Acute Pain and Posttraumatic Stress after Pediatric Injury

A Thesis
Submitted to the Faculty
of
Drexel University
by
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in partial fulfillment of the
requirements for the degree
of
Master of Science in Clinical Psychology
July 2014
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Acknowledgements

I am sincerely grateful for the support and guidance of my faculty advisors, Drs. Brian Daly and Douglas Chute, and mentors at the Children’s Hospital of Philadelphia, Drs. Meghan Marsac and Nancy Kassam-Adams, in designing and conducting this project. I would also like to express my deepest gratitude towards those involved in the research studies from which the current project was developed, including Dr. Flaura Winston, Dr. Michael Nance, Kristen Kohser, and Grace Good. Lastly, I would like to thank my lab members at Drexel and CHOP for their support, as well as the children and families who participated.
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Abstract

Acute Pain and Posttraumatic Stress after Pediatric Injury
Aimee K. Hildenbrand

**Background:** Unintentional injury is a leading health concern for children across the globe (Peden, 2008). In the United States alone, 20 million children suffer injuries each year (Borse et al., 2009). After an injury, many children experience persistent posttraumatic stress symptoms (PTSS) that negatively impact functioning and recovery (Balluffi et al., 2004; Daviss et al., 2000; DeVries et al., 1999; Holbrook et al., 2005; Kazak et al., 2001; Kean, Kelsay, Wamboldt, & Wamboldt, 2006; Mintzer et al., 2005; Rennick et al., 2004; Stoddard & Saxe, 2001; Winston, Kassam-Adams, Garcia-Espana, Ittenbach, & Cnaan, 2003). Although prior research on risk factors for PTSS has advanced our understanding of emotional recovery after pediatric injury, most investigations have focused on processes in the post-trauma period. Acute pain during the peri-trauma phase represents a promising avenue for identifying children who may be at risk for developing PTSS. Acute pain and PTSS share neurobiological pathways and commonly present after pediatric injury, yet their interactions are poorly understood given the paucity of research in this area (Gold, Kant, & Kim, 2008; Langeland & Olff, 2008). Investigations examining the association between pain and posttraumatic stress have largely focused on adults or small samples primarily composed of pediatric burn patients. As such, much remains unknown regarding the complex interactions between acute pain and PTSS among children who have sustained injuries.
**Aims:** This research aims to add to our understanding of the development of PTSS and contribute to more effective screening and prevention approaches for children who have experienced medical trauma. Specifically, the current study examined the relationship between acute pain and PTSS and investigated whether pain medications conferred a protective effect against PTSS.

**Methods:** This study utilized data collected as part of two large, prospective longitudinal studies of children following injury (Kassam-Adams et al., 2011; Kassam-Adams & Winston, 2004). In Study 1, children ages 8 – 17 years who had sustained an injury ($N = 243$) and their parents participated in baseline interviews to assess children’s current and worst pain since the injury. Approximately six months later, children and parents completed follow-up interviews to assess child PTSS. In Study 2, children hospitalized for unintentional injury ($N = 292$) and their parents completed baseline assessments of traumatic stress symptoms. Approximately six weeks and six months later, follow-up assessments were conducted to assess PTSS. Children’s pain ratings and opiate medications administered during hospitalization were obtained via chart reviews.

**Results:** Worst pain as assessed by the Color Analogue Pain Scale predicted child PTSS six months post-injury, even when controlling for demographic and empirically-based risk factors (e.g., heart rate, prior trauma history, acute stress symptoms). In contrast, pain as assessed by the Faces Pain Rating Scale and a numeric 0-10 rating system did not emerge as significant independent predictors of persistent PTSS. Opiate medication use during hospitalization did not moderate the relationship between acute pain and PTSS six weeks or six months following pediatric injury.
**Conclusions:** The Color Analogue Pain Scale may be a useful addition to existing screening tools for PTSS among children. Additional research is needed to examine differences between pain assessment tools as well as the impact of opiate medication use during hospitalization with regards to the development of PTSS. Further research is also warranted to better understand underlying mechanisms linking acute pain and subsequent PTSS in order to improve assessment, prevention, and treatment approaches and promote optimal recovery to pediatric injury.
Introduction

Unintentional injury is unfortunately an extremely common experience and a leading cause of disability for children across the globe (Peden, 2008). Each year, 20 million children in the United States alone suffer injuries, with nearly 9 million of these children requiring emergency medical care (Borse et al., 2009). For U.S. children older than one year of age, injury is the leading cause of morbidity and mortality (Peden, 2008). In fact, it is estimated that an average of 890 years of potential life per 100,000 children are lost each year due to unintentional injuries (Borse, Rudd, Dellinger, & Sleet, 2013). Common mechanisms of pediatric injury include falls, motor vehicle traffic (e.g., occupant, pedestrian), drowning, and fires/burns (Borse et al., 2009). Given the high prevalence of pediatric injury both domestically and globally, understanding children’s recovery after injury has clear public health significance. In particular, identifying potential risk factors for poor psychosocial outcomes post-injury could inform screening and secondary prevention approaches, thereby facilitating healthy recovery for children with injuries.

Posttraumatic Stress Symptoms (PTSS)

While most children who sustain injuries recover physically, a significant subset experience negative psychological consequences (e.g., new fears, depression) that significantly impact functioning (Basson et al., 1991; DeVries et al., 1999; Di Gallo, Barton, & Parry-Jones, 1997). In particular, a considerable proportion of injured children and their parents report persistent posttraumatic stress symptoms (PTSS; Daviss et al., 2000; DeVries et al., 1999; Holbrook et al., 2005; Stoddard & Saxe, 2001; Winston et al., 2003). According to the National Child Traumatic Stress Network (2003), pediatric
medical 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.” More specifically, PTSS include re-experiencing or intrusion (i.e., recurrent distressing memories), avoidance of trauma reminders, and hyperarousal (e.g., exaggerated startle, sleep disturbance), as well as dissociation (e.g., altered sense of reality as dreamlike or surreal) and a myriad of negative moods or cognitions (e.g., feelings of detachment, blaming others, diminished interest in activities; American Psychiatric Association, 2013). In order to meet diagnostic criteria for PTSD according to the DSM-5 (American Psychiatric Association, 2013), individuals must demonstrate at least one symptom of intrusion, one avoidance symptom, three changes in cognition or mood, and three alterations in arousal (Bryant, Friedman, Spiegel, Ursano, & Strain, 2011; Friedman, Resick, Bryant, & Brewin, 2011). Although the DSM-5 indicates lower symptom thresholds for children under the age of six, these adjustments may not sufficiently address the nature of PTSS in school-age children (Kassam-Adams, Marsac, & Cirilli, 2010; Scheeringa, Zeanah, & Cohen, 2011). Furthermore, research suggests that even sub-syndromal PTSS (i.e., significant yet not meeting full diagnostic criteria) are related to negative outcomes in children (Holbrook et al., 2005; Stoddard & Saxe, 2001). Given the limitations of PTSD diagnostic criteria for children, investigations of traumatic stress among youth should examine the continuum of PTSS severity rather than solely PTSD diagnostic status.

After an injury, many children and their parents experience distressing PTSS. A meta-analysis of medical traumatic stress studies found that, on average, 19% of children develop significant PTSS following injury (Kahana, Feeny, Youngstrom, & Drotar,
One year after an injury, approximately 10-30% of injured children still experience distressing PTSD symptoms (Daviss et al., 2000; DeVries et al., 1999; Holbrook et al., 2005; Stoddard & Saxe, 2001; Winston et al., 2003). Research suggests that PTSS can occur regardless of injury severity (Alisic, Jongmans, van Wesel, & Kleber, 2011; Cox, Kenardy, & Hendrikz, 2008) or mechanism, with impairing symptoms documented after violent injury (Pailler, Kassam-Adams, Datner, & Fein, 2007), road traffic injuries (Kassam-Adams, Fleisher, & Winston, 2009; Kassam-Adams & Winston, 2004; Meiser-Stedman, Smith, Glucksman, Yule, & Dalgleish, 2008), burns (De Young, Kenardy, Cobham, & Kimble, 2012; Hall et al., 2006; Saxe et al., 2005; Stoddard et al., 2006), and animal bites (Ji, Xiaowei, Chuanlin, & Wei, 2010).

Psychological trauma symptoms are problematic in that they can be extremely distressing for children and parents and can significantly hinder recovery after an injury. In particular, PTSS are strong predictors of impaired functioning and health-related quality of life (HRQOL), interfere with adherence to medical recommendations, and are linked to poorer health and greater use of healthcare services (Graham-Bermann & Seng, 2005; Holbrook et al., 2005; Landolt, Buehlmann, Maag, & Schiestl, 2009; Landolt, Vollrath, Gnehm, & Sennhauser, 2009; Seng, Graham-Bermann, Clark, McCarthy, & Ronis, 2005; Thorp & Stein, 2005; Vanderbilt et al., 2008; Zatzick et al., 2008). For instance, several studies suggest that PTSS are associated with worse functional recovery (i.e., mobility, physical and social activity) among youth during the years following an injury (Holbrook et al., 2005; Zatzick et al., 2008). As a result, PTSS pose a major threat to children’s physical and emotional health and recovery after injury.
Risk Factors for PTSS

Given the substantial impact of PTSS on children’s recovery following injury, a growing body of research has examined potential risk factors in order to better target preventive interventions. This research suggests that various biological, psychological, and social-environmental factors may place children at risk for developing PTSS post-injury (Alisic et al., 2011; Cox et al., 2008; Kahana et al., 2006; Trickey, Siddaway, Meiser-Stedman, Serpell, & Field, 2012). These biopsychosocial etiological factors may operate prior to the injury (i.e., pre-trauma), during the injury event (i.e., peri-trauma), and/or after the injury (i.e., post-trauma). Although pre-trauma variables may not be malleable, they can serve as important indicators and could be included in screening inventories to identify those who may be at risk for negative outcomes. Some peri- and post-trauma etiological factors can be targets of intervention to promote recovery and prevent the development of adverse psychological reactions such as PTSS.

Biological Factors

Biological risk factors for PTSS in children include intense acute physiological responses such as high cortisol levels and heart rate. For instance, Ostrowski and colleagues (2007) found that at both six weeks and seven months following pediatric injury, higher levels of cortisol predicted PTSS. Interestingly, this association was found for boys only, though prior research has suggested that girls may be at higher risk for developing PTSS after injury and other traumatic events (Alisic et al., 2011; Cox et al., 2008). Other studies examining the biopsychology of PTSD in children support these findings. For example, children diagnosed with PTSD following maltreatment (i.e., emotional, physical, or sexual abuse), separation or loss, or violence exposure
demonstrate higher cortisol levels when compared to healthy controls without PTSD (Carrion et al., 2002; De Bellis et al., 1999). Furthermore, Delahanty and colleagues (2003) found that cortisol levels collected upon hospital admission mediated the relationship between injury severity and PTSS as well as trauma history and PTSS following pediatric motor-vehicle related injury. The authors suggest that cortisol levels post-trauma may represent a mechanism by which other factors can increase risk for PTSS among youth.

Heart rate (HR) in the early period post-injury may also be indicative of risk for PTSS among children. Several studies suggest that HR during hospitalization for pediatric injury predicts PTSS while controlling for age, gender, and injury severity (Bryant, Salmon, Sinclair, & Davidson, 2007a; De Young, Kenardy, & Spence, 2007; Kassam-Adams, Garcia-Espaňa, Fein, & Winston, 2005; Nugent, Christopher, & Delahanty, 2006). For example, one study found that earliest available HR predicted PTSS six weeks and six months post-injury, even when holding gender, parent income, and depression constant (Nugent et al., 2006). Similarly, Bryant and colleagues (2007a) found that children with elevated HR (i.e., 1.5 SD above norms for gender and age) were more likely to develop full or partial PTSD than children without elevated HR when controlling for age, sex, injury severity, and length of hospital stay. Additional research is needed to better understand the mechanisms by which these biophysiological factors can increase risk for PTSS among children following injury. Likewise, further research is warranted to explore other potential biopsychological systems or pathways (e.g., acute pain) that may be implicated in the development of PTSS.
Psychological Factors

Research suggests that certain psychological processes may play an important role in the development of PTSS. Not surprisingly, pre-existing psychopathology and prior exposure to trauma have been associated with PTSS in children after injury (Alisic et al., 2011; Cox et al., 2008; Le Brocq, Hendrikz, & Kenardy, 2010; Trickey et al., 2012). Additionally, early traumatic stress symptoms predict later PTSS in children who have sustained injuries (Kassam-Adams & Winston, 2004; Ostrowski et al., 2011; Vernberg, LaGreca, Silverman, & Prinstein, 1996). Studies also indicate that the way in which children subjectively interpret the injury (i.e., cognitive appraisal) may influence risk of PTSS. For instance, perceived life threat, loss of control during the injury, self-vulnerability to future harm, and negative interpretations of trauma symptoms are predictive of worse PTSS in children (Aaron, Zaglul, & Emery, 1999; Bryant, Salmon, Sinclair, & Davidson, 2007b; Ehlers, Mayou, & Bryant, 2003; Holbrook et al., 2005; McDermott & Cvitanovich, 2000; Stallard & Smith, 2007).

The way in which children cope with an injury may also represent an etiological factor for PTSS. Stallard and colleagues (2007) found that appraisals and coping accounted for a large proportion (64%) of the variance in PTSS eight months after an injury. In particular, research suggests that strategies such as social withdrawal, blaming others, and resignation are related to worse psychological symptoms (Marsac, Donlon, Winston, & Kassam-Adams, 2013; Trickey et al., 2012). On the other hand, the use of social support to cope with an injury and reactions to it may be linked to reduced PTSS among children (Stallard, Velleman, & Baldwin, 2001; Stallard, Velleman, Langsford, & Baldwin, 2001). These findings highlight the importance of examining psychological
processes that may be related to the development of PTSS. In particular, children’s subjective appraisals of physiological symptoms of injury (e.g., acute pain) represent a promising avenue for future empirical investigation.

**Social and Environmental Factors**

Parents play a significant role in children’s emotional recovery after injury and may impact the development of PTSS. A number of studies have revealed a strong relationship between parent and child PTSS (Alisic et al., 2011; Cox et al., 2008; Daviss et al., 2000; Landolt, Vollrath, Laimbacher, Gnehm, & Sennhauser, 2005; Nugent, Ostrowski, Christopher, & Delahanty, 2007; Ostrowski, Christopher, & Delahanty, 2007; Trickey et al., 2012). For instance, a study conducted by Le Brocque and colleagues (2010) indicated that child and parent traumatic stress symptom trajectories were strongly associated. Parent PTSS can impact how caregivers interpret their children’s emotional responses, with parent ratings of child PTSS biased towards their own symptoms (Kassam-Adams, Garcia-Espona, Miller, & Winston, 2006).

Beyond parents’ own PTSS, they may also influence children’s emotional recovery after injury by shaping child appraisals and coping efforts. Research from the child anxiety literature suggests that anxious youth are more likely to have parents who suggest and reward negative appraisals and maladaptive (e.g., avoidant) coping strategies than those without anxiety (Barrett, Rapee, Dadds, & Ryan, 1996; Chorpita, Albano, & Barlow, 1996; Cobham, Dadds, & Spence, 1999; Luis, Varela, & Moore, 2008; Micco & Ehrenreich, 2008). Similarly, the way in which parents help their children cope (i.e., coping assistance) is associated with child coping following injury as well as medical procedures (Blount et al., 1989; Marsac et al., 2013). Finally, low levels of social support
and poor family functioning are also related to increased PTSS in children (Stallard, Velleman, & Baldwin, 2001; Stallard, Velleman, Langsford, et al., 2001). Additional research is warranted to understand the ways in which parents’ own interpretations of children’s emotional and physical symptoms can impact parent coping assistance and child recovery.

**Screening for Risk of PTSS**

Understanding potential risk factors for the development of PTSS is essential for effective screening and early preventative intervention efforts. Although prior research on risk factors for PTSS has clearly advanced our understanding of emotional recovery after pediatric injury, most investigations have focused on processes during the post-trauma period (Marsac et al., under review). Furthermore, a number of barriers interfere with effective screening of children at risk for psychological sequelae during acute trauma care, including limitations in time and training and the challenge of differentiating between normative distress and indicators of risk for PTSS. Additionally, seemingly intuitive indicators such as injury severity have not been shown to be useful predictors of psychological outcomes (Daviss et al., 2000; DeVries et al., 1999). As a result, clinicians’ perceptions of patients at risk for psychological sequelae are often inaccurate (Baxt, Kassam-Adams, Kazak, Balluffi, & Helfaer, 2005; Ziegler, Greenwald, DeGuzman, & Simon, 2005). Hence, there is a need to systemically identify empirically-based indicators of PTSS risk during the peri-trauma period that can be easily integrated into medical care.
**Acute Pain after Pediatric Injury**

Acute pain represents a promising avenue for identifying children who may be at risk for developing PTSS, as pain is widely assessed as part of routine medical care. Most children report significant pain during and after an injury (Hagan et al., 2001). In fact, acute pain resulting from injury is the most common type of pain experienced by children (Hagan et al., 2001). Pain is often defined as an “unpleasant sensory and emotional experience associated with actual or potential tissue damage” (McCulloch & Collins, 2006; World Health Organization, 1995). An inherently subjective experience, pain encompasses physiological, sensory, emotional, cognitive, and behavioral components (Hagan et al., 2001). Pain is also highly contextual in that it varies across sociocultural, environmental, and developmental factors (Hagan et al., 2001). For instance, similar types of injury may be seemingly painless in certain situations yet extremely painful in others (Renn & Dorsey, 2005). Beginning suddenly, acute pain is usually sharp in quality and serves as a warning of disease or threat to the body (Carr & Goudas, 1999; Helms & Barone, 2008). To differentiate it from chronic pain, acute pain is defined as pain that does not persist beyond six months, is focal to the site of injury, and typically resolves when an underlying cause has been treated or remedied (Grichnik & Ferrante, 1991).

An extremely complex phenomenon, processing of acute pain involves both the peripheral (PNS) and central nervous systems (CNS; Renn & Dorsey, 2005). Nociception, the initial processing of a noxious stimulus, occurs by a series of mechanisms that involve transmission of pain signals by specialized nerves (i.e., nociceptors) along an ascending pathway from the point of stimulation to the CNS (Willis & Coggeshall, 1991). Once transmitted to the thalamus, nociceptive information
is encoded and transmitted onward to limbic structures and the cerebral cortex, where
cognitive and emotional interpretation occurs to identify the signals as pain (Julius &
Basbaum, 2001; Melzack & Wall, 1965; Millan, 1999). Subsequently, brainstem
processes are triggered and initiate descending modulatory effects on the nociceptive
signals. This pain modulation circuitry involves several brainstem sites, including the
periaqueductal gray, the locus coeruleus, and the rostral ventromedial medulla (Fields &
Basbaum, 1999; Millan, 1999). The descending modulation either inhibits (i.e., less pain)
or facilitates further nociceptive transmission (i.e., greater pain; Renn & Dorsey, 2005).
The transmission of pain signals can be interrupted by the administration of
pharmacological agents that hinder sensitization of nociceptors (e.g., nonsteroidal anti-
inflammatory drugs [NSAIDs]) or nerve transmission (e.g., lidocaine; Renn & Dorsey,
2005). The perception of pain can also be attenuated by drugs that impact the CNS (e.g.,
opioids; Renn & Dorsey, 2005).

**Acute Pain as a Risk Factor for PTSS**

Although both acute pain and PTSS are common after pediatric injury, their
interactions are poorly understood given the paucity of research in this area (Gold et al.,
2008; Langeland & Olff, 2008). Pain has traditionally been viewed as an outcome of
physical injury, yet preliminary evidence suggests that pain may also be an etiological
factor for the development of PTSS (Gold et al., 2008). For example, Saxe and colleagues
(2005) utilized pathway analyses to develop a model of risk factors for PTSD among
pediatric burn victims. This study found that for acutely burned children, pain during
hospitalization indirectly influenced PTSS measured at three months through a pathway
mediated by separation anxiety. Evidence from studies with adults corroborates these
findings. For example, research examining psychological functioning of adults following whiplash injuries and motor-vehicle crashes (MVCs) has suggested that pain is associated with acute PTSS and the incidence of diagnosable PTSD (Drottning, Staff, Levin, & Malt, 1995; Kuch, Cox, & Evans, 1996). Furthermore, Fedoroff and colleagues (2000) found that decreased pain severity significantly predicted reduced PTSS in adults following MVCs. Likewise, a prospective study of adult burn patients found that pain during hospitalization significantly predicted PTSS at one month, one year, and two year follow-ups and was a stronger predictor of psychological functioning than burn size or length of hospitalization (Patterson, Tininenko, & Ptacek, 2006). However, additional research using larger, more diverse samples of children is needed to verify these preliminary findings and better understand the relationship between pain and pediatric PTSS.

**Theoretical Model: Pain and Fear Conditioning**

Several theories highlight potential mechanisms by which pain can serve as a risk factor for PTSS among children. Based in recent advances from neurobiological research, fear conditioning models suggest that pain may contribute to the development of PTSS by stimulating the release of hormones that enhance fear conditioning and overconsolidation of trauma memories (Amstadter, Nugent, & Koenen, 2009; Pitman, 1989). More specifically, fear conditioning models propose that an extremely stressful event triggers the “fight or flight” stress response by activating the hypothalamic-pituitary-adrenal (HPA) axis and noradrenergic systems (e.g., locus coeruleus; Amstadter et al., 2009). In turn, this physiological response leads to overstimulation of endogenous stress hormones and neuromodulators, resulting in over-consolidated traumatic
memories. This deeply engraved traumatic memory subsequently manifests itself as intrusive recollections and conditioned emotional responses that are extremely difficult to extinguish, the hallmark symptoms of PTSD (Pitman, 1989; Yehuda, McFarlane, & Shalev, 1998). The neurobiologic processes underlying this fear-conditioning model have been well described across both animal models and human research (Amstadter et al., 2009).

Because pain and PTSD have shared neurobiological pathways (Asmundson, Coons, Taylor, & Katz, 2002; McLean, Clauw, Abelson, & Liberzon, 2005; Norman, Stein, Dimsdale, & Hoyt, 2008), it has been suggested that acute pain during or after an injury can enhance HPA and noradrenergic activation and thereby increase risk of over-consolidated trauma memories and the development of PTSS. This model is supported by research indicating that memories of painful events are readily retrievable (Morley, 1993), indicating that strong encoding occurs during painful events such as physical injury. Additionally, initial research examining pharmacological pain management lends further support to this model. A small study of pediatric burn victims ($N = 24$) found that children who received higher doses of morphine during hospitalization had greater reductions in PTSS six months post-injury (Saxe et al., 2001). The authors speculated that morphine attenuates the adrenergic activation associated with enhanced memory consolidation and fear conditioning, thereby reducing the risk of PTSS. These initial findings received partial support in three additional studies with very young (12 – 48 months of age) burn victims (Stoddard et al., 2009) and modest samples of children after injuries ($N = 48$; Nixon et al., 2010) and pediatric burn and non-burn injury patients ($N = 61$; Saxe et al., 2006). These findings are also consistent with animal research, which has
demonstrated that morphine blocks fear conditioning processes (Good & Westbrook, 1995; McNally & Westbrook, 2003).

**Current Study**

Fear conditioning models provide a useful framework to understand how acute pain may play a role in the development of PTSS following injury. A limited number of investigations of acute pain and pharmacological pain management provide initial yet compelling empirical support for these models. However, the complex relationship between acute pain and PTSS has yet to be thoroughly investigated in robust samples of children following injury. Similarly, the utility of opiate medications as a secondary prevention approach for PTSS following pediatric injury warrants additional investigation in more robust samples. The vast majority of extant research in this area has been conducted with adults and small pediatric samples primarily composed of burn patients. Furthermore, prior studies have predominantly focused on the co-occurrence of PTSS and chronic pain, rather than how acute pain may increase risk for the development of PTSD symptomatology. As such, much remains unknown regarding the interactions between acute pain, opiate medications, and PTSS after pediatric injury.

**Aims**

The current study aimed to help fill this gap in the literature by examining the relationship between acute pain and PTSS in two large, prospective longitudinal studies of children following injury. More specifically, this study examined the relationship between pain and PTSS and investigated whether pain medications confer a protective effect against PTSS. This research will add to our understanding of the development of PTSS in children and may stimulate additional research examining the interacting...
influences of pathophysiological and psychological factors post-trauma. In turn, this research could also contribute to more effective screening and prevention approaches for children who have experienced medical trauma.

**Hypotheses**

We hypothesized (1) that acute pain (i.e., worst pain rating during hospitalization) would predict PTSS (at 6 weeks and 6 months) when controlling for demographic factors (i.e., age, sex, ethnicity) and empirically-based risk factors for PTSS (i.e., HR, prior trauma, acute stress disorder (ASD) symptoms, and perceived life threat), and (2) that opiate medications (i.e., mean dosage during hospitalization) would moderate the relationship between acute pain and PTSS (at 6 weeks and 6 months). Specifically, we expected that children who experience more severe levels of acute pain during hospitalization would be more likely to develop PTSS. We also expected that the relationship between acute pain and PTSS would be strongest for children who receive lower doses (mg/kg/day) of opiate medications. In other words, we predicted that the slope of PTSS by acute pain would be steeper for children who receive lower doses of opiate medications.

**Methods**

This study utilized data collected as part of two prospective longitudinal studies conducted at The Children’s Hospital of Philadelphia (CHOP). Study 1 was designed to identify risk factors for PTSD following hospitalization for pediatric injury and to develop a brief PTSD risk assessment tool (Kassam-Adams & Winston, 2004). Study 2 was designed to examine the efficacy of a stepped preventative care intervention for
pediatric injury that involved screening for risk of distress, psychoeducation, and evidence-based psychosocial care (Kassam-Adams et al., 2011).

**Study 1**

**Procedures**

Parents of eligible children enrolled in the Trauma Service at CHOP were informed of the study and invited to participate by a research assistant, trauma fellow, or trauma research nurse. After providing consent and assent, children and parents participated in baseline interviews that included assessment of children’s worst pain since the injury. Pain was assessed using both the Color Analogue Pain Scale (McGrath et al., 1996) and the Faces Pain Rating Scale (Bieri, Reeve, Champion, Addicoat, & Ziegler, 1990), administered in counterbalanced order. Baseline assessments were conducted in either the child’s hospital room or home within four weeks of injury ($M = 11.5$ days, $SD = 10.63$). Approximately six months later, children and parents participated in follow-up interviews conducted in children’s homes. At the six-month follow-up assessment, child PTSS was assessed using the Clinician-Administered PTSD Scale for Children and Adolescents (CAPS-CA; Nader et al., 2004). This study was approved by the hospital’s Institutional Review Board.

**Participants**

Participants included 243 children ages 8 – 17 years ($M = 11.36$, $SD = 2.49$) who had sustained an injury requiring medical treatment and one parent per child. Eligibility criteria included incurring a traffic-related physical injury within the past four weeks, receiving medical care in the hospital, being between 8 and 17 years of age, and having adequate proficiency in the English language to complete questionnaires. Children were
excluded from participating in the study if their current medical status or cognitive functioning precluded the completion of assessments, or no parent was available to consent and participate in the study. A large majority of children (75%) were male. Information obtained from hospital records revealed that most children were Black (56%) or White (40%). Children in this sample sustained traffic-related injuries as pedestrians (33%), bicyclists struck by a vehicle (23%), bicyclists who fell (28%), or motor vehicle occupants (16%). Approximately 42% of children suffered at least one extremity fracture, and 26% of children were admitted to the intensive care unit. Most participating parents (88%) were mothers or other female guardians. Six-month follow-up assessments were completed by 171 children (70% of participants enrolled at baseline). There were no significant differences with regard to sex, age, or ethnicity for those retained to follow-up versus those completing the baseline assessment only. See Table 1 for full participant demographics.

**Measures**

**Pain.** The Color Analogue Pain Scale (CAS; McGrath et al., 1996) is a self-report assessment of pain intensity that utilizes a visual analogue instrument. This instrument is comprised of a 10-cm line with increasingly intense red coloring to indicate pain severity. Children slide a marker over the line to indicate the level of pain they are experiencing. The reverse side of this instrument indicates corresponding numeric values (0 – 10, rounded to the nearest 1/8 unit) for children’s pain ratings. The CAS has been used to assess pain among a wide variety of pediatric populations that endorse pain (Bulloch & Tenenbein, 2002). Scores on this measure are strongly correlated with those from visual
analogue instruments, and research suggests that the CAS is easier to administer than other visual analogue scales (McGrath & Brigham, 1992).

The Faces Pain Rating Scale (FPRS; Bieri et al., 1990) is a self-report measure of pain intensity in which children choose a face that corresponds with their level of pain. The seven faces range from scores of 0 (no pain) to 6 (severe pain). This instrument has been employed in many different clinical settings (Bulloch & Tenenbein, 2002). While scores on the FPRS are correlated with those on traditional visual analogue scales (de Tovar, Wood, & Alibeu, 2002; Hicks, von Baeyer, Spafford, van Korlaar, & Goodenough, 2001), some studies suggest that pediatric patients prefer the FPRS to visual analogue scales in clinical settings (de Tovar, Wood, & Alibeu, 2002).

Furthermore, the FPRS is considered an ideal instrument for assessing pain severity in children across ages, gender, and cultural backgrounds (Belville & Seupaul, 2005). The scale is thought to be superior in design to other faces scales because it does not confound pain intensity with affect (i.e., smiling and crying anchor faces; Belville & Seupaul, 2005).

Research has demonstrated that both the CAS and FPRS have good construct, content, and convergent validity and reliability in measuring acute pain in children (Bulloch & Tenenbein, 2002). In this study, children were asked to use the CAS and the FPRS to retrospectively report the worst or most severe level of pain they experienced since their injury.

Child PTSS. The Clinician-Administered PTSD Scale for Children and Adolescents (CAPS-CA; Nader et al., 2004) is a semi-structured interview that assesses PTSD symptoms and associated features. The CAPS-CA assesses the frequency and
intensity of seventeen symptoms corresponding to the DSM-IV criteria for PTSD. The CAPS-CA also evaluates the impact of these symptoms on youth’s social, occupational, and developmental functioning, subjective distress, and global severity of PTSD. The CAPS-CA is an adaptation of the adult CAPS (Blake et al., 1990), the most widely recommended PTSD interview measure for adults. The measure has good internal consistency for each subscale (alphas ranging from .75 to .81), and reasonable concurrent validity with self-report measures of PTSD (Newman, McMackin, Morrissey, & Erwin, 1997).

**Study 2**

**Procedures**

Children hospitalized for unintentional injury and their parents were informed of the study and invited to participate. After parent consent and child assent was obtained, children completed a brief screening protocol consisting of measures of current traumatic stress symptoms, current depression symptoms, and risk of persistent PTSD to determine eligibility for the randomized controlled trial (RCT). Screening measures were scored immediately, and children who screened positive on at least one measure were considered at risk and were eligible for the randomized trial. An additional sample of children screening as low risk were enrolled for follow-up. Baseline assessments were completed within two weeks of injury ($M = 2.55$ days, $SD = 2.60$). Participants were then randomly assigned to the intervention (i.e., two sessions incorporating assessment and psychoeducation for psychological symptoms) or usual care group (i.e., standard social work services such as assessment, counseling, and community resource planning). Approximately six weeks and six months later, follow-up assessments were conducted by
personnel blind to the child’s study assignment to assess PTSD and depression symptoms, health-related quality of life (HRQOL), and health service utilization. Chart reviews were used to obtain children’s pain ratings and pain medications administered during the entire period of hospitalization. The stepped care intervention did not reduce PTSD or depression severity or increase HRQOL as compared to the usual care group (Kassam-Adams et al., 2011). Given that there were no differences between groups in psychosocial, health, or functional outcomes, we determined that it was appropriate to utilize data from the entire sample for the current study. This study was approved by the hospital’s Institutional Review Board.

**Participants**

Participants included 177 children ages 8 – 17 years ($M = 12.12$, $SD = 2.47$) admitted to the hospital’s Trauma Center for treatment of unintentional injury and their parents. Children and their parents were eligible to participate if they were adequately proficient in English to complete the measures and participate in the interview and if the family had access to a telephone. Exclusion criteria included: (a) child’s medical status or cognitive function precluded participation; (b) moderate or severe head injury, defined as a Glasgow Coma Scale (GCS) score $\leq 12$; (c) child’s injury involved family violence or abuse; and, (d) no parent or guardian available to participate in the study. Nearly three-quarters of the children in this sample (69%) were male. About half of the children in this study identified as White (56%), while 31% were Black/ African American. Mechanisms of injury included organized sports or recreation (56%), falls (22%), MVC (16%), animal bites (4%), and other unintentional injury mechanisms (2%). Approximately 68% of children suffered at least one extremity fracture. Most participating parents (84%) were
mothers. Six-week and six-month follow-up assessments were completed by 141 (80%) and 142 children (80%), respectively. There were no significant differences with regard to sex, age, or ethnicity for those retained to the six-week and six-month follow-ups versus those completing the baseline assessment only. See Table 1 for full participant demographics.

**Measures**

**Pain.** Chart reviews were used to obtain children’s pain ratings collected as part of clinical care over the course of their hospitalization. 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 (Bailey, Daoust, Doyon-Trottier, Dauphin-Pierre, & Gravel, 2010; Voepel-Lewis, Burke, Jeffreys, Malviya, & Tait, 2011). In particular, this measure is highly correlated with visual analogue scales and clinically relevant outcomes such as perceived need for medication (Bailey et al., 2010; Voepel-Lewis et al., 2011). 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.

**Opiate medications.** Chart reviews were used to obtain information on pain medications administered to children. All opiate medications (e.g., morphine) administered during each child’s entire hospitalization were recorded. Similar to the procedures described by Saxe et al. (2001), all doses for opiate medications were summed and divided by each child’s weight and the number of days hospitalized to create a mean dosage (mg/kg/day) value. Given the various forms of opiate medications (e.g.,
morphine, meperidine, oxycodone) and routes of administration (e.g., oral, intravenous), equivalency doses were calculated using a standard pharmacology protocol (Reisine & Pasternak, 1996).

**Child PTSS.** The Child PTSD Symptom Scale (CPSS; Foa, Johnson, Feeny, & Treadwell, 2001) 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 17 symptom items were administered at baseline (prior to randomization). The CPSS was again administered at the 6-week and 6-month follow-up assessments to assess persistent PTSS. The CPSS has demonstrated excellent internal consistency (alpha = .89), test-retest reliability, and convergent validity with structured clinical interviews for PTSD (Foa et al., 2001).

**Data Analysis**

**Study 1**

**Primary Analyses.** To evaluate whether pain predicted child PTSS when controlling for demographics (i.e., age, sex, ethnicity) and empirically-based risk factors (i.e., HR, prior trauma, ASD symptoms, perceived life threat) (Hypothesis 1), we employed two multiple regression analyses. For the first analysis, worst pain rating on the CAS was entered as the predictor, demographic and empirical risk factors as covariates, and child PTSS (i.e., CAPS-CA) as the dependent variable. We then attempted to replicate results by repeating this analysis using worst pain rating on the FPRS as the predictor. For all analyses, alpha was set at .05.
Exploratory Analyses. Given emerging literature suggesting that mild traumatic brain injury (mTBI) is associated with symptom exaggeration and increases risk for and may alter the course and nature of PTSS (Bryant, 2011; Bryant, 2001), we used an ANCOVA to examine potential differences in pain ratings between children who sustained a mTBI versus those who did not, controlling for injury severity. If significant differences in pain ratings emerged, we re-ran primary analyses for Study 1 with the presence of mTBI included as an additional control variable. For all analyses, alpha was set at .05.

Study 2

Primary Analyses. We attempted to replicate findings from Study 1 by utilizing similar multiple regression analyses to evaluate Hypothesis 1. We again employed multiple regressions using worst pain rating during hospitalization (0-10 scale) as the predictor, demographics (i.e., age, sex, ethnicity) and empirical risk factors (i.e., HR, prior trauma, ASD symptoms, perceived life threat) as covariates, and child PTSS (i.e., CPSS at 6 weeks or 6 months) as the dependent variable. Subsequently, we examined whether pain medications moderated the relationship between pain and PTSS (Hypothesis 2) by using multiple regression analyses with interaction terms according to guidelines outlined by Baron and Kenny (1986). Specifically, pain rating was entered as the predictor, an interaction term of opiate medication equivalency dosage by pain rating (i.e., mg/kg/day x pain rating) as the moderator, and child PTSS as the outcome variable. Pain ratings and equivalency dosages were centered prior to creating the interaction term. For all analyses, alpha was set at .05.
**Exploratory Analyses.** Similar to Study 1, we used an ANCOVA to examine potential differences in pain ratings between children who sustained a mTBI versus those who did not, controlling for injury severity. If significant differences in pain ratings emerged, we re-ran primary analyses for Study 2 with the presence of mTBI included as an additional control variable. For all analyses, alpha was set at .05.

**Power Analysis**

For both studies, power analyses were conducted using G*Power version 3.1.7 (Faul, Erdfelder, Lang, & Buchner, 2007). Given that few studies have examined the relationship between pain and PTSS in children and the lack of reported effect sizes, there is little basis for predicting effect sizes for planned analyses. As such, a reasonable middle ground is to predict medium effect sizes. According to conventions outlined by G*Power, the recommended sample size for a multiple regression with one predictor variable and seven control variables (Hypothesis 1), using an alpha level of .05 and a predicted medium effect size, would be 89 participants. Both studies ($N_1 = 243, N_2 = 177$) exceed this requirement. With regards to Hypothesis 2, the recommended sample size for a multiple regression with three predictor variables (pain, average opiate dosage over hospitalization, and their interaction) with a predicted medium effect size is 119 participants. Study 2 was thus adequately powered to examine this hypothesis. For exploratory analyses, the recommended sample size for an ANCOVA with one dichotomous independent variable (mTBI presence), one dependent variable (pain ratings at 6 week or 6 months), and one covariate (injury severity) with a predicted medium effect size is 128 participants. Both studies were thereby adequately powered to examine
exploratory analyses. Observed power for each analysis is reported, and in the event analyses were insufficiently powered, effect sizes were relied upon for interpretation.

**Results**

**Study 1**

**Descriptive Data.** Among this sample of children who had sustained traffic-related injuries, worst pain by child report on the CAS ranged from 0 – 10 (\(N = 238, M = 7.80, SD = 2.50\)). On the FPRS, worst pain by child report ranged from 0 – 6 (\(N = 239, M = 5.06, SD = 1.32\)). While these distributions were negatively skewed, linear regression models do not make assumptions about the normality of predictor variables. Additionally, results were nearly identical when using the original and transformed (i.e., square root, logarithmic) CAS and FPRS variables. As such, results from regression models using the original CAS and FPRS variables are presented below. Total CAPS-CA scores ranged widely from 0 – 122 (\(N = 177, M = 27.33, SD = 21.38\)). Given that scores on the CAPS-CA were highly positively skewed, a square root transformation was used to better approximate a normal curve. See Table 2 for a summary of relationships between primary study variables.

**Primary Analyses.** Multiple regression analyses revealed that worst pain rating on the CAS significantly predicted child PTSS when holding demographic and empirically-based risk factors constant, \(b_{CAS} = .18, SE_{CAS} = .08, p_{CAS} = .02\) (see Table 3). In this model, age and ASD symptoms likewise emerged as significant independent predictors of child PTSS, \(b_{age} = .15, SE_{age} = .08, p_{age} = .05; b_{ASD} = .09, SE_{ASD} = .02, p_{ASD} < .001\). However, worst pain as assessed by the FPRS did not significantly predict child PTSS when controlling for demographic and evidence-based risk factors, \(b_{FPRS} = .19, \)
In this second regression model, age and ASD symptoms again emerged as significant independent predictors of child PTSS, $b_{age} = .17$, $SE_{age} = .08$, $p_{age} = .03$; $b_{ASD} = .09$, $SE_{ASD} = .02$, $p_{ASD} < .001$. For each of the multiple regression analyses, all statistical assumptions were met.

**Exploratory Analyses.** A one-way between subjects ANCOVA revealed no significant differences between those who sustained an mTBI and those who did not in worst pain ratings on the CAS when controlling for injury severity, $F(2,225) = .73$, $p = .39$, $n^2_p = .003$. Likewise, there were no significant differences between those who sustained an mTBI and those who did not in worst pain ratings on the FPRS when holding injury severity constant, $F(2,226) = 1.97$, $p = .16$, $n^2_p = .009$. For these analyses, logarithmic transformations were employed for CAS and FPRS scores to better approximate a normal curve. All other assumptions were met.

**Study 2**

**Descriptive Data.** In this sample, worst pain by child report on a numeric pain rating scale ranged from 0 – 10 ($N = 171$, $M = 6.44$, $SD = 2.84$). Similar to pain ratings observed in Study 1, this distribution was negatively skewed, yet results of primary analyses were nearly identical regardless of whether the original or transformed (i.e., square root, logarithmic) numeric pain rating variable was employed. As such, results presented below represent analyses using the original numeric pain rating variable.

Dosage of opiate medications administered during hospitalization ranged from .02 – 3.60 mg/kg/day ($N = 129$, $M = .41$, $SD = .37$). Opiate medication dosage was significantly associated with average pain [$r(139) = .25$, $p = .003$] but not with worst pain reported over the course of hospitalization [$r(139) = .12$, $p = .15$]. CPSS total scores ranged widely
from 0 – 43 (N = 141, M = 9.77, SD = 9.10) and 0 – 41 (N = 142, M = 8.66, SD = 9.15) at the 6 week and 6 month follow-up assessments, respectively. Given that scores on the CPSS were highly positively skewed, a square root transformation was used to better approximate a normal curve. See Table 4 for a summary of relationships between primary study variables.

**Primary Analyses: Hypothesis 1.** Multiple regression analyses revealed that worst pain rating on the 0-10 numeric scale did not significantly predict child PTSS 6 weeks after pediatric injury when holding demographic and empirically-based risk factors constant, $b_{\text{pain}} = -.01, SE_{\text{pain}} = .04, p_{\text{pain}} = .89$ (see Table 5). In this model, only ASD symptoms emerged as a significant independent predictor of child PTSS 6 weeks post-injury, $b_{\text{ASD}} = .08, SE_{\text{ASD}} = .02, p_{\text{ASD}} < .001$. Similarly, multiple regression analyses indicated that worst pain rating on the 0-10 numeric scale did not significantly predict child PTSS 6 months post-injury when controlling for demographic and empirical risk factors, $b_{\text{pain}} = .05, SE_{\text{pain}} = .04, p_{\text{pain}} = .20$. In this second regression model, ASD symptoms again emerged as a significant independent predictor of child PTSS 6 months after pediatric injury, $b_{\text{ASD}} = .10, SE_{\text{ASD}} = .01, p_{\text{ASD}} < .001$. For each of the multiple regression analyses, all statistical assumptions were met.

**Primary Analyses: Hypothesis 2.** Multiple regression analyses with interaction terms indicated that opiate medications did not moderate the relationship between worst pain rating and child PTSS at 6 weeks, Child PTSS$_{6\text{wk}} = 2.10 + .09(\text{Pain}) - .01(\text{Opiate}) - .10(\text{Pain}_c*\text{Opiate}_c); R^2 = .02, SE_{\text{pain*opiate}} = .27, p_{\text{pain*opiate}} = .72$. Similarly, the relationship between children’s most severe pain and PTSS 6 months post-injury did not vary as a function of opiate medications administered during hospitalization, Child PTSS$_{6\text{mo}} = 1.37$.
+ .18(Pain) - .52(Opiate) + .06(Pain \times Opiate); R^2 = .07, SE_{pain \times opiate} = .28, p_{pain \times opiate} = .84.

There were no significant main effects for opiate medications on child PTSS at six weeks ($p = .71$) or six months ($p = .72$). While there was no significant main effect for worst pain on child PTSS at six weeks ($p = .23$), a significant effect of worst pain emerged for PTSS at six months ($p = .01$). All statistical assumptions for these analyses were met.

**Exploratory Analyses.** A one-way between subjects ANCOVA revealed that there were significant differences between those who sustained an mTBI and those who did not with regards to worst pain ratings on a 0-10 numeric pain scale when controlling for injury severity, $F(2, 153) = 8.71, p < .001, n_p^2 = .10$. More specifically, those without mTBI reported more severe pain ($N = 122, M = 7.03, SD = 2.48$) than those with mTBI ($N = 30, M = 4.90, SD = 3.02$). For this analysis, all statistical assumptions were met.

Subsequently, primary analyses for Study 2 were re-run with the presence of mTBI included as an additional control variable. Findings from these updated multiple regression analyses corresponded to those from the initial models. See Table 5 for full updated model statistics.

**Discussion**

**Does Acute Pain Predict PTSS after Pediatric Injury?**

Findings from this investigation extend the literature on potential risk factors for pediatric medical traumatic stress by exploring the relationships between pain, opiate medication use, and PTSS after injury. Results indicate that the most severe pain children experience during hospitalization for pediatric injury, as measured by the Color Analogue Pain Scale, predicts subsequent PTSS, even after controlling for demographic and empirically-based risk factors. Notably, this model accounted for approximately one third
of the variance in PTSS. On the other hand, worst pain as assessed by the Faces Pain Rating Scale and a 0-10 numeric pain rating system did not emerge as significant independent predictors of child PTSS. While these pain assessments significantly predicted PTSS in simple linear regressions ($p_{FPRS} = .001$, $p_{0-10} = .03$), worst pain on the FPRS and 0-10 scale were no longer significantly associated with subsequent PTSS after controlling for demographic and empirical risk factors.

Numerous differences between these pain assessment instruments may help to explain these inconsistent findings. For instance, it is possible that by providing a more nuanced assessment of pain, the CAS more accurately identified children at risk for persistent PTSS relative to the other pain assessments. Indeed, greater variance in pain ratings on the CAS (i.e., 0 – 10, rounded to the nearest 1/8 unit) as well as in PTSS severity scores on the CAPS-CA (Range = 0 – 122) likely afforded this model increased statistical power relative to the other models using the FPRS (Range = 0 – 6) or the 0-10 numeric scale to measure pain and the CPSS for traumatic stress symptom severity (Range = 0 – 43). Furthermore, there may have been more measurement error associated with the FPRS and 0-10 numeric rating scale. Results suggest greater error associated with the regression weight for the FPRS relative to that of the CAS regression weight, and it is unclear how consistently the 0-10 numeric rating system was administered across medical providers and children in this sample. Additionally, while numerical rating systems have shown validity with older children and adolescents, children under the age of eight years may prefer and better understand faces or visual analogue scales (von Baeyer, 2009). Thus, the 0-10 numeric pain rating scale may not have been as developmentally appropriate for the younger children in this study.
Furthermore, the relationship between the FPRS and 0-10 numeric scale and child PTSS diminished once demographic and empirical risk factors were held constant, suggesting that these pain instruments primarily captured shared variance in child PTSS scores. In contrast, the CAS appeared to capture unique variance not accounted for by other risk factors. Differences in the way in which worst pain ratings were obtained between Studies 1 and 2 and recall bias may partially account for this pattern of findings. In particular, children in Study 1 were asked to rate their most severe pain since the injury (CAS/ FPRS), whereas chart reviews were used to identify the most severe current pain rating (0-10 scale) reported by those in Study 2. Children who recall their pain as more intense may also endorse greater anxiety, pain catastrophizing, or hypervigilance towards aversive interoceptive cues, thereby increasing their arousal, fear, and overall susceptibility to the development and maintenance of PTSS. A growing body of research suggests that state as well as trait anxiety strongly predict children’s memory for pain (Noel, Chambers, McGrath, Klein, & Stewart, 2012; Rocha, Marche, & von Baeyer, 2009; von Baeyer, Marche, Rocha, & Salmon, 2004). Furthermore, cognitive factors such as catastrophic thoughts about pain and hypervigilance or hypersensitivity to unpleasant stimuli likewise influence both the experience and recall of pain (Esteve, Marquina-Aponte, & Ramirez-Maestre, 2014; Keefe et al., 2001; Tsao et al., 2009). Hence, it is possible that the CAS captured some variance in PTSS associated with pain recall bias. Given that we did not directly investigate cognitive and affective factors that may influence children’s recall of pain, additional research is needed to examine the relationship between these constructs and pain assessment tools such as the CAS.
Additionally, differences in sample characteristics may help to explain findings from the current study. All participants in Study 1 were injured during a MVC (i.e., as occupants, pedestrians, or bicyclists), whereas Study 2 included youth with a wider range of injury mechanisms, with more than half injured during sports or recreational activities. Although prior research suggests that PTSS does not vary as a function of injury mechanism (Brosbe, Hoefling, & Faust, 2011), the cognitive appraisal of pain may differ based on the type of traumatic event which produced the injury. For instance, children who sustain injuries while engaging in sports and recreational activities may appraise their pain as less threatening or aversive relative to those who are injured during an unexpected, uncontrollable event such as a MVC. Hence, it may be that children in Study 1 viewed their pain as more dangerous or threatening and thereby experienced greater pain-related distress than those in Study 2. Given that we did not assess for children’s appraisals of pain beyond severity, future research is needed to explore how children understand and make meaning of their pain and other symptoms following an injury.

Lastly, it is possible that the independent relationship between CAS and PTSS documented in this study represents Type I error (i.e., false positive). However, it is unlikely that this finding is merely a statistical anomaly considering the low standard error associated with the CAS regression weight and the aforementioned methodological and statistical limitations of the FPRS and 0-10 numeric rating scale (i.e., restricted variance, greater measurement error). Furthermore, the pattern of results from this study is generally consistent with prior research examining the relationship between pain and PTSS among children. In particular, Saxe and colleagues (2005) similarly found that pain as assessed by the CAS predicted subsequent PTSS among children with burn injuries.
Additionally, a small study of children with injuries likewise suggested a relationship between pain (as assessed by the FPRS) and later PTSS using bivariate correlations, yet the FPRS did not emerge as a significant predictor of PTSS in the final model that included risk factors such as fear at time of trauma, morphine use, and posttraumatic appraisals (Nixon et al., 2010). Future research should attempt to replicate these findings with these and other pain assessment instruments in order to further explore and clarify the relationship between acute pain and PTSS after pediatric injury.

Potential Mechanisms Underlying the Relationship between Pain and PTSS

Several mechanisms may account for the relationship between acute pain after injury and subsequent PTSS among children. Recent neurobiological advances have provided compelling evidence for fear conditioning models of posttraumatic stress (Amstadter et al., 2009). In support of these models, several small studies have proposed that morphine may attenuate fear conditioning processes in children with burns and other injuries, thereby reducing the risk for PTSS (Nixon et al., 2010; Saxe et al., 2006; Saxe et al., 2001; Stoddard et al., 2009). However, in the current study, opiate medications did not moderate the relationship between pain and PTSS among a robust sample of injured children. Moreover, children who received larger doses of opioid analgesics did not demonstrate reduced risk for persistent PTSS. Several methodological factors may help to explain these divergent findings. Whereas prior studies have generally examined small samples of burn patients, this investigation used a larger sample 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 opiate medications than those with non-burn injuries. Indeed, relative to the pediatric burn sample investigated by Saxe and
colleagues (2001), children in this study received lower mean doses of opiate medications (.80 mg/kg/day versus .41 mg/kg/day). It may be that the effects of opiates on fear conditioning processes and thereby PTSS are only observed at higher doses. Furthermore, prior research has relied on change scores to examine the relationship between morphine use and PTSS following pediatric injury. In contrast, this study used PTSS severity as its main outcome measure. As noted by Nixon and colleagues (2010), the use of change scores versus symptom severity may produce variable patterns of results, with symptom severity measures employed more frequently for examining etiological factors for PTSD. Clearly, further research is needed to more thoroughly examine the role of fear conditioning processes and the efficacy of morphine or other opiate medications as a secondary prevention approach after pediatric injury.

Several other models may also help to explain the relationship between acute pain and subsequent PTSS that emerged within this study. A recent investigation by Brown and colleagues (Brown, Kenardy, & Dow, 2014) provides empirical support for the mutual maintenance model, which suggests that pain and PTSD exacerbate and perpetuate one another simultaneously, thereby creating a self-sustaining pattern of co-morbidity (Sharp & Harvey, 2001). However, when the conceptual models nested within the mutual maintenance model (i.e., pain predicting PTSS and vice versa) were examined separately, data suggested that only PTSS facilitated the maintenance of pain (Brown et al., 2014). In contrast to the current study, Brown and colleagues (2014) found that pain did not contribute to PTSD at any time point, although it approached statistical significance. It is possible that our findings differ from those of Brown and colleagues due to varying sample characteristics and assessment methods. In particular, Brown et al.
(2014) examined children with mild to severe TBI, a population that may experience pain differently given changes in cognitive, affective, and neurological functioning. This may limit the applicability of their findings to children with other types of injuries. Furthermore, Brown et al. (2014) relied upon parent assessment of children’s pain, whereas this study examined children’s self-reported pain. Prior research suggests that parents’ own trauma reactions influence their assessment of child symptoms (Kassam-Adams, Garcia-Espana, Miller, & Winston, 2006). Furthermore, pain is an inherently subjective experience that encompasses sensory, emotional, cognitive, and behavioral components that parents may not be able to directly observe. Thus, it is important to obtain children’s self-reported pain whenever possible (von Baeyer, 2009).

Finally, it is also possible that a third variable such as pain catastrophizing increases the risk for both heightened pain and PTSS (Leung, 2012; Sullivan, 2012; Sullivan et al., 2001). This cognitive bias toward aversive, painful sensations and negative interpretations of pain enhances emotional distress, anxiety, and physiological arousal (Sullivan et al., 2001), which may increase susceptibility to intense pain as well as PTSS. Among adults with whiplash and musculo-skeletal injuries, pain catastrophizing is predictive of heightened pain intensity, functional disability, and PTSS (Buitenhuis, de Jong, Jaspers, & Groothoff, 2008; Carroll, Cassidy, & Cote, 2006; Carty, O'Donnell, Evans, Kazantzis, & Creamer, 2011; Sullivan, Stanish, Sullivan, & Tripp, 2002; Sullivan et al., 2009; Vangronsveld, Peters, Goossens, & Vlaeyen, 2008). Emerging research suggests that catastrophic interpretations of pain and other aversive sensations develop early in life and remain relatively stable across painful experiences (Sullivan et al., 2001). Moreover, the relationship between pain catastrophizing and pain intensity has shown
notable consistency across clinical populations (Sullivan et al., 2001). Thus, it may be that children prone to catastrophizing endorse more significant pain and PTSS following an injury. However, little is known regarding the role of pain catastrophizing in the development of PTSS after pediatric injury, and this study did not assess this construct. Further research is needed to carefully examine biopsychosocial mechanisms underlying the association between pain and PTSS among children who have sustained injuries.

Limitations and Future Directions

Several limitations of the current study should be noted. Most notably, this investigation utilized secondary data analysis, and the larger studies from which these data were obtained were not designed specifically to examine the relationship between pain, opiate medication use, and PTSS. Additionally, this investigation used a naturalistic prospective design, and thus relevant variables (e.g., opiate medication) were not experimentally manipulated. We did not obtain information on opiate medication use for the children in Study 1, and we were unable to account for any medications administered at the scene of injuries or on route to the hospital (if applicable) for children in Study 2. Likewise, we did not examine, control for, or manipulate family and medical providers’ responses to children’s pain, which can influence child perception of pain intensity and pain-related distress (Wilson, Moss, Palermo, & Fales, 2014). Future research should seek to employ experimental designs to enable greater control over potential confounding variables and thereby provide more accurate conclusions. Additionally, research is needed to examine the feasibility and efficacy of integrating the CAS or other pain assessment instruments into existing screening protocols for traumatic stress among children. Finally, while this study advances our understanding of potential risk factors for
the development of PTSS, it did not explore underlying mechanisms of action. Future investigations should include measures of relevant physiological, psychological, and social/environmental factors to test mechanisms and models linking pain and PTSS. A more comprehensive understanding of the complex relationship between acute pain and PTSS could help to improve assessment, prevention, and treatment approaches to promote optimal recovery to pediatric injury.

**Clinical Implications**

Results from this investigation have several implications for clinical practice and the refinement of screening tools for posttraumatic stress following pediatric injury. While the field has identified various risk factors for PTSS, a number of barriers impede effective screening during acute trauma care (e.g., time and training limitations, differentiating normative distress vs. indicators of risk). Consequently, clinician perceptions of children at risk for poor psychosocial recovery are often inaccurate. Findings from this study suggest that more severe pain, as assessed by the CAS, may be associated with greater risk for persistent PTSS, even when controlling for demographic and evidence-based risk factors. Given that pain is regularly assessed by a variety of clinicians during routine medical care, the CAS and other pain assessment instruments may thereby represent highly feasible PTSS screening tools. The CAS requires minimal training, is simple and quick to administer, has demonstrated validity across a range of pediatric populations (McGrath & Brigham, 1992), and may facilitate the identification of children who may benefit from more comprehensive assessment or monitoring. As such, clinicians should consider establishing standard screening protocols that include assessments of acute pain. In turn, effective screening during the peri-trauma period
would facilitate more targeted prevention or early intervention efforts. Given that PTSS pose a considerable threat to children’s physical and emotional health after injury, further research is needed to examine the feasibility and efficacy of implementing pain assessment tools to enhance screening and prevention approaches and thereby promote optimal recovery.


### Table 1. Participant Demographics

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<th>CHILDREN</th>
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<th>Study 2 (N = 177)</th>
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<td>Mechanism of Injury</td>
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<tr>
<td>Sports / Recreational Activities</td>
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<td>Falls</td>
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<td>Traffic-related Injury Mechanisms</td>
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<td>Occupants</td>
<td>39 (16)</td>
<td>4 (14)</td>
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<tr>
<td>Pedestrians</td>
<td>81 (33)</td>
<td>12 (41)</td>
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<td>Bicyclists</td>
<td>123 (51)</td>
<td>7 (24)</td>
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<td>Motorcyclists</td>
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<td>Age (years)</td>
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<tr>
<td>Length of hospital stay (days)</td>
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<td>Injury Severity Score</td>
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<th>Study 2 (N = 177)</th>
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<tr>
<td>Mother</td>
<td>164 (79)</td>
<td>249 (85)</td>
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<tr>
<td>Father</td>
<td>25 (12)</td>
<td>39 (13)</td>
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<td>Other caregiver</td>
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<td>4 (1)</td>
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Participant demographics as reported at baseline enrollment for each study.
### Table 2. Correlations among Primary Variables in Study 1

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<td>Acute Stress Symptoms</td>
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<td>.14*</td>
<td>-.14*</td>
<td>.07</td>
<td>-.07</td>
<td>.14*</td>
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<td>Perceived Life Threat</td>
<td>.26**</td>
<td>.28***</td>
<td>.02</td>
<td>.15*</td>
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<td>.07</td>
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*p < .05  
**p < .01  
***p < .001
Table 3. Predictors of child PTSS (CAPS-CA) in Study 1

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Table 4. Correlations among Primary Variables in Study 2

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<tr>
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<td>.03</td>
<td>.01</td>
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*p < .05  
**p < .01  
***p < .001
### Table 5. Predictors of child PTSS (CPSS) in Study 2

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<td>(R^2)</td>
<td>(R^2_{adj})</td>
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<td>Prior trauma history</td>
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<tr>
<td>Acute stress symptoms</td>
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\(p < .05\)
\(p < .01\)
\(p < .001\)
Appendix A: Color Analogue Pain Scale

Figure 1. Color Analogue Pain Scale (McGrath et al., 1996)
Appendix B: Faces Pain Rating Scale

Figure 2. Faces Pain Rating Scale (Bieri et al., 1990)