessments. As shown, scores for all symptom clusters indicated significant reductions across the course of treatment (95% CI values did not include zero), with the largest change observed for the arousal and numbing clusters. Notwithstanding relatively modest changes in intrusion symptoms from intake to posttreatment, this cluster also evidenced significant reductions from posttreatment to 3-month follow-up as well as small changes from 3-month to 9-month follow-up. In contrast, there were no indications of significant change in the other symptom clusters from posttreatment to 3-month follow-up although arousal symptoms also showed small reductions from 3-month to 9-month follow-ups.

Growth Curve Modelling

Table 3 provides the model fit indices for a series of GCMs, which provide further evaluations of the pattern of change in different symptom clusters. These models used mean scores, rather than total scores, across items to summarize standings on symptom clusters, which allowed for more interpretable comparisons across clusters in regards to change over time. As shown, there was a clear preference for fourth root models in describing change in the avoidance, numbing, and arousal clusters; compared with linear and square root models, the fourth root model produced higher values for the CFI and TLI along with lower values for the AIC, BIC, SS-BIC, and RMSEA. In contrast, the results for the intrusion cluster indicated that the square root model provided the best fit to the data as indicated by the excellent approximate fit indices (CFI, TLI, and RMSEA) and the comparative fit indices (AIC, BIC, SS-BIC), suggesting superiority over the fourth root model. These findings, depicted graphically in Figure 1, suggest that the trajectory for the intrusion cluster varies relative to the other clusters. Rather than a sharp fall in symptom level from intake to discharge

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followed by relatively minor change thereafter, the intrusions cluster exhibited a more gradual and sustained pattern of change over time.

Discussion

The results of this study indicated that all PTSD symptom clusters displayed significant reductions over the course of treatment within a large cohort of veterans undergoing psychological treatment, and that treatment gains were maintained, if not extended, across the 9-month follow-up. Although it was not the key focus of this paper, it should be noted that the clinical importance of changes was modest, with effect size changes from intake to 9-month follow-up in the small-to-moderate range. There was evidence of variability in the magnitude and timing of reductions across symptom clusters. For example, repeated–measures effect sizes indicated that arousal and numbing symptoms exhibited the largest changes between intake and posttreatment whereas avoidance and intrusion symptoms were subject to more modest (although still statistically significant) reductions. However, the intrusions symptom cluster continued to show significant changes between posttreatment and 3-month follow-up whereas there were no such reductions for the avoidance, numbing, and arousal clusters. Intrusion and arousal symptoms also indicated continued changes between 3-month and 9-month follow-ups although these effects were very small in magnitude. The GCM analyses produced consistent findings, which indicated that a square root model provided the best account of change in intrusion symptoms, relative to the fourth root models, which provided best fit to data from avoidance, numbing, and arousal. These models also support the findings of the repeated-measures analyses. That is, for the avoidance, numbing, and arousal clusters, rapid improvement was followed by relatively little change. In contrast, for the intrusions cluster, the initial change in symptom level was more modest but continued across the 12-month period, albeit at a reducing rate. Unlike many PTSD symptoms that are

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shared with common comorbid disorders such as depression and anxiety, intrusive symptoms (nightmares and dissociative flashbacks in particular) are unique to the PTSD. According to psychological models of PTSD (Foa & Rothbaum, 1989, 1998), intrusions reflect unprocessed memory of trauma and are at the core of the disorder, driving a cycle between increased arousal and avoidance that perpetuates the PTSD. That is, when the memory of trauma is reexperienced in daytime intrusions or triggered by reminders, this leads to an escalation in arousal and the tendency to push away reminders that is reflected in both active avoidance and negative mood and cognition. Thus, addressing intrusions is the cornerstone of many evidence-based treatments for PTSD. Whereas trauma-focused treatment (including prolonged exposure, cognitive processing therapy, EMDR, and other cognitive therapy) is provided in the accredited group treatment programs, this occurs alongside a range of treatment elements that directly target behavioral elements of avoidance, numbing, and arousal. By virtue of their participation in these intensive group-based trauma treatment programs, veterans are reducing their avoidance of trauma reminders. Posttreatment avoidance cluster ratings are likely to reflect this. Similarly, through participation in the program, ratings on numbing symptoms, such as feelings of detachment or estrangement from others, diminished interest or participation in activities, and sense of foreshortened future, are likely to be impacted by making new connections within the group, engaging in significant activities, and actively planning for the future, respectively. With respect to arousal, the immediate impact of learning anxiety reduction techniques is likely to influence posttreatment ratings on some of the symptoms. Thus, behavioral changes as a function of program participation may bring about early changes in the ratings on avoidance, numbing and arousal symptom clusters. These changes may be important for subsequent reductions in intrusions for two reasons. First, to the extent that these symptoms contribute to the

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maintenance of PTSD through preventing habituation and the emotional processing of trauma, a reduction in their severity allows for more sustained exposure to trauma memories and triggers in the real world, bringing about the gradual reduction in intrusions observed through to the 9-month follow-up point. Second, changes in avoidance and hyperarousal symptoms have been found to predict changes in the "quality of life" domain of achievement, comprising health, self-esteem, goals and values, money, and work (Lunney & Schnurr, 2007). Such quality of life changes may contribute to greater capacity and preparedness to confront the intrusive symptoms of PTSD. This finding therefore highlights the importance of directly targeting the avoidance, numbing, and arousal symptoms as part of a comprehensive approach to the treatment of PTSD. Our observation that some of the improvement in the intrusion cluster occurred postdischarge raises the possibility that intrusive symptoms may change only after improvements have been made in the other symptom clusters. There may be value in examining this possibility through casual modelling in further studies.

We acknowledge the limitations in the data used for this study. First, the data were collected as part of routine program participation (with no control condition), and although the GCMs used all available data, it was not possible to account for missing data from noncompleters. Second, there was evidence of clustering associated with the hierarchical data structure (patients nested within treatment services), which could not be readily accommodated in analyses given the small number of Level 2 units (k = 14). Further, standard errors were downwardly biased, suggesting increased rates of Type I error. Third, although the Australian PTSD program standards specify the components of individual and group treatment, information was not available about which specific treatment modalities were employed (e.g., prolonged exposure vs. cognitive restructuring therapy). This information would have been valuable in the interpretation of results, and the examination of

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treatment outcomes by specific treatment modality is an important direction for future research. Furthermore, treatment integrity was not independently assessed by fidelity investigations, so some level of heterogeneity in program content and delivery must be acknowledged. Fourth, there was no measure of change at midtreatment or during treatment. Fifth, the average age of participants was 55.92 years and 64.6% of veterans in the sample had a comorbid diagnosis; thus, our participants represented a chronic and complex cohort of veterans with PTSD, limiting the generalizability of findings to PTSD sufferers more broadly. Finally, the nature of this multicomponent, group-based treatment limited the generalizability of findings to alternative individual and group-based treatments.

This study investigated treatment outcomes for veterans with PTSD by symptom cluster (intrusions, avoidance, numbing, and arousal). All symptom clusters showed improvement from intake through to follow-up, but the intrusion cluster showed less initial, though more sustained, improvement relative to the other three clusters. In addition, the improvement in intrusions took longer than improvement in the other symptom clusters, with some change occurring between posttreatment and 9-month follow-up. This may have been related to the longer time required for emotional processing of traumatic experience compared with potential short-term behavioral changes in avoidance, numbing, and arousal symptoms. The findings of the study reinforce the importance of prioritizing individual trauma-focused therapy as the core component of trauma recovery programs to ensure adequate treatment of intrusive symptoms.

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Table 1

Participant Demographics

Variable	*Mean*	*SD*	%
Age (at intake)	55.92	10.54	
Number of co-occurring psychiatric disorders	1.04[a]	0.97	
Any co-occurring psychiatric disorder			64.6
Currently applying for DVA pension			42.7
Currently applying for increase in DVA pension			47.3
Employment status			
Full-time			10.2
Part-time			3.0
Retired			21.7
Unemployed			5.8
Unable to work			57.3

Note. DVA = Department of Veterans Affairs. [a]Range: 0–8.

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Table 2 Descriptive Statistics for Posttraumatic Stress Disorder (PTSD) Symptom Clusters and Repeated-Measures Effect Size Estimates (d_{RM}) Indicating the Magnitude of Change Over Time

			d_{RM}[a]	
Variable	*M*	*SD*	*d*	95% CI
Intrusion				
Intake	16.45	4.36		
Posttreatment	15.33	4.52		
3-month follow-up	14.58	4.49		
9-month follow-up	14.22	4.39		
Intake to posttreatment			-0.30*	[-0.36, -0.24]
Posttreatment to 3-month follow-up			-0.21	[-0.27, -0.15]
3-month to 9-month follow-up			-0.09	[-0.15, -0.02]
Avoidance				
Intake	7.27	2.04		
Posttreatment	6.47	2.12		
3-month follow-up	6.39	2.14		
9-month follow-up	6.29	2.18		
Intake to posttreatment			-0.36*	[-0.42, -0.30]
Posttreatment to 3-month follow-up			-0.05	[-0.11, 0.01]
3-month to 9-month follow-up			-0.04	[-0.10, 0.03]
Numbing				
Intake	18.14	3.93		
Posttreatment	16.04	4.40		

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3-month follow-up	16.01	4.36		
9-month follow-up	15.70	4.43		
Intake to posttreatment			-0.52*	[-0.58, -0.46]
Posttreatment to 3-month follow-up			-0.02	[-0.09, 0.04]
3-month to 9-month follow-up			-0.07	[-0.13, 0.00]
Arousal				
Intake	19.52	3.51		
Posttreatment	17.31	4.17		
3-month follow-up	17.30	4.12		
9-month follow-up	16.95	4.20		
Intake to posttreatment			-0.61*	[-0.67, -0.55]
Posttreatment to 3-month follow-up			-0.01	[-0.08, 0.05]
3-month to 9-month follow-up			-0.09*	[-0.16, -0.02]

Note. d_{RM} = repeated measures effect size estimate, standardized by variability in change scores. [a]95% CIs for d_{RM} that do not include zero indicate effects that are significant at conventional levels.

*$p < .05$.

Table 3 Model Fit Statistics From Growth Curve Models (GCMs) for Posttraumatic Stress Disorder Symptom Clusters

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Model	χ^2	*df*	CFI	TLI	RMSEA		AIC	BIC	SS-BIC
					Estimate	90% CI			
Intrusion									
Linear	175.61	5	0.94	0.93	0.11	[0.10, 0.13]	19113.4	19166.4	19137.8
Square root	44.41	5	0.99	0.98	0.05	[0.04, 0.07]	18977.6	19030.7	19002.1
Fourth root	54.35	5	0.98	0.98	0.06	[0.05, 0.08]	18987.4	19040.5	19011.9
Avoidance[a]									
Linear	209.75	6	0.89	0.89	0.11	[0.10, 0.13]	23888.5	23935.7	23910.3
Square root	64.96	6	0.97	0.97	0.06	[0.05, 0.07]	23735.1	23782.3	23756.9
Fourth root	11.62	6	1.00	1.00	0.02	[0.00, 0.04]	23678.4	23725.6	23700.1
Numbing									

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	479.2						19413.	19466.	19437
Linear	5	5	0.82	0.78	0.19	[0.17, 0.20]	0	0	.4
Square	177.4						19100.	19153.	19125
root	6	5	0.93	0.92	0.11	[0.10, 0.13]	8	8	.2
Fourth							18962.	19015.	18986
root	39.83	5	0.99	0.98	0.05	[0.04, 0.07]	0	0	.4
Arousal									
	613.7						18203.	18256.	18227
Linear	1	5	0.75	0.69	0.21	[0.20 , 0.23]	5	5	.9
Square	225.8						17785.	17838.	17810
root	7	5	0.91	0.89	0.13	[0.11, 0.14]	6	7	.1
Fourth							17598.	17651.	17622
root	49.41	5	0.98	0.98	0.06	[0.04, 0.07]	0	1	.5

Note. [a]For all models of avoidance symptoms, it was necessary to constrain the covariance between intercept and slope to zero in order to achieve convergence. CFI = Confirmatory Fit Index; TLI = Tucker-Lewis Index; RMSEA = Root Mean Square Error of Approximation; AIC = Akaike Information Criterion; BIC = Bayesian Information Criterion; SS-BIC = Sample Size Adjusted BIC.

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Table 4 Parameter Estimates from Best-Fitting Growth Curve Models (GCMs) for Posttraumatic Stress Disorder Symptom Clusters

	Intrusion			Avoidance			Numbing			Arousal		
Symptom Cluster	Estimate	*SE*	*p*	Estimate	*SE*	*p*	Estimate	*SE*	*p*	Estimate	*SE*	*p*
Intercept												
Mean	3.28	0.02	.000	3.62	0.02	.000	3.62	0.02	.000	3.90	0.01	.000
Variance	0.52	0.03	.000	0.52	0.02	.000	0.42	0.04	.000	0.34	0.03	.000
Slope[a]												
Mean	-0.22	0.01	.000	-0.35	0.02	.000	-0.35	0.01	.000	-0.38	0.01	.000
Variance	0.06	0.01	.000	0.07	0.01	.000	0.13	0.03	.000	0.13	0.02	.000
Intercept WITH Slope	-0.03	0.01	.013	0.00[b]	-	-	-0.04	0.03	.190	-0.03	0.03	.278

Note. [a]The latent slope represents a parameter from a square root model for intrusion symptoms, and from a fourth root model for avoidance, numbing, and arousal symptoms. [b]Parameter was constrained to zero to achieve model convergence.

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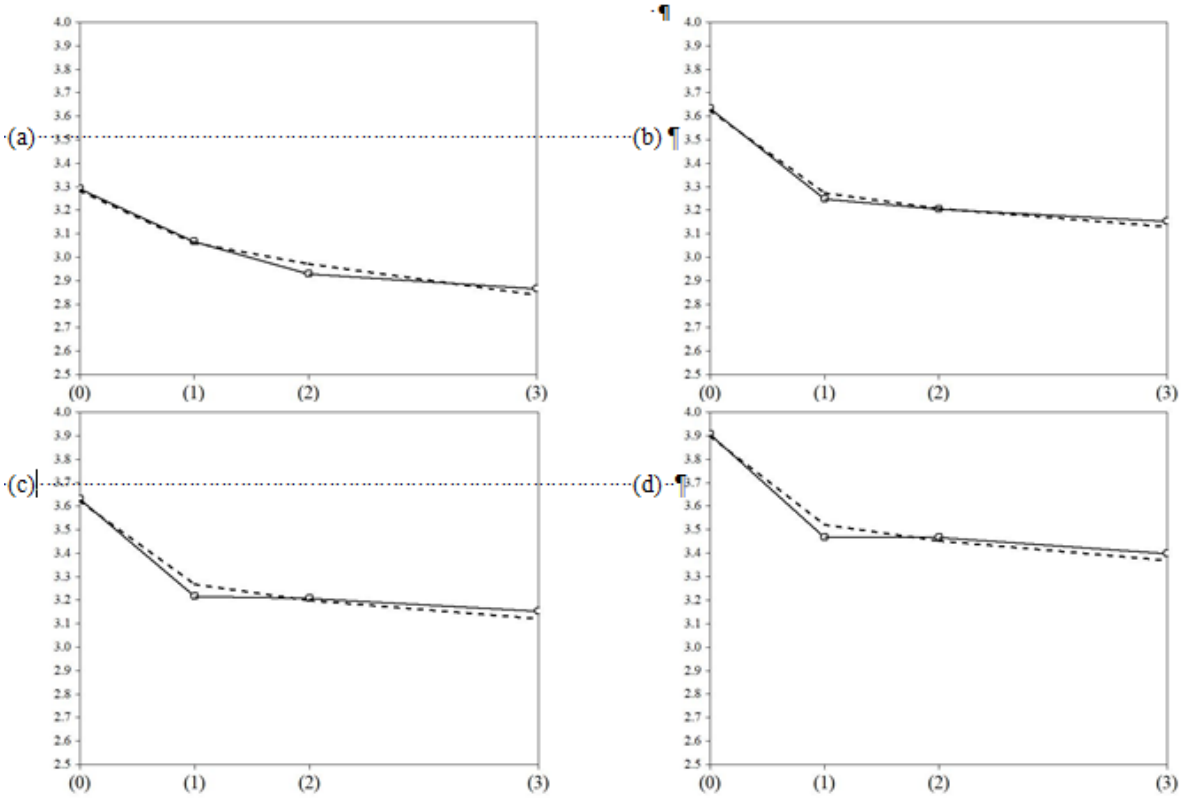

Figure 1. Observed means (solid line) and estimated means (dashed line) from GCMs for (a) intrusion, (b) avoidance, (c) numbing, and (d) arousal symptoms, across (0) intake, (1) posttreatment, (2) 3-month follow-up, and (3) 9-month follow-up.

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