Published in final edited form as:

Pediatr Emerg Care. 2020 October ; 36(10): e549–e557. doi:10.1097/PEC.0000000000001404.

Posttraumatic stress in children after injury: The role of acute pain and opioid medication use

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Introduction

Unintentional injury is a leading cause of morbidity and mortality for youth around the world, with tens of millions requiring hospital care each year.^{1,2} Although most children recover physically after an injury, a significant subset experience negative psychological sequelae that impair emotional and physical functioning.^{3,4} In particular, nearly one fifth of injured children demonstrate persistent posttraumatic stress symptoms (PTSS),⁵ which can occur regardless of injury severity or mechanism.^{6,7} Given the prevalence of pediatric injury, identifying PTSS risk factors has notable public health significance and may facilitate improved screening and prevention efforts to promote optimal recovery and quality of life after injury.

In the context of pediatric medical events, traumatic stress refers to a “set of psychological and physiological responses of children and their families to pain, injury, serious illness, medical procedures, and invasive or frightening treatment experiences.”⁸ Specifically, PTSS can include re-experiencing, avoidance of trauma reminders, hyperarousal, dissociation, and mood or cognition changes.⁹ Traumatic stress reactions can be extremely distressing for children and families and can significantly hinder recovery post-injury. In particular, PTSS interfere with medical adherence and are linked to greater healthcare service utilization as

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Conflicts of Interest: None to declare

well as poorer functioning, quality of life, and health outcomes.^{10–13} Hence, PTSS represent a considerable barrier to physical and emotional health and recovery after pediatric injury.

Identifying risk factors for PTSS development and maintenance is essential for effective screening and prevention. Evidence suggests that various biological (e.g., high heart rate or cortisol), psychological (e.g., prior trauma history, preexisting mental health concerns, acute stress reactions, maladaptive cognitions), and environmental factors (e.g., parent PTSS, poor family functioning, low social support) may increase risk for the development of PTSS in children after injury.^{5–7,14} However, investigations of pediatric PTSS have largely neglected factors during the peri-trauma phase (i.e., time between the potentially traumatic event and completion of acute medical care),¹⁵ when screening and preventive interventions may be most feasible and as trauma memories and associated symptoms are consolidating. In addition to gaps in the knowledge base, effective screening for risk of psychosocial sequelae during acute medical care is challenging given time and training limitations for healthcare professionals and difficulties distinguishing normative distress from signs of risk. Furthermore, seemingly intuitive indicators of risk such as injury severity do not reliably predict psychological outcomes in youth.^{3,16} As a result, clinicians' perceptions of psychological risk are often inaccurate.^{17,18} In particular, emergency care clinicians are often on the front lines of post-injury treatment and can play a significant role in early identification of high emotional distress; however, emergency physicians and nurses vary widely in their awareness of medical traumatic stress following pediatric injury.¹⁸ Identifying peri-trauma risk factors for PTSS that can be easily assessed during medical care would greatly enhance our ability to direct early prevention and intervention efforts towards children and families who are most in need.

As a routine component of medical care, acute pain assessment may be a promising avenue for screening for risk of PTSS. While interactions between acute pain and traumatic stress are not well understood,^{19,20} preliminary evidence suggests that pain may heighten risk for PTSS development after injury.^{19,21} For example, Saxe et al.²² found that among acutely burned children, pain during hospitalization indirectly influenced PTSS three months later via a pathway mediated by separation anxiety. Similarly, findings from a prospective, longitudinal investigation conducted by Hildenbrand et al.²¹ indicated that acute pain predicted PTSS six months post-injury, even after controlling for other risk factors. Evidence from studies with adults following traffic-related and burn injuries likewise suggests that pain is associated with PTSS.^{23–25}

Fear conditioning models provide a useful framework to understand how acute pain may play a role in the development of PTSS following injury. These models propose that extremely stressful events trigger the “fight or flight” response by activating the hypothalamic-pituitary-adrenal (HPA) axis and noradrenergic systems.²⁶ In turn, this physiological response leads to overstimulation of endogenous stress hormones and neuromodulators, resulting in over-consolidated traumatic memories. These memories subsequently manifest as intrusive recollections and conditioned physiological and emotional responses, the distinctive characteristics of PTSD.²⁷ The neurobiological processes underlying fear conditioning have been demonstrated in both animal and human models.²⁶ Pain during or after injury may enhance HPA and noradrenergic activation,

thereby increasing risk for deeply engraved traumatic memories and rapidly acquired learned associations that are difficult to extinguish.^{23,28,29} Memories related to painful events are strongly encoded and easily retrievable.³⁰

Several naturalistic studies of pharmacological pain management in children^{31–35} and adults^{36–39} provide further support for the role of pain in enhancing fear conditioning after injury. Findings from these preliminary investigations suggest that higher dosage of morphine post-injury is associated with reduced risk for PTSD diagnosis or greater reductions in symptoms over time. In addition to diminishing pain itself, morphine may have a direct inhibitory effect on neurophysiological systems involved in PTSS development.^{31–34} Specifically, morphine may attenuate noradrenergic activation associated with enhanced memory consolidation.⁴⁰ This hypothesis is consistent with findings from animal research, which suggest that morphine blocks fear conditioning processes.⁴¹ However, research on pharmacological secondary prevention of PTSD is in its infancy,^{42–44} and the relationships between morphine and other opioids and PTSS following injury warrant additional investigation in more robust samples of children.

Previous research has advanced our understanding of biopsychosocial factors impacting adjustment and recovery in pediatric injury, yet much remains unknown regarding the complex interactions between acute pain, opioid medications, and PTSS during the peri-trauma period. Most research conducted to date has employed adult and small pediatric samples primarily comprised of individuals with burn injuries. Furthermore, prior studies have largely focused on comorbidity of PTSD and chronic pain, rather than the role of acute pain and pharmacological intervention on subsequent development of persistent PTSS. Thus, the current study aimed to investigate the relationships among acute pain, opioid medications, and PTSS after pediatric injury utilizing a prospective longitudinal design. We hypothesized that: (1) acute pain during hospitalization would predict PTSS assessed 12 weeks later, when controlling for other empirically-based risk factors (e.g., prior trauma history, heart rate, early PTSS, traumatic appraisals); and, (2) acute opioid medication administration would mediate the relationship between pain and subsequent PTSS in children who have sustained injuries.

Materials and Methods

Procedures

This study utilized data collected as part of a prospective, longitudinal study of parent-child interactions and risk factors for PTSD after pediatric injury.⁴⁵ Participants were recruited while receiving inpatient treatment for an unintentional injury at a large, Level I pediatric hospital in the northeastern region of the United States. After obtaining parent consent and child assent, children completed a brief validated screener derived from the Acute Stress Checklist for Children.⁴⁶ Those who indicated perceptions of the injury event as potentially traumatic on the screener were then eligible for the full study and immediately participated in baseline interviews. Baseline assessments (T1) were conducted in the hospital within two weeks of injury ($M = 3.01$, $SD = 2.96$) and included measures of trauma history and PTSS. Approximately 12 weeks later (T2; $M = 94.84$, $SD = 16.25$), children and parents participated in phone follow-up interviews to assess PTSS. Pain ratings, heart rate data, and

opioid medication administration over the course of hospitalization were obtained via electronic chart review. Medical record abstraction was conducted by trained research assistants and reviewed for accuracy by the lead author. This study was approved by the hospital's Institutional Review Board.

Participants

Participants included 96 children ages 8 – 13 years ($M = 10.60$, $SD = 1.71$) who had sustained an injury requiring inpatient medical treatment. Eligibility criteria included children who incurred a physical injury within the past two weeks, were between 8 and 13 years of age at recruitment, had adequate proficiency in the English language to complete questionnaires, and perceived their injury event as potentially traumatic. Children were excluded from participating in the study if their current medical status or cognitive functioning precluded completion of assessments, Glasgow Coma Score < 14 , injury was a result of family violence or suspected child maltreatment, or no parent was available to consent. Nearly two-thirds of children (65%) were male and most identified as White (52%) or Black (41%). Most injuries were sustained during recreational activities (57%; e.g., fall from bicycle, all-terrain vehicle [ATV], trampoline, skateboard, playground equipment, horse), organized sports (17%; e.g., football, baseball, basketball), and motor vehicle crashes (16%). Most participating parents (81%) were mothers. Follow-up assessments were completed by 75 children (78% retained to follow-up). See Table 1 for full participant demographics.

This study employed convenience sampling; of those assessed for study eligibility, approximately one-third (30%) declined participation for various reasons (e.g., disinterested, anticipated hospital discharge, child fatigue or feeling unwell) and half (52%) of potentially eligible participants were missed by the study team. There were no significant differences in child sex, race, or age between those who enrolled in the study and those who were eligible but were missed by the research team. Children who chose to participate and those who declined study participation did not differ with regards to age or sex. However, a higher subset of children who identified as White declined participation relative to those who identified as Black (71% vs 52%; $\chi^2(1) = 9.2$, $p < .01$). There were no significant differences with regard to age, sex, or race for participants who completed the T2 follow-up assessment versus those that only completed the baseline assessment.

Measures

Pain—Chart reviews were used to obtain children's pain ratings collected as part of clinical care over the course of their hospitalization. As part of standard care, pain was assessed by nurses using a 0-10 numeric rating system, with a score of 0 corresponding to "no pain" and 10 corresponding to "worst pain imaginable". Research suggests that among children with acute and postoperative pain, this numeric rating scale is a valid and reliable measure of pain intensity.^{47,48} This measure is highly correlated with visual analogue scales and clinically relevant outcomes (e.g., perceived need for medication).^{47,48} In this study, children's most severe rating recorded in the medical chart was used to represent the worst pain children experienced during their hospitalization.

Opioid medications—Chart reviews were used to obtain information on pain medications administered to children. All opioids administered during each child's entire hospitalization were recorded. Similar to the procedures described by Saxe and colleagues,³¹ all doses for opioid medications were summed and divided by the child's weight and number of days hospitalized to create a mean dosage (mg/kg/day) value. Given the various forms of opioid medications (e.g., morphine, meperidine, oxycodone) and routes of administration (e.g., oral, intravenous), equivalency doses were calculated using a standard pharmacology protocol.⁴⁹ In this study, we examined both mean dosage of all opioids combined and mean dosage of morphine alone, as prior investigations have focused on morphine in particular^{31–34} or opioids broadly.³⁵

Child PTSS—The Child PTSD Symptom Scale (CPSS)⁵⁰ is a 24-item self-report instrument assessing PTSD symptoms. The CPSS yields both a continuous severity score and a determination of likely PTSD diagnostic status. Seventeen items correspond to DSM-IV symptom criteria, and seven items assess impairment from these symptoms. The CPSS has demonstrated excellent internal consistency, test-retest reliability, and convergent validity with structured clinical interviews for PTSD.⁵⁰ In this sample, internal consistency for total symptom severity and subscale scores were acceptable across all study time points: total ($\alpha = .85 - .92$), re-experiencing ($\alpha = .71 - .87$), avoidance ($\alpha = .65 - .83$), and arousal ($\alpha = .64 - .79$). Given limitations of PTSD diagnostic criteria for children^{51,52} and research indicating that sub-syndromal PTSS (i.e., significant yet not meeting full diagnostic criteria) are related to negative outcomes in children,^{10,53} this study utilized the CPSS symptom severity score in analyses rather than PTSD diagnostic status.

Traumatic Appraisals of Injury Event—A 4-item screener derived from the Acute Stress Checklist for Children (ASC-Kids)⁴⁶ was used to determine whether children appraised the injury event as potentially traumatic. Items are rated on a 3-point scale (0 = never/not true, 1 = sometimes/somewhat true, 2 = often/very true). Children were eligible to participate in this study if at least one item was rated as "often/very true".^{46,54} In current analyses, the total summed score from this screener was used as a measure of trauma-specific appraisals of the injury event (i.e., the extent to which children perceived the injury event as traumatic).

Prior Trauma History—The UCLA PTSD Index⁵⁵ is a 12-item assessment of exposure to a wide range of traumatic events (e.g., natural disaster, war, violence, medical treatment). In this study, we utilized a dichotomous variable indicating presence versus absence of any prior trauma.

Heart Rate—Heart rate was assessed as part of clinical care and obtained via chart review. For this study, we utilized the first available heart rate, which varied by course of medical care (e.g., assessed by emergency medical services, in the Emergency Department, or upon admission to the hospital). Prior research has indicated that relative to other heart rate measurements, first available heart rate is the most robust predictor of later PTSS.^{56,57} Raw heart rate values were standardized (i.e., converted to Z-scores) based on sex and age-specific reference values obtained from a sample of healthy children.^{58,59}

Data Analysis

Analyses examining descriptive data and bivariate relationships between key study variables were conducted using SPSS statistical software, version 24. Subsequently, we employed structural equation modeling (SEM) using Mplus⁶⁰ to evaluate study hypotheses. Missing data were managed using a Full Information Maximum Likelihood approach within SEM. Application of SEM allowed for modeling with both latent and manifest variables.

To test Hypothesis 1 (i.e., acute pain during hospitalization will predict T2 PTSS), we included pain (manifest variable using 0-10 numeric rating system) as well as T1 and T2 PTSS (latent variables with CPSS subscale scores as indicators). Latent variables were identified with a fixed factor loading of one for the first indicator. Correlated uniqueness was added across time points for the indicators of PTSS latent variables. Subsequently, to test Hypothesis 2 (i.e., opioids would mediate the relationship between acute pain and T2 PTSS), we examined two additional models in which we added morphine and overall opioid medications (manifest variable using morphine or overall opioid mean dosage [mg/kg/day] administered during hospitalization) as mediators to the model described above. In each model, child age, sex, traumatic appraisals of injury event, prior trauma history, and heart rate (manifest variables) were included as covariates.

Overall model fit was evaluated using three indices: the chi-square statistic, the comparative fit index (CFI), and the root mean square error of approximation (RMSEA). Values of chi-square increase with decreasing fit between a model and empirical data; a non-significant chi-square is desirable. The CFI is a widely used relative fit index comparing the fit of an independence model (in which all variables are unrelated) to the fit of the substantive model. CFI values of .95 or greater are desirable and indicate adequate fit to data.⁶¹ The RMSEA provides a measure of absolute fit between the observed and model-implied covariance matrices. RMSEA values below .05 are desired to indicate close fit of a proposed model to data.⁶¹ Hypothesis 2 was tested using bootstrapping procedures; confidence intervals of indirect effects were calculated using the bias-corrected bootstrap,^{62,63} as implemented in Mplus.

Results

Among this sample of children who sustained unintentional injuries, worst pain by child report on a numeric pain rating scale ranged from 0 – 10 ($M = 7.52$, $SD = 2.44$). Dosage of opioid medications administered during hospitalization ranged from 0 – 55.78 mg/kg/day ($M = 1.59$, $SD = 6.49$). Children received 0 – .24 mg/kg/day of morphine during hospitalization ($M = .05$, $SD = .05$). CPSS total scores ranged widely from 0 – 39 ($M = 15.96$, $SD = 9.30$) at T1 and 0 – 48 ($M = 13.35$, $SD = 12.02$) at T2. See Table 2 for a summary of bivariate relationships between primary study variables.

Structural Models

Each of the hypothesized models provided good fit to the data (see Figures 1–3 for model fit statistics and standardized effects). All factor loadings for indicators of latent variables were statistically significant (all p -values < .001), with standardized estimates .71. Consistent

with Hypothesis 1, analyses revealed that worst pain rating was positively associated with concurrent (T1) and later PTSS (T2) when holding evidence-based risk factors constant (see Figure 1). In this model, prior trauma history ($\beta = .22$; $p = .03$) and traumatic appraisals of the injury event ($\beta = .25$; $p = .01$) also emerged as significant predictors of PTSS at T1 but not at T2.

With regards to Hypothesis 2, structural equation modeling indicated that neither opioid medications overall nor morphine specifically (mg/kg/day) administered during hospitalization mediated the relationship between pain and T2 PTSS, controlling for evidence-based risk factors (see Figures 2–3). The bootstrapped estimate of the indirect effect was .006 ($p = .78$, 95% *C.I.* = $-.04 - .05$) for all opioids combined and $-.007$ ($p = .85$, 95% *C.I.* = $-.09 - .07$) for morphine.

Discussion

Findings from this study contribute to the growing literature on the intersection between acute pain and posttraumatic stress in children. Relative to most prior studies, this investigation is unique in that it employed a prospective longitudinal design, involved a larger sample of youth with more diverse injuries, examined pain assessments administered via routine clinical care (vs. research protocol), and investigated the impact of both morphine specifically and opioids in general on later PTSS. Results suggest that the most severe pain endorsed by children during hospitalization for unintentional injury predicts concurrent and later PTSS, even while controlling for the contribution of other risk factors. Mean dosage of morphine and other opioids during hospitalization did not mediate the relationship between pain and PTSS in this sample. Findings suggest that pain assessment may be a useful addition to pediatric PTSS screening tools and highlight the need for additional research on pharmacological secondary prevention approaches.

Consistent with prior studies in children^{19,21,22} and adults after injuries,^{23–25} findings from this investigation suggest an association between high levels of acute pain and subsequent PTSS. Several theoretical frameworks have been proposed to explain the mechanisms underlying this relationship. As applied to fear conditioning models of PTSD, peri-trauma pain may contribute to physiological stress responses that fuel over-consolidated trauma memories and enduring fear conditioned responses.^{23,28,29} It has been suggested that, in addition to acting on pain itself, morphine and other opioids inhibit neurobiological systems involved in fear conditioning.^{40,41,64} Several observational studies provide preliminary support for this model in children with burns and other injuries.^{31–35} However, in the current study, opioids did not mediate the relationship between pain and PTSS. Moreover, children who received larger doses of opioid analgesics did not demonstrate reduced risk for persistent PTSS. Methodological factors may help to explain these divergent findings. Whereas prior studies have primarily focused on burn patients, this sample was comprised of children who sustained a variety of injuries. Given that burn injuries tend to be extremely painful, children with burns may receive higher doses of opioids than those with non-burn injuries. Indeed, relative to pediatric burn samples used in previous investigations, children in this study received far lower mean doses of morphine (0.05 mg/kg/day versus 0.41 mg/kg/day)³¹ and of opioid drugs more broadly (1.59 mg/kg/day versus 5.00 mg/kg/day).³⁵ It may

be that the effects of opioids on fear conditioning processes are only observed at higher, standard therapeutic doses. Clearly, further research is needed to more thoroughly examine the role of fear conditioning processes and the efficacy of opioids as secondary prevention approaches for PTSS after pediatric injury.

Beyond fear conditioning models, evidence also suggests that pain and PTSS may be mutually maintaining.^{65,66} In other words, pain and traumatic stress may perpetuate each other simultaneously through shared maintenance factors (e.g., anxiety sensitivity, cognitive biases, hyperarousal, avoidance). For instance, pain catastrophizing (i.e., negative interpretations of painful bodily sensations)^{67–69} has been linked to worsened pain intensity and PTSS following whiplash and musculoskeletal injuries in adults.^{70–73} This cognitive bias appears to emerge during childhood and is generally stable across painful situations.⁶⁸ As such, children who tend to appraise aversive internal experiences in a catastrophic manner may endorse both heightened pain and PTSS after an injury. In addition, evidence suggests that hyperarousal after an injury (e.g., muscle tension, insomnia, exaggerated startle response) may maintain both pain and PTSS.⁷⁴ However, the mutual maintenance model emphasizes factors contributing to the comorbidity of chronic pain and PTSD in adults.^{65,66} Less is known regarding its applicability to pediatric acute pain and PTSS, though preliminary evidence suggests that the mutual maintenance model is relevant following traumatic brain injury in youth.⁷⁵ Additional research examining biopsychosocial mechanisms accounting for the association between pain and PTSS after pediatric injury is warranted.

Limitations and Future Directions

Results of this investigation should be evaluated with consideration of its limitations. This study used an observational prospective design, and thus opioid dosing was not experimentally manipulated. Moreover, we did not assess the associations between non-opioid drugs, pain, and subsequent PTSS. Non-steroidal anti-inflammatory drugs (NSAIDs) have shown efficacy in treating pediatric fracture pain,⁷⁶ and future studies should assess the impact of non-opioid medications on children's pain and PTSS following injury. With the exception of pharmacological pain management interventions administered during hospitalization, this study did not explore underlying mechanisms of action for the association between acute pain post-injury and subsequent PTSS. Future research should test other physiological, psychological, and social/environmental pathways between peri-trauma pain and persistent PTSS. In addition, studies should seek to utilize multidimensional pain assessments (e.g., measures of pain quality and functional interference), longer follow-up periods, and clinician-administered diagnostic interviews to more comprehensively assess PTSS. Given that this investigation focused on hospitalized patients, additional research is needed to examine whether these findings generalize to children with injuries discharged after treatment in the emergency department. Better understanding the multifaceted relationship between acute pain and PTSS may improve screening, prevention, and treatment approaches to enhance recovery after pediatric injury.

Clinical Implications

Findings from this study indicate that the highest pain reported during hospitalization predicted persistent PTSS, above and beyond the influence of other risk factors. These results highlight the potential utility of incorporating pain assessment into screening for current distress and future risk, a key component of trauma-informed pediatric medical care.⁷⁷ Pain is routinely evaluated across levels of care (e.g., emergency, inpatient, outpatient, primary care) and by various providers (e.g., nurses, physicians, medical and physician assistants, physical therapists), rendering pain assessment a practical approach to identifying youth who may benefit from more targeted assessment or continued monitoring for PTSS. In particular, emergency medicine clinicians are among the first to assess children's injury pain and emotional distress and thereby play an important role in early identification of youth and families who may benefit from psychosocial services (e.g., child life, social work, psychology) during medical care. Clinicians may also consider developing standard PTSS screening protocols involving multidimensional pain assessment at various time points across acute injury care, as both the injury and medical care (e.g., diagnostic procedures, treatments) can be potentially painful and traumatic events for children.¹⁵ In turn, more effective peri-trauma screening could help direct the allocation of limited psychosocial resources and supportive services to children with the highest levels of current and potential future need.

In addition, results from this investigation suggest the importance of effective pain management post-injury to reduce risk for the development or maintenance of PTSS. Emergency clinicians can promote positive post-injury emotional recovery by minimizing painful and frightening aspects of emergency medical care as well as by providing basic anticipatory guidance and education about injury-related psychological reactions (see www.HealthCareToolbox.org for evidence-based assessment and educational resources for patients and families). However, relative to other pharmacological prevention approaches for PTSD (e.g., hydrocortisone, propranolol, SSRIs, benzodiazepines), very few studies and no randomized controlled trials have evaluated opioids.⁴³ If preventive benefits of opioids are substantiated in future research, clinicians will need to carefully balance these with potential risks (i.e., opioid-related adverse events and side effects). Given that inadequate pain control and persistent PTSS each hinder recovery and long-term functioning, better understanding interactions between acute pain and PTSS after injury is essential for improving screening, prevention, and early intervention efforts.

Acknowledgments

Source of Funding: This work was supported by a Mentored Career Award grant 1K23MH093618-01A1 from NIMH.

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Figure 1. Model Results for Associations between Pain and PTSS Over Time

Fit indices: $\chi^2[29, N = 96] = 33.86, p = .24$; CFI = .98; RMSEA = .04

PTSS = posttraumatic stress symptoms; T1 = baseline assessment; T2 = 12 week follow-up assessment

* .05, ** .01, *** .001

Not pictured: covariate relationships (child age, sex, trauma history, heart rate, and traumatic appraisals of injury event).

Figure 2. Model Results for Associations between Pain, Opioids, and PTSS Over Time

Fit indices: $\chi^2[34, N = 96] = 35.45, p = .40$; CFI = 1.00; RMSEA = .02

PTSS = posttraumatic stress symptoms; T1 = baseline assessment; T2 = 12 week follow-up assessment

* .05, ** .01, *** .001

Not pictured: covariate relationships (child age, sex, trauma history, heart rate, and traumatic appraisals of injury event).

Figure 3. Model Results for Associations between Pain, Morphine, and PTSS Over Time

Fit indices: $\chi^2[34, N = 96] = 37.36, p = .32$; CFI = .99; RMSEA = .03

PTSS = posttraumatic stress symptoms; T1 = baseline assessment; T2 = 12 week follow-up assessment

* .05, ** .01, *** .001

Not pictured: covariate relationships (child age, sex, trauma history, heart rate, and traumatic appraisals of injury event).

Table 1

Participant Demographics

CHILDREN	N (%)
Gender	
Male	62 (65)
Female	34 (35)
Racial/Ethnic Background	
White	50 (52)
Black/African American	39 (41)
Hispanic	4 (4)
Other	7 (3)
Injury Mechanisms	
Recreational Activities	55 (57)
Organized Sports	16 (17)
Motor Vehicle Crash	15 (16)
Gunshot	2 (2)
Dog Bite	5 (5)
Burn	3 (3)
Injury Type	
Fracture(s)	60 (63)
Other	36 (38)
<i>M (SD)</i>	
Age (years)	10.60 (1.71)
Length of hospital stay (days)	2.76 (3.32)
Injury Severity Score	5.82 (4.40)
CAREGIVERS	N (%)
Relationship to child	
Mother	78 (81)
Father	18 (19)
Highest Education	
High School	19 (20)
Some College	29 (31)
College Degree	28 (30)
Graduate/Professional Degree	18 (19)
Household Income	
< \$30,000	25 (26)
\$30,000 – \$74,999	29 (31)
\$75,000	41 (43)
<i>M (SD)</i>	

CHILDREN	N (%)
Age (years)	40.72 (6.75)

Note: Participant demographics as reported at baseline enrollment.

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Table 2

Correlations among Primary Study Variables

	1	2	3	4	5	6	7	8	9
1 Age									
2 Sex	-.25*								
3 Heart Rate	-.10	.11							
4 Prior Trauma History	.11	-.23*	.05						
5 Traumatic Appraisals	-.05	.04	.06	.03					
6 Pain	.13	.11	.32**	.01	.13				
7 Opioids (Combined)	-.17	-.04	-.23*	.01	-.002	-.03			
8 Morphine	.11	.03	.15	-.14	-.06	.27**	.06		
9 T1 PTSS	-.01	-.15	.17	.24*	.27**	.26*	.08	.11	
10 T2 PTSS	.03	-.03	.01	.14	.27*	.34**	-.14	.04	.46***

*
p < .05**
p < .01***
p < .001

Note: T1 = baseline assessment; T2 = 12 week follow-up assessment; PTSS = posttraumatic stress symptoms