Background: The objectives of the current study were to investigate 1) the longitudinal, reciprocal associations between pain and posttraumatic stress symptoms as proposed by the mutual maintenance model, and 2) to assess the predictive value of the three clusters of posttraumatic stress, where the model revealed that posttraumatic stress symptoms maintained pain in a consecutive cohort of whiplash-injured.

Methods: Participants (n=253; 66.4% women) were people with WAD grade I-III following motor-vehicle crashes in Australia. Pain and posttraumatic stress symptoms were assessed by questionnaires over the course of a year (at baseline (<4 weeks), 3, 6, and 12 months post-injury). The objectives were tested using auto-regressive cross-lagged modelling and two additional structural equation models.

Results: The analyses revealed that posttraumatic stress symptoms at baseline predicted an increase in pain between baseline and 3 months, and that posttraumatic stress symptoms at 6 months predicted an increase in pain between 6 months and 12 months, beyond the stability of pain over time. Furthermore, hyperarousal at baseline significantly predicted pain at 3 months and hyperarousal at 6 months significantly predicted pain at 12 months with 16 and 30% explained variance, respectively.

Conclusions: The results point to a temporal main effect of posttraumatic stress symptoms on pain over and above the stability of pain itself within the first 3 months post-injury and again in the chronic phase from 6 to 12 months with hyperarousal symptoms driving these effects. From 3 to 6 months, there was a slip in the maintenance patterns with no cross-lagged effects.

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Reciprocal associations of pain and posttraumatic stress symptoms after whiplash-injury: A longitudinal, cross-lagged study

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Significance: Investigating mutual maintenance of pain and PTSS in whiplash, the present study found evidence suggesting a maintaining effect of PTSS on pain the first 3 months post-injury and from 6 to 12 months driven by hyperarousal, highlighting the importance of addressing PTSS.

Abstract

Background: The objectives of the current study were to investigate 1) the longitudinal, reciprocal associations between pain and posttraumatic stress symptoms as proposed by the mutual maintenance model, and 2) to assess the predictive value of the three clusters of posttraumatic stress, where the model revealed that posttraumatic stress symptoms maintained pain in a consecutive cohort of whiplash-injured.

Methods: Participants (n=253; 66.4% women) were people with WAD grade I-III following motor-vehicle crashes in Australia. Pain and posttraumatic stress symptoms were assessed by questionnaires over the course of a year (at baseline (<4 weeks), 3, 6, and 12 months post-injury). The objectives were tested using auto-regressive cross-lagged modelling and two additional structural equation models.

Results: The analyses revealed that posttraumatic stress symptoms at baseline predicted an increase in pain between baseline and 3 months, and that posttraumatic stress symptoms at 6 months predicted an increase in pain between 6 months and 12 months, beyond the stability of pain over time. Furthermore, hyperarousal at baseline significantly predicted pain at 3 months and hyperarousal at 6 months significantly predicted pain at 12 months with 16 and 30% explained variance, respectively.

Conclusions: The results point to a temporal main effect of posttraumatic stress symptoms on pain over and above the stability of pain itself within the first 3 months post-injury and again in the chronic phase from 6 to 12 months with hyperarousal symptoms driving these effects. From 3 to 6 months, there was a slip in the maintenance patterns with no cross-lagged effects.

Keywords: Pain, whiplash-associated disorders, WAD, posttraumatic stress symptoms, PTSS

Introduction

This study investigates the longitudinal, reciprocal associations between pain and posttraumatic stress symptoms (PTSS) following whiplash-injuries. Associations between pain and PTSS have received growing attention in recent years (Moeller-Bertram et al., 2012; Otis et al., 2003). According to DSM-IV, posttraumatic stress disorder (PTSD) encompasses symptoms of intrusion, hyperarousal, and avoidance after a trauma (APA, 2000) with PTSS representing symptoms on the same clusters, but not necessarily compliant with a diagnosis. One of the most well-known models on pain and PTSS proposes mutual maintenance (Sharp and Harvey, 2001), and recent reviews have stated that there is indeed evidence of mutual influence (Beck and Clapp, 2011) and reciprocal interdependence (Brennstuhl et al., 2015). However, only few studies have used statistics enabling examinations of reciprocal, longitudinal relations and here, findings are mixed. All studies reported mutual maintenance of pain and PTSS within the initial months post-injury, respectively in a severe injury-sample (<3 months; Carty et al., 2011), a mixed accident-sample (<6 months; Jenewein et al., 2009), and a military-sample (<6 months; Stratton et al., 2014). However, when looking at the chronic period (>3 and 6 months), one study reported that only pain maintained PTSS (Carty et al., 2011), while the opposite was the case for the other two (Jenewein et al., 2009; Stratton et al., 2014). Hence, there is evidence of mutual maintenance in the initial months post-accident, while relational patterns may change to unidirectional maintenance in the more chronic months with mixed findings on the direction of this. Further, two studies reported mutual maintenance between hyperarousal and pain and between intrusion and pain among motor-vehicle-crash-survivors and injury-patients, respectively (Feinberg et al., 2017; Liedl et al., 2010), indicating that maintenance of PTSS on pain might be driven by hyperarousal and/or intrusion. Besides mixed findings, the heterogeneity between samples and variations in time points make it difficult to draw more definite conclusions on relational patterns between pain and PTSS within specific musculoskeletal pain populations such as whiplash-associated disorders (WAD).

The traumatic onset of WAD makes it highly relevant to assess the maintenance patterns of pain and PTSS. Indeed, PTSS are common in WAD and are associated with greater pain and disability (Andersen et al., 2011; Dunne-Proctor et al., 2016; Stålnacke, 2009). In addition, high levels of initial stress symptoms are predictive of poor functional recovery (Buitenhuis et al., 2006; Kongsted et al., 2008; Walton et al., 2013), and PTSS and pain-related disability follow parallel recovery-courses, indicating a common link over time (Sterling et al., 2011). These findings suggest that PTSS and pain

in WAD are entangled over time, but, to our knowledge, no study has yet assessed the potential reciprocal, longitudinal relations.

The aim the current study was to investigate the overall premise of the mutual maintenance model (Sharp and Harvey, 2001) with an investigation of the longitudinal, reciprocal associations between pain and PTSS among a representative cohort of whiplash-injured (objective 1) and further to assess the predictive value of the symptom-clusters of PTSS, where the model revealed that PTSS maintained pain (objective 2).

Methods

Participants and procedure

Participants were individuals with acute WAD (grades I, II, or III) following a motor vehicle crash (MVC) in Australia. Participants were recruited from hospital emergency rooms, primary care practices, and advertisements in newspapers in Brisbane and Sydney. All participants attended a baseline assessment within the first 4 weeks after their MVC's. Inclusion criteria were neck pain after a MVC, age between 18 and 65 years, and fluency in spoken and written English. Exclusion criteria were WAD grade 4 (fracture or dislocation), head injuries, a serious medical condition, or/and prior WAD that required treatment. Participants were assessed at baseline within 4 weeks of injury and at 3, 6, and 12 months post-injury. All assessments were carried out by a researcher, who was also a registered physiotherapist. For the present study, data from two earlier studies (Pedler et al., 2016; Sterling et al., 2010) were merged (n=253). The same criteria, scales, and design were used. All participants volunteered freely, and their written consent to participate in the research project was given before baseline measurements.

Measures

All data were based on self-report questionnaires.

Pain intensity was assessed with a visual analogue scale (VAS; Hawker et al., 2011; Woodforde & Merskey, 1972) asking for average pain within the last 24 hours with a higher score indicating more pain. Earlier studies have found VAS to be reliable to assess both acute and chronic pain (e.g., Bijur, Silver, & Gallagher, 2001; Ferraz et al., 1990; Price et al., 1983).

PTSS related to the MVC were assessed using the Posttraumatic Diagnostic Scale (PDS) for DSM-IV (Foa et al., 1997). The PDS is a reliable self-report measurement with 49 items where 17

assess the core PTSD-symptoms on the 3 clusters of intrusion, avoidance, and hyperarousal. Answers are given on a 4-point Likert scale with as higher score indicating more symptom severity. The original validation study demonstrated high internal consistency along with acceptable test-retest reliability, a high diagnostic agreement with a diagnostic interview, and good sensitivity and specificity (Foa et al., 1997). Recently, high levels of internal consistency and reliability were also found among Swedish chronic pain patients (Åkerblom et al., 2017).

Statistical analyses

Prior to testing the hypotheses, all data were screened for errors and missing values. Overall, 0.4 to 18.6% of data were missing across waves. To decide whether the data had missing values in a random pattern, we conducted the Little's Missing Completely at Random (MCAR; Collins et al., 2001) test. The analysis revealed that the data were missing at random (MAR), $\chi^2(332) = 351.72$, $p = 0.219$. Skewness and kurtosis values indicated asymmetric distributions (Table 1). Moreover, to examine the multivariate normality of measures, Mardia's multivariate normality tests were also conducted. The result showed that Mardia coefficient was 110.99 (CR=36.78), which indicated that the data in this study did not belong to multivariate normal distribution. Thus, in accordance with Wang, Wang, and Jiang's (2011) suggestions, we adopted robust maximum likelihood estimates (MLR) to handle missing data and estimate the model. Before this, however, descriptive statistics of the dataset was performed. In order handle non-normal data, bootstrapped means (standard deviations), medians (minimum-maximum), and correlations were assessed based on 1000 bootstrap samples.

Insert Table 1.

To examine the bi-directional relationships between pain and PTSS over time, we used auto-regressive cross-lagged modelling (ARCL; e.g., Anderson, 1960). ARCL provides an indicator of temporal precedence in the absence of an experimental design. Such designs allow for simultaneous assessment, enabling the examination of whether earlier PTSS predicts later measures of pain and vice versa. In this way, structural relations of repeatedly measured variables of two or more variables are examined (Selig and Little, 2012), also called reciprocal associations (Kearney, 2017). This modeling strategy incorporates two main components. First, later measures of a construct are predicted by earlier measures of the same construct, thus giving rise to the term "autoregressive" (Kearney, 2017). Second,

the "cross-lagged" component is created by which later measures of a construct are predicted by earlier measures of other constructs (Kearney, 2017). The analysis was conducted via structural equation modeling (SEM) as it allows for these more advanced and complex models to be tested (Schumacker and Lomax, 2016). Age and gender were also included in the SEM and were controlled for as covariates. We used Mplus statistics and estimated the model's fit by using several fit indices.

A model is judged as fitting well if the comparative fit index (CFI), that considers sample sizes, normed-fit index (NFI), and the Tucker−Lewis index (TLI) are greater than .95 and as adequate fit if these indices are greater than .90 and root mean square error of approximation (RMSEA) has to be equal or lower than .09. We computed the chi square test, but since it is sensitive to sample size (e.g., Kline, 1998), we used the ratio of chi-square to degrees of freedom. Values between 1 and 5 indicated a satisfactory fit between the theoretical model and empirical data with a more severe cut-off of 3 being ideal (Kline, 1998). Further, as an evaluation of a model is not purely a statistical matter, the model also needs to be evaluated based on theory and earlier empirical findings.

As an add-on to the above model, additional SEMs were conducted in order to examine the prediction and unique variance explained of pain by each cluster of PTSS within the model after controlling for age and gender. These analyses were conducted for each significant path from PTSS to pain in the model.

Results

Descriptive characteristics

253 participants (mean age 38.06 ± 13.34; 66.4% women) were included in the analyses. As this study did not aim to investigate the effect of treatment, participants were free to pursue any form of treatment with 48% of participants reporting doing so. Physiotherapy was the most common form (25%), while other treatments received included chiropractic, acupuncture, and massage. Regarding medication usage, simple analgesics (30%) and nonsteroidal anti-inflammatory drugs (38%) were the most common, while also opioid based medication (20%) and adjuvant medications (10%) were used. At baseline, 20.4% of participants reported that they were not working because of their whiplash-injury with a further 9.7% participants reporting working reduced hours. 48% had post-secondary school education. Table 2 contains means (standard deviations), medians (minimum-maximum), and the correlations of pain and PTSS at all time points.

Insert Table 2.

Model testing

Fit indices that the theoretical model, $\chi^2(24)=78.61$, p=.00, $\chi^2/df=3.28$, CFI=.94, TLI=.89, RMSEA=.10, containing all paths, had a poor fit, indicating that the theoretical model was not a good representation of our data. We wanted to estimate a simpler and parsimonious model, also referred to as a nested model, containing only the significant paths found. In the process of building this parsimonious model, we deleted insignificant paths sequentially one after the other, so that the most insignificant path was first deleted and so on. We compared its fit indices to the general model, arguing for many paths of impact. Non-significant difference of the two chi-squares suggests that the omission of the non-significant parameters did not reduce the model and indicate favors to the simpler model, $\chi^2(4)=6.76$, p=.15 (Ledermann, Macho, & Kenny, 2011). Fit indices of the simpler model indicated that the model, $\chi^2(28)=85.37$, p<.001, $\chi^2/df=3.05$, CFI=.94, TLI=.90, RMSEA=.09, was a good representation of the data. No significant difference was found between the models, $\chi^2(4)=6.76$, p=.149. Hence, we proceeded with the more parsimonious simple model.

Figure 1 displays the standardized coefficients and significant paths for the nested model after controlling age and gender. The analysis revealed high stability of pain and PTSS total score concerning individual differences so that those with high levels of pain and PTSS total score at baseline tended to have high levels of pain and PTSS total score at 3, 6, and 12 months. The relation between pain and PTSS total score at 3 months ($\beta = .33$, $p < .001$), 6 months ($\beta = .32$, $p < .001$), and 12 months ($\beta = .19$, $p = .003$) were found to be significant. More importantly, the analysis revealed that PTSS total score at baseline predicted an increase in pain between baseline and 3 months, and PTSS total score at 6 months predicted an increase in pain between 6 months and 12 months, beyond the stability of pain score over time.

Insert Figure 1.

Further analyses

Two separate SEMs after controlling age and gender were performed in order to examine the prediction and unique variance explained of pain by each cluster of PTSS in the specific associations found in the model. Hence, avoidance, hyperarousal, and intrusion at baseline were tested

as predictors of pain at 3 months and avoidance, hyperarousal, and intrusion at 6 months were tested as predictors of pain at 12 months. First, it was found that after controlling age and gender, baseline hyperarousal significantly predicted pain at 3 months and explained 16.0% of the variance. Second, it was found that hyperarousal at 6 months significantly predicted pain at 12 months and explained 23.0% of the variance. Hence, the higher the hyperarousal symptoms at T1 and T3, the higher the pain at T2 and T4, respectively. For details, see figure 2 and 3.

Insert figure 2 and 3.

Discussion

To our knowledge, this is the first study to investigate the reciprocal, longitudinal associations of pain and PTSS in a cohort of whiplash-injured. Our first objective was to investigate the longitudinal, reciprocal associations between pain and PTSS. Here, we found a temporal effect of PTSS on pain within the first 3 months and again from 6 months onwards, but not the other way around. Our second objective was to assess the predictive value of the PTSS-clusters in order to dig deeper into the specific roles of these symptoms. Here, hyperarousal at baseline and 6 months predicted pain at 3 months and 12 months, respectively, while avoidance and intrusion were not significant predictors at any time point. Taken together, the results point to a temporal main effect of PTSS on pain over and above the stability of pain itself rather than the other way around within the more sub-acute months (<3 months) and again from 6 to 12 months after a whiplash-trauma with symptoms of hyperarousal driving these effects, while there is a slip in the maintenance patterns in the beginning of the chronic phase between 3 and 6 months.

As for our first objective, the results are partially consistent with existing literature. In agreement with Carty et al. (2011), Feinberg et al. (2017), Jenewein et al. (2009), Liedl et al. (2010), and Stratton et al. (2014), it was found that PTSS maintained pain within the first months post-injury; however, we did not find that pain maintained PTSS. Hence, we did not find indication of mutual maintenance in this sub-acute period as previously found (Carty et al., 2011; Feinberg et al., 2017; Jenewein et al., 2009; Liedl et al., 2010; Stratton et al., 2014). This could in part be due to the heterogeneity both within and between samples. Further, more severe physical injuries as included in 3 out of 5 studies (Carty et al., 2011; Jenewein et al., 2009; Liedl et al., 2010) may be associated with higher pain levels that feed directly into the emotional trauma reaction as opposed to a minor, non-

hospitalised injury as WAD. In contrast, it seems that PTSS has a significant effect on pain in WAD in the more sub-acute months post-injury (and again from 6 to 12 months) and not vice versa. Indeed, this importance of sub-acute PTSS on pain (and pain-disability) over time in WAD is consistent with longitudinal studies using other designs (Buitenhuis et al., 2006; Kongsted et al., 2008; Ritchie et al., 2013; Walton et al., 2013). During the beginning of the more chronic phase from 3 to 6 months post-injury, we found that PTSS and pain followed separate trajectories, and once again from 6 to 12 months PTSS maintained pain, but not vice versa. No other study included measurement at both 3 and 6 months, hence making the comparisons of findings complicated. This slip in maintenance from 3 to 6 months is contrary to all existing studies that found continuing inter-relations, however, with mixed findings on the specific direction. The re-occurring of maintenance of PTSS on pain from 6 to 12 months is, on the other hand, in accordance with Jenewein et al. (2009) and Stratton et al. (2014) that found that PTSS maintained pain in chronicity, while it was partly in accordance with Feinberg et al. (2017) and Liedl et al. (2010) who found mutual maintenance to continue into chronicity when looking at specific PTSS-clusters. Finally, this finding was different from Carty et al. (2011) that reported that only pain maintained PTSS in this phase. Taken together, the picture is truly blurred. These differences in results could be due to differences in methods and samples; however, even with similar samples (Carty et al., 2011; Jenewein et al., 2009; Liedl et al., 2010), results are inconsistent. Generally, there was a lack of information on injury and trauma and importantly whether same trauma caused both conditions in existing studies, which makes it difficult to compare. Further, existing studies had only 3 measurement-points with differences in the second follow-up (either at 3 or 6 months), while our study had 4 measurement-points (0, 3, 6, and 12 months). This may illuminate the relationship more accurately and also explain the differences in findings.

Additionally, our secondary findings indicate that hyperarousal is the driving factor of PTSS' effect on pain. This is in agreement with Liedl et al., who proposed hyperarousal to have direct effect on pain in a theoretical model (Liedl and Knaevelsrud, 2008), and after empirical testing found that hyperarousal indeed was a key mechanism (Liedl et al., 2010). The importance of hyperarousal in WAD has been highlighted previously (Buitenhuis et al., 2006; Kenardy and Dunne, 2011; Ritchie et al., 2013; Sterling and Chadwick, 2010) and in chronic pain in general (Cho et al., 2011; López-Martínez et al., 2014). Hence, it seems evident that hyperarousal is a driving factor of PTSS in relation to pain. This could be due to catastrophic misperceptions and negative interpretations of somatic

sensations that enhance the fear of and focus on pain and in turn fuel the pain experience itself (Kenardy and Dunne, 2011; Sharp and Harvey, 2001).

In relation to our theoretical framework, our first finding partially supports the mutual maintenance model (Sharp and Harvey, 2001) in that PTSS maintain pain, but we did not find support for the proposition that pain too maintains PTSS. Our second findings also partly support the model as hyperarousal is proposed as an important process within the model (Sharp and Harvey, 2001). However, we did not find evidence of the importance of avoidance nor intrusion symptoms with, e.g., pain being a chronic trigger of fear responses (Liedl and Knaevelsrud, 2008; Sharp and Harvey, 2001; Taylor et al., 2012). Likewise, the apparent time-dependent development of the inter-relations suggested by this study is not addressed by Sharp and Harvey (2001), but it makes sense that these are ongoing, changing processes, where the conditions could relate differently at different stages.

In terms of discussing the applicability of the mutual maintenance-perspective, it is important to keep in mind that the current sample is a consecutive cohort of whiplash-injured and consequently report a range of PTSS-levels. It is plausible that there could be a more entangled relational pattern in a selected sample of whiplash-injured with clinical levels of PTSS. Additionally, studies relying on self-report measurement of PTSS are likely to capture a much broader construct characterized by hyperarousal. Furthermore, it is possible that pain and PTSS influence each other indirectly through other processes not captured by the current model. For example, it has been suggested that PTSS causes altered pain processing and perception (Moeller-Bertram et al, 2012; Pedler et al., 2016; Sterling and Chadwick, 2010), and it could be that PTSS indirectly affect pain through these altered mechanisms. Likewise, it has also been reported that the relationship between pain and PTSS is mediated by fear-avoidance-variables (Andersen et al., 2016) as also indicated by the earlier studies (Carty et al., 2011; Martin et al., 2010). This suggests that PTSS and pain might exacerbate one another through mechanisms not tested in this study. Hence, there might be a multi-causal explanatory model in which acute distress and hyperarousal initiate catastrophizing and fear which leads to avoidance and then again to disability and enhanced pain, and thereby trigger a dysfunctional fear-avoidance cycle similar to the one proposed by the fear-avoidance-model (Vlaeyen and Linton, 2000). Here, PTSS should be included as a maintaining factor, as also proposed recently by Pedler et al. (2016).

From a clinical viewpoint, these results highlight the importance of screening for PTSS among whiplash-patients as early as possible. Early targeting of PTSS may be important, as these

symptoms seem to maintain pain within the initial period, while the two conditions seem to run more separate courses in the beginning of the more chronic phase before returning to a unidirectional maintenance pattern again after 6 months. This suggests a need for independent treatment initiatives in which both conditions are addressed and that treatment of PTSS at some time may have a direct effect on pain as well.

Besides several strengths of the study such as the longitudinal design with four time points, the homogeneity of the sample, and the powerful statistical analyses, a number of limitations should be considered in interpretation of the results. Firstly, the main objective of the study was to test the unique interrelations of pain and PTSS. However, a number of covariates may have an impact such as more process-related variables (e.g., pain-catastrophizing) or background information (e.g., medication usage). Unfortunately, the present sample size did not allow analysis of more complex models. Similarly, two samples were merged for the current study. These may differ in important ways that could impact the results. Therefore, the results ought to be replicated in a sample consecutively collected for the purpose. Secondly, the measurement relied on self-report measurements. This may cause the PTSS ratings to be inflated by others symptoms. A clinician-administered PTSD-interview would have minimized this problem and strengthened the findings. Also, a larger sample could enable testing of reciprocal relations in a subgroup of whiplash-injured with clinical PTSS. Thirdly, usage of the newer DSM-5 and not DSM-IV would have been ideal; however, this is considered acceptable, as it was only PTSS and not PTSD that was tested. Finally, as very different findings emerge across studies, it may be that these symptoms and the reciprocal fuelling of them may differentiate a great deal over time and may be more dominant within initial days and weeks post-injury, something not tested with the current design.

Conclusions

Taken together, the current results give several important insights into the reciprocal, longitudinal relationship between pain and PTSS, and hereby add to the knowledge on the specific role of PTSS in WAD. Moreover, the results may have important clinical relevance in terms of prevention. However, whether early targeting of PTSS can prevent the development of chronic WAD will need to be tested in a randomized, controlled trial. Likewise, future studies need to address some of the challenges pointed out in the current study as well as looking more into other methods for dealing with time ordering and causal analysis.

Acknowledgments and conflict of interest

The authors declare no conflict of interest and have no acknowledgements.

Author contributions

All authors have participated in shaping and discussing the study and commenting critically on the paper several times during the process. Dr. Lahav has been in charge of conducting the SEMs.

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Legends

Table 1. Skewness and kurtosis values of pain and PTSS at all time points.
SE = standard errors; PTSS = posttraumatic stress symptoms.

Table 2. Bootstrapped characteristics and correlations of pain and PTSS at all time points.
SD = standard deviations; min = minimum; max = maximum; PTSS = posttraumatic stress symptoms.

Figure 1. Auto-regressive cross-lagged model assessing bidirectional relationships between pain and PTSS total score across time (standardized coefficients). Curved lines represent covariates between constructs. Solid lines represent significant predictions. * p <.05, ** p <.01, *** p <.001.
PTSS = posttraumatic stress symptoms.

Figure 2. Hyperarousal, intrusion, and avoidance at T1 as predictors of pain at T2.
$\chi^2(2)=4.78$, CFI=.99, TLI=.94, RMSEA=.07

Figure 3. Hyperarousal, intrusion, and avoidance at T3 as predictors of pain at T4.
$\chi^2(2)=.44$, CFI=1.00, TLI=1.03, RMSEA=.00

	Pain T1	Pain T2	Pain T3	Pain T4	PTSS T1	PTSS T3	PTSS T3	PTSS T4
Skewness (SE)	.34 (.15)	.78 (.16)	.96 (.17)	.69 (.17)	.71 (.15)	1.32 (.17)	1.85 (.17)	1.59 (.17)
Kurtosis (SE)	-.59 (.31)	-.16 (.32)	-.07 (.33)	-.72 (.33)	-.33 (.31)	1.09 (.33)	4.06 (.34)	2.07 (.34)

Measure	1	2	3	4	5	6	7	8
1. Pain T1	-							
2. Pain T2	.49 (.34-.62)	-						
3. Pain T3	.47 (.35-.57)	.66 (.52-.76)	-					
4. Pain T4	.40 (.25-.52)	.57 (.43-.71)	.74 (.64-.83)	-				
5. PTSS T1	.35 (.20-.48)	.42 (.26-.58)	.32 (.18-.47)	.37 (.23-.51)	-			
6. PTSS T2	.22 (.10-.34)	.46 (.29-.62)	.42 (.25-.56)	.42 (.27-.55)	.73 (.63-.81)	-		
7. PTSS T3	.33 (.20-.46)	.39 (.20-.57)	.49 (.34-.61)	.48 (.34-.61)	.62 (.51-.72)	.83 (.73-.89)	-	
8. PTSS T4	.28 (.15-.41)	.45 (.26-.61)	.47 (.33-.60)	.53 (.39-.66)	.56 (.43-.69)	.81 (.71-.88)	.84 (.74-.91)	-
Mean (SD)	3.98 (2.09)	2.44 (2.22)	2.28 (2.37)	2.18 (2.42)	15.23 (11.17)	10.27 (10.39)	8.45 (9.53)	7.99 (9.56)
Median (min-max)	4.00 (0.10-9.60)	2.00 (0.00-8.70)	1.40 (0.00-8.60)	1.30 (0.00-9.30)	13.50 (0-41)	7.00 (0-45	6.00 (0-46)	4.00 (0-48)