PTSD Perpetuates Pain in Children With Traumatic Brain Injury

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Objective  This study tested theoretical models of the relationship between pain and posttraumatic stress disorder (PTSD) in children with traumatic brain injury (TBI).  Methods  Participants consisted of 195 children aged 6–15 years presenting to 1 of 3 Australian hospitals following a mild–severe TBI. Children were assessed at 3, 6, and 18 months after their accident for PTSD (via the Clinician-Administered PTSD Scale for Children and Adolescents [CAPS-CA] clinical interview) as well as physical pain (via the Child Health Questionnaire, 50-item version [CHQ-PF50]). Trained clinicians administered the CAPS-CA at home visits, and the CHQ-PF50 was collected through questionnaires.  Results  Structural equation modeling found the data supported the mutual maintenance model and also the nested perpetual avoidance model.  Conclusions  Both models indicate PTSD is driving the presence of pain, and not vice versa. A fourth model stating this was proposed. Therefore, it may be useful to address PTSD symptoms in treating child pain for expediting recovery.

Key words  children; mutual maintenance; pain; perpetual avoidance; posttraumatic stress; PTSD; TBI; traumatic brain injury.

Recovering from an injury is a relatively common experience for a child. Australian children are more commonly injured than adults, with almost one in five children (0–14 years old) sustaining injuries severe enough to need medical attention (Australian Bureau of Statistics, 2006). Children with injuries tend to have elevated rates of posttraumatic stress disorder (PTSD): Full and subsyndromal rates are as high as 47.5% (Aaron, Zaglul, & Emery, 1999; Daviss et al., 2000; Scheeringa, Wright, Hunt, & Zeanah, 2006). A systematic review (King et al., 2011) has also found high prevalence of pain in children, varying by bodily region. For example, abdominal pains were prevalent in 4–53% of children, while head pains ranged from 8 to 83%. With regard to traumatic brain injury (TBI) specifically, a cohort study has found pediatric TBI cases continued to report higher pain levels than pediatric arm injury cases, up to 12 months after injury (Blume et al., 2012).

In general, PTSD and pain are highly comorbid (Otis, Keane, & Kerns, 2003; Villano et al., 2007), and research on this relationship within TBI samples also find this relationship (Nampiaparampil, 2008; Taylor et al., 2012). However, research into the perpetuating influences, particularly in children, is less established.

A recent meta-analysis on risk factors for PTSD in children did not examine the role of pain (Trickey, Siddaway, Meiser-Stedman, Serpell, & Field, 2012) despite pediatric TBI samples showing high comorbidity of pain (Blume et al., 2012). Other research suggests children react differently than adults to pain (Otis, Pincus, & Keane, 2006) and also show differences in the manifestation of posttraumatic stress (Scheeringa, Zeanah, & Cohen, 2011). These differences highlight the relationship between PTSD and pain may be different in children. Therefore, any research explaining the relationship between PTSD and pain in adults should not assume replication in child samples.

Saxe et al. (2001) have begun to investigate this relationship in children with acute burns. They found morphine administration reduced long-term PTSD in a
small sample of children (n = 24), but initial reported pain was not predictive of PTSD symptoms at 6 months. More recently, Saxe et al. (2005) presented a research-driven model that suggested injury severity predicted PTSD at 3 months. However, this model omitted a direct pain and PTSD relationship despite the results reporting a significant association. This finding provides support for the interaction of pain and PTSD in children, although further research needs to define the nature of the relationship.

There are three proposed models in the literature to explain the relationship between pain and PTSD. Each model has empirical support using adult samples, but the present study will test these models in a child sample. Sharp and Harvey (2001) conceptualized the mutual maintenance model, and this model was empirically supported by Liedl et al. (2010) using an adult hospitalized injury sample. The mutual maintenance model suggested that just as pain stimulates the presence of PTSD symptoms, the presence of PTSD also perpetuates the presence of pain. The other two models are nested within this first model. Norman, Stein, Dimsdale, and Hoyt (2008) theorized and tested a model in which pain underpins the continuance of PTSD (but PTSD does not prolong pain). For the purposes of this article, we named this the pain model. Alternatively, Liedl and Knaevelsrud (2008) proposed the perpetual avoidance model, taking the opposite view to Norman et al. (2008), that PTSD symptoms (namely, arousal) facilitate the maintenance of pain (which in turn prolongs PTSD). The perpetual avoidance model also has been empirically supported in an adult disaster sample (Dirkzwager, van der Velden, Grievink, & Yzermans, 2007). The empirical support for these models indicates there is a definite relationship between PTSD and pain. However, it also suggests more work is required to discern the direction of relationship. Only Liedl et al. (2010) used structural equation modeling (SEM) to test predictors; the other studies used logistic regression, which only allows testing one hypothesized direction. Another benefit to further testing is to find support for the application of these models in other populations, such as children.

The present study aims to test the mutual maintenance, pain, and perpetual avoidance models of pain and PTSD in children with TBI. SEM will be used to compare the three models to clarify the exact nature of the relationship between pain and PTSD in children.

**Method**

**Participants**

Data for the current report were collected as part of a wider prospective longitudinal study analyzing the longitudinal neurocognitive and psychosocial outcomes of children with a TBI (Kenardy et al., 2012). Participants were recruited between April 2004 and December 2006 at three Australian hospitals: Royal Children’s Hospital Melbourne, Royal Children’s Hospital Brisbane, and Mater Children’s Hospital Brisbane. Children aged 6–15 years who attended the emergency department or were admitted to the hospital following a mild to severe TBI were eligible for inclusion in the study. Children were required to have a documented alteration in consciousness (e.g., loss of consciousness, posttraumatic amnesia (PTA), coma, disorientation and confusion, or loss of recall for event). Families were not eligible for the study if their English was insufficient to complete questionnaires, the child had previous neurological or developmental disorders, PTA was >28 days, or the injury was deliberate (i.e., from abuse or self-harm). Participants of interest were included in this analysis if they had provided questionnaire or interview data at 3 months (T1), 6 months (T2), or 18 months (T3) after TBI. Of 428 eligible families, 195 participants had available data for this analysis. As this is a clinical sample recruited through general hospitals, uptake was considered reasonable, especially because no intervention or compensation was offered. Australian federal privacy regulations prohibit collection of data on approached families who refused to participate; however, as explored in previous papers on this data set (Anderson et al., 2012; Iselin, Le Brocque, Kenardy, Anderson, & McKinlay, 2010), consenting participants with missing data, and dropped out versus those who completed each time point, did not significantly differ.

The retention rate was high: 85% of the available sample completed the final time point. The study invited participants to be involved at later time points regardless of earlier participation to maintain a larger sample size. See Figure 1 for full participant flow.

The majority of the participants were male (n = 137, 70%), and the average age of child at the time of the TBI was 10.78 years (range: 6–15 years, standard deviation [SD] = 2.49). TBIs were primarily caused by falls (n = 106, 54%) or motor vehicle accidents (n = 39, 20%), and a minority were attributed to sporting injuries (n = 27, 14%) or receiving a physical blow to the head (n = 23, 12%).

**Measures**

**Posttraumatic Stress**

Posttraumatic stress was measured with the widely used Clinician-Administered PTSD Scale for Children and Adolescents (CAPS-CA, Nader et al., 1996). The CAPS-CA was administered by graduates with at least honors degree qualifications in psychology with high inter-rater
consistency (intraclass correlation) ICC = .983, p < .001). Posttraumatic stress symptoms were analyzed as a continuous scale (total severity). A greater score indicated more PTSD symptom severity.

Bodily Pain
Pain was assessed using the bodily pain scale of the Child Health Questionnaire, 50-item version (CHQ-PF50, Landgraf, Abetz, & Ware, 1996). This questionnaire is completed by parents and is about their child. The bodily pain and discomfort scale consists of two items scored on a 6-point scale: “During the past four weeks, how much bodily pain or discomfort has your child had?” (1 = none, 6 = very severe) and “During the past four weeks, how often has your child had bodily pain or discomfort?” (1 = none of the time, 6 = every day or almost every day). Consistent with the CHQ-PF50 scoring instructions, these items were recoded and transformed into standardized scores ranging from 0 to 100. Higher scores indicated less reported pain, such that higher levels of bodily pain were indicated in the data by lower scores on the scale. The bodily pain subscale has shown good internal consistency (α = 0.88) and test–retest reliability (ICC > 0.60) in an Australian sample (Waters, Salmon, & Wake, 2000).

Procedure
This study was approved and conducted in accordance with the Mater Health Services Human Research Ethics Committee, Royal Children’s Hospital and Health Services District Ethics Committee (Brisbane), Royal Children’s Hospital and Melbourne Ethics and Research Office, and The University of Queensland Ethics Committee. Eligible children were identified by nursing staff in the emergency department or hospital wards, and parents were invited to take part shortly thereafter. Informed written consent was obtained from parents and children aged ≥10 years. The CAPS-CA was administered at T1 to allow instances of acute neurological dysfunction to remit. Follow-up assessments were conducted at T2 and T3. Interviews were conducted during home visits, and the CHQ-PF50 questionnaires were returned by post. Demographic data were collected before the T1 assessment.

Results
Data Analyses
Descriptive analyses were run with SPSS 21 for Windows, and SEM was performed in AMOS 20. Preliminary analyses revealed all variables were within acceptable skewness and kurtosis ranges for SEM (Kline, 2005). The majority (62%) of participants had complete data (overall 86% data points were complete). Statistical methods used provided valid inferences under the assumption that data were missing at random. Schafer and Graham (2002) suggest all data are used to estimate longitudinal data using maximum likelihood and this technique was used through the AMOS program.

Intercorrelations
Intercorrelations are shown in Table I. At each time point, bodily pain and PTSD symptoms were significantly correlated, such that more PTSD symptoms were associated with more bodily pain. Bodily pain scores were consistently correlated across time points, as were PTSD symptoms. For example, T1 bodily pain was positively associated with T2 bodily pain. Correlations were also found across variables, across time. For example, T1 PTSD symptoms were associated with T2 bodily pain, such that more PTSD symptoms were correlated with more bodily pain. The high degree of the correlations shows the theorized models should test the strength of relationships, after accounting for other relationships.

Substantive Analyses
The time-lag models were set up to be recursive, in line with Kline (2012), such that the exogenous variables (T1 PTSD symptoms and T1 bodily pain) were allowed to covary, as were the error terms of the endogenous variables (T2 and T3 variables), both within and across time points.
T1 and T3 associations were investigated (i.e., T1 PTSD predicting T3 PTSD; T1 PTSD predicting T3 bodily pain); however, none significantly added to the model and therefore were omitted from final analyses. The models presented in Figure 1 show standardized regression weights and significances, which are also selectively reported below. Weights varied slightly between models. Table II shows the associated unstandardized regression weights and confidence intervals.

The mutual maintenance model fit the data well, $\chi^2 (2, n = 195) = 1.21, p = .545$. The model had good absolute fit: comparative fix index (CFI) = 1.00, incremental fit index (IFI) = 1.02, normed fit index (NFI) = 1.00, and residual fit, root mean square error of approximation (RMSEA) = 0.00. T1 PTSD symptoms predicted T2 bodily pain, $\beta = -0.20, p < .001$. However, T1 bodily pain did not predict T2 PTSD symptoms, $\beta = -0.10, p = .062$. Neither relationship was significant at 18 months: T2 PTSD symptoms did not predict T3 bodily pain, and T2 bodily pain did not predict PTSD symptoms, $\beta$s < 0.01, ps > .874.

The perpetual avoidance model also fit the data well, $\chi^2 (3, n = 195) = 4.57, p = .206$. Absolute fit indices indicated the model had good fit: CFI = 1.00, IFI = 1.00, NFI = 0.99, and acceptable residual fit, RMSEA = 0.05. T1 PTSD symptoms predicted T2 bodily pain, $\beta = -0.20, p < .001$. However, T2 PTSD symptoms did not predict T3 bodily pain, nor did T2 pain predict T3 PTSD symptoms, $\beta$s < 0.02, ps > .818.

The pain model did not fit the data well, $\chi^2 (4, n = 195) = 13.71, p = .008$. Absolute fit indices were acceptable: CFI = 0.97, IFI = 0.97, NFI = 0.96, but the residual fit was poor, RMSEA = 0.11. T1 bodily pain as a predictor of T2 PTSD symptoms was not significant, $\beta = -0.11, p = .053$, nor did T2 bodily pain predict T3 PTSD symptoms, $\beta = 0.00, p = .976$. The lack of absolute

### Table I. Intercorrelations, Means, and Standard Deviations of PTSD and Bodily Pain Scores Across Time Points

<table>
<thead>
<tr>
<th></th>
<th>T1: 3 months after TBI</th>
<th>T2: 6 months after TBI</th>
<th>T3: 18 months after TBI</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Bodily pain</td>
<td>PTSD symptoms</td>
<td>Bodily pain</td>
</tr>
<tr>
<td>Mean</td>
<td>77.23</td>
<td>18.77</td>
<td>80.01</td>
</tr>
<tr>
<td>T1 PTSD symptoms</td>
<td>$-0.19^*$</td>
<td>$-0.29^{**}$</td>
<td>$-0.18^*$</td>
</tr>
<tr>
<td>T2 PTSD symptoms</td>
<td>$-0.51^{***}$</td>
<td>$-0.32^{***}$</td>
<td>$-0.18^*$</td>
</tr>
<tr>
<td>T1 bodily pain</td>
<td>$0.50^{***}$</td>
<td>$0.59^{***}$</td>
<td>$0.48^{***}$</td>
</tr>
</tbody>
</table>

Note. The CHQ-PF50 Bodily Pain scale is negatively scored, such that lower scores indicate more pain. **p < .001, ***p < .01, *p < .05. PTSD = posttraumatic stress disorder.

### Table II. Unstandardized Regression Weights and Confidence Intervals for Each Theoretical Model

<table>
<thead>
<tr>
<th>Model</th>
<th>B</th>
<th>95% CI</th>
<th>B</th>
<th>95% CI</th>
<th>B</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mutual maintenance model</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>T1 bodily pain ↔ T1 PTSD symptoms$^a$</td>
<td>$-46.56$</td>
<td>$-88.41$ to $-4.71$</td>
<td>$-46.56$</td>
<td>$-88.41$ to $-4.71$</td>
<td>$-46.56$</td>
<td>$-88.41$ to $-4.71$</td>
</tr>
<tr>
<td>T1 bodily pain → T2 bodily pain</td>
<td>$0.50$</td>
<td>$0.38$ to $0.61$</td>
<td>$0.48$</td>
<td>$0.36$ to $0.60$</td>
<td>$0.53$</td>
<td>$0.41$ to $0.65$</td>
</tr>
<tr>
<td>T1 bodily pain → T2 PTSD symptoms</td>
<td>$-0.06$</td>
<td>$-0.10$ to $0.02$</td>
<td>$-0.06$</td>
<td>$-0.12$ to $0.00$</td>
<td>$-0.06$</td>
<td>$-0.12$ to $0.00$</td>
</tr>
<tr>
<td>T1 PTSD symptoms → T2 PTSD symptoms</td>
<td>$0.49$</td>
<td>$0.40$ to $0.58$</td>
<td>$0.51$</td>
<td>$0.41$ to $0.60$</td>
<td>$0.46$</td>
<td>$0.37$ to $0.56$</td>
</tr>
<tr>
<td>T1 PTSD symptoms → T2 bodily pain</td>
<td>$-0.30$</td>
<td>$-0.43$ to $-0.17$</td>
<td>$-0.31$</td>
<td>$-0.43$ to $-0.18$</td>
<td>$-0.31$</td>
<td>$-0.43$ to $-0.18$</td>
</tr>
<tr>
<td>T2 bodily pain → T3 bodily pain</td>
<td>$0.61$</td>
<td>$0.56$ to $0.66$</td>
<td>$0.59$</td>
<td>$0.54$ to $0.64$</td>
<td>$0.61$</td>
<td>$0.56$ to $0.66$</td>
</tr>
<tr>
<td>T2 bodily pain → T3 PTSD symptoms</td>
<td>$0.01$</td>
<td>$0.07$ to $0.05$</td>
<td>$0.01$</td>
<td>$0.06$ to $0.05$</td>
<td>$0.00$</td>
<td>$0.06$ to $0.05$</td>
</tr>
<tr>
<td>T2 PTSD symptoms → T3 PTSD symptoms</td>
<td>$0.74$</td>
<td>$0.54$ to $0.95$</td>
<td>$0.71$</td>
<td>$0.52$ to $0.91$</td>
<td>$0.72$</td>
<td>$0.52$ to $0.91$</td>
</tr>
<tr>
<td>T2 PTSD symptoms → T3 bodily pain</td>
<td>$-0.01$</td>
<td>$-0.25$ to $0.23$</td>
<td>$-0.02$</td>
<td>$-0.22$ to $0.20$</td>
<td>$-0.02$</td>
<td>$-0.22$ to $0.20$</td>
</tr>
<tr>
<td>T2 pain error ↔ T2 PTSD error$^a$</td>
<td>$-24.13$</td>
<td>$-46.23$ to $-2.03$</td>
<td>$-23.81$</td>
<td>$-46.05$ to $-1.57$</td>
<td>$-26.16$</td>
<td>$-48.24$ to $-4.08$</td>
</tr>
<tr>
<td>T2 pain error ↔ T3 pain error$^a$</td>
<td>$-107.21$</td>
<td>$-190.90$ to $-23.52$</td>
<td>$-102.25$</td>
<td>$-183.65$ to $-20.85$</td>
<td>$-107.32$</td>
<td>$-188.29$ to $-26.35$</td>
</tr>
<tr>
<td>T3 pain error ↔ T3 PTSD error$^a$</td>
<td>$-31.08$</td>
<td>$-52.05$ to $-10.11$</td>
<td>$-31.81$</td>
<td>$-52.74$ to $-10.88$</td>
<td>$-31.48$</td>
<td>$-52.43$ to $-10.53$</td>
</tr>
<tr>
<td>T2 PTSD error ↔ T3 PTSD error$^a$</td>
<td>$-27.68$</td>
<td>$-48.38$ to $-6.98$</td>
<td>$-24.61$</td>
<td>$-44.64$ to $-4.58$</td>
<td>$-25.44$</td>
<td>$-45.41$ to $-5.47$</td>
</tr>
</tbody>
</table>

Note. $^a$Covariance estimate.

T1 = 3 months, T2 = 6 months, T3 = 18 months; B = unstandardized regression weight; CI = confidence interval; PTSD = posttraumatic stress disorder.
fit indicated that the other two models better explained the data than the pain model. A statistical comparison of models revealed the mutual maintenance model did significantly fit the data better than the nested pain model, according to a chi-square difference test, $\Delta \chi^2 (2) = 12.50$, $p = .002$, offering a better account of the observed relationships in the data. However, when comparing the mutual maintenance model with the nested perpetual avoidance model, the chi-square difference test was not significant, $\Delta \chi^2 (1) = 3.36$, $p = .067$, suggesting the perpetual avoidance model fit just as well as the mutual maintenance model.

**Discussion**

This article investigated the directional nature of the pain and PTSD relationship in the months after a TBI in a sample of Australian children. While there is clear evidence that PTSD and pain are related after injury, the directional relationship, particularly in children, remains relatively unexplored. Within the pediatric literature, two studies have found pain to have an indirect association with PTSD (Saxe et al., 2005; Stoddard et al., 2006), but no study has examined the relationship of PTSD and pain in children. Within adult samples, evidence for the nature of the relationship has been conflicting (i.e., different models supported by Dirkzwager et al., 2007; Liedl et al., 2010; Norman et al., 2008). Our aim was to test the nature of the relationship in a pediatric sample and, in general, extend empirical knowledge of the PTSD–pain relationship. SEM was used to test and compare three proposed models: Mutual maintenance, perpetual avoidance, and the pain model.

We found both the mutual maintenance and perpetual avoidance models fit the data equally well (similar to Dirkzwager et al., 2007; Liedl et al., 2010), seemingly because both models included the significant T1 PTSD predicting T2 pain pathway. The data did not support the pain model (Norman et al., 2008). Closer inspection of the pathways revealed pain did not contribute to PTSD at any time point (although it approached significance), even though the correlation analyses suggest a reciprocal relationship. Moreover, the data suggest that PTSD predicted pain (but only in the short term), which also does not reflect the perpetual avoidance model (Liedl & Knaevelsrud, 2008). This suggests that a fourth model is required to explain the data. We propose the posttraumatic maintenance model, wherein posttraumatic distress influences the level of bodily pain; however, bodily pain does not encourage posttraumatic distress. The other relationships in the tested models were as expected: Earlier pain levels predicted later pain levels, and, similarly, earlier PTSD symptoms predicted later PTSD symptoms. Our data also replicated results found in Liedl et al. (2010) that PTSD and pain correlations strengthened over time.

Underlying mechanisms for these relationships have been hypothesized by Liedl et al. (2010). For PTSD predicting pain, they suggest (a) the hypervigilance associated with PTSD creates attentional bias to pain, (b) reexperiencing associated with PTSD disrupts interpretations of pain sensation, or (c) the avoidance behaviors for rehabilitation may in turn result in more pain long term. As these three symptoms make up the broad PTSD diagnosis, it is possible a combination of all three hypotheses is at work to prolong the experience of pain. Conversely, for pain predicting PTSD symptoms, they suggest the experience of pain provokes traumatic memories of the incident, which activates PTSD symptoms of arousal and avoidance.
behaviors. Our SEM analysis did not find support for this specific pathway, suggesting the former hypotheses more likely.

While an association was found between T1 PTSD symptoms and T2 bodily pain, the lag between T2 PTSD symptoms and T3 bodily pain was not significant. A 1-year gap is potentially too long to test an association, although Liedl et al. (2010) found 3-month PTSD to significantly drive 12-month pain (9-month gap). Perhaps a shorter gap (i.e., 6 months) would elicit a similar association, and future research could consider this.

The T1 bodily pain to T2 PTSD symptoms association was marginally significant. It is possible our relatively small sample size contributed to this marginal finding: While our sample consisted of <200 participants, Liedl et al. (2010), who found significant pain to PTSD relationships, used >800 participants. The larger sample size would have increased power and potentially helped find significant effects. Further, we found significant correlations between earlier pain and later PTSD, which also suggests support for the mutual maintenance model.

This article pertained to bodily pain that occurs in a TBI sample, but we believe our finding is potentially generalizable to any pain sample in a pediatric setting. Other studies have used samples with a range of causes of pain to examine PTSD and pain relationships (i.e., hospitalized injuries: O’Donnell et al., 2013; burn victims: Saxe et al., 2005). However, factors associated with TBI, such as the unique symptomatology of pain, may affect the interpretation and generalizability of current findings. Nampiaparampil (2008) found TBI to be directly associated with ongoing pain (beyond the influence of a PTSD diagnosis), and Olek and Defrin (2007) found chronic pain developed in TBI patients with damaged pain and temperature systems in the brain. With regard to TBI severity, it appears patients with mild TBI report more pain than patients with severe TBI (Nampiaparampil, 2008). Further, patients with TBI most commonly report headaches (Nampiaparampil, 2008); however, patients with muscle tension experience more pain than patients with headaches (De Leeuw, Schmidt, & Carlson, 2005). Therefore, it is possible patients with TBI experience pain differently than patients with other injuries. These particulars of pain and TBI may explain why our data did not find pain to predict PTSD, and future research using TBI samples might consider controlling for pain location.

Another point to raise is the significant associations between the error variances. These associations suggest a third unidentified variable is affecting the child’s recovery. For example, family dynamics may affect the child: Research has found the level of parent’s distress to affect child’s distress following a burn injury (De Young, Hendrikz, Kenardy, Cobham, & Kimble, 2014). Another explanation may be the extent of medical treatment: Research has found an increased morphine dose to children with burns significantly reduces the child’s level of distress (Saxe et al., 2001). It is possible these outside factors, or even others unnamed, are responsible for relating the pain and PTSD error variances. Future research could look at the addition of these factors in similar model testing.

A strength of the study was the longitudinal follow-up with minimal dropout. Also, previous pain and PTSD relationship testing within child samples has only analyzed data in one direction. The use of SEM in this study enabled us to test both directions simultaneously. A clear limitation of the present study was that the pain assessment was a proxy. However, researchers have tested the validity of the CHQ-PF50 on large samples, and it has been established as a valid measure of child pain (Raat, Botterweck, Landgraf, Hoogeveen, & Essink-Bot, 2005; Waters et al., 2000). Future research could replicate this relationship with a more direct measure of child pain such as the Faces Pain Scale-Revised, suitable for children aged 4–12 years, or the visual analog scale, relevant for children ≥8 years (McGrath et al., 2008).

Assessment and treatment of PTSD symptoms in children recovering from an accident or injury will likely promote better psychological outcomes for children and improve pain management. Our research shows it makes sense to screen for PTSD, and if screening is positive, an appropriate intervention needs to be available. The first step, however, is to develop a predictive screen for the range of pediatric TBI samples. This concept is not unique: Research using a whiplash and PTSD adult sample has developed a risk screen for PTSD hyperarousal to predict ongoing pain (Ritchie, Hendrikz, Kenardy, & Sterling, 2013). A similar TBI screener may include preaccident functioning (Anderson et al., 2012).

Beyond a predictive screen, an intervention should be readily available for at-risk children. We recommend using a multidisciplinary approach of psychologists and medical staff working in an integrated way to address the aftereffects of injuries and pain. Again, research on whiplash injury has theorized (Dunne, 2010) and successfully demonstrated this approach in a clinical setting (Dunne, Kenardy, & Sterling, 2012). One caveat with developing an intervention is the availability and cost-effectiveness of the intervention. March et al. (2014) report the need to ensure feasibility utility of screen and treat programs in routine care. Likewise, any screening tool and subsequent
intervention developed for PTSD in pediatric TBI samples with pain should follow these guidelines.

In summary, this article tested different theoretical models of the relationship between PTSD and pain. The data suggest PTSD is the driving cause of lingering pain in children with TBI.

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**Conflicts of interest:** None declared.

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