SLEEP DISTURBANCES FOLLOWING PEDIATRIC TRAUMATIC BRAIN INJURY

A Thesis Presented to

The Faculty of the Department of Psychology

University of Houston

In Partial Fulfillment

Of the Requirements for the Degree of

Master of Arts

By

Jesse T. Fischer

August, 2016

SLEEP DISTURBANCES FOLLOWING PEDIATRIC TRAUMATIC BRAIN INJURY

Jesse Fischer, B.A.

APPROVED:

Linda Ewing-Cobbs, Ph.D.
Committee Co-Chair
Department of Pediatric Neurology
University of Texas Health Sciences Center at Houston

H. Julia Hannay, Ph.D.
Committee Co-Chair
Department of Psychology
University of Houston

Candice Alfano, Ph.D.
Department of Psychology
University of Houston

Patrick Taylor, Ph.D.
Department of Psychology
University of Houston

Steven Craig, Ph.D.
Interim Dean, College of Liberal Arts and Social Sciences
Department of Economics

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ABSTRACT

Traumatic brain injury (TBI) remains a glaring issue for public health in the United States, particularly in children and adolescents. One of the most frequent complaints of patients following TBI is the presence of sleep disturbances (SD). Bodily injury in children and adolescents also presents a public health concern as well. The association between pediatric TBI and SD is vastly understudied, and even less is known regarding SD after bodily injury. Importantly, SD in children and adolescents has been linked independently to a number of adverse health outcomes, such as internalizing behavior problems. Additionally, posttraumatic stress symptoms have been linked to traumatic injury and to SD. The current study aimed to investigate chronic SD after TBI and bodily injury in children and adolescents, as well as relations between SD, internalizing behavior problems, and posttraumatic stress after injury. Study design utilized data from 8-15 year olds following TBI, extracranial/bodily injury (EI), and typically developing children. At 6 and 12-month time points, mixed model analyses of variance were used to assess group differences in SD, internalizing behavior problems, and posttraumatic stress, while multiple mediation models analyzed mechanisms of action. SD was significantly positively associated with severity of injury in the EI group. SD was higher in injury groups than in typically developing children when TBI and EI groups were combined. Internalizing behavior problems were higher in both injury groups. SD and internalizing behavior problems had a persisting bidirectional association where each mediated the other in each injury group when compared to typically developing children. Posttraumatic stress symptoms did not vary between injury groups and were not mediated by SD after injury. Findings are relevant for further understanding and clinical management of SD and their relation to internalizing problems after traumatic injury.

TABLE OF CONTENTS

Introduction

Traumatic brain injury (TBI) is a leading cause of death and disability in the U.S., as an average of 1.7 million persons every year have a TBI either alone or in conjunction with other injury (Faul, Xu, Wald, & Coronado, 2010). These estimates are based on emergency-related visits, and do not include the large number of patients who either do not seek care or receive only outpatient care. Thus, epidemiological numbers are likely even higher. The National Institute of Health defines TBI as an alteration in brain function, or other evidence of pathology, caused by an external force (NINDS, 2015). Among all ages, the highest combined rates of TBI-related emergency department (ED) visits, hospitalizations, and deaths occur in young children, followed by adolescents (Coronado, Thurman, Greenspan, & Weissman, 2009; Faul et al., 2010). Pediatric TBI remains a stark public health risk, as survivors are often struck with debilitating and cumulative deficits in multiple domains after injury.

After TBI, chronic problems arise in academic achievement (Anderson, Catroppa, Haritou, & Rosenfeld, 2005; Ewing-Cobbs et al., 1997; Ewing-Cobbs, Fletcher, Levin, Iovino, & Miner, 1998; Ewing-Cobbs et al., 2006), social functioning (Catroppa et. al, 2014; Levin & Hanten, 2005), overall quality of life (Anderson, Brown, Newitt, & Holle, 2011; Di Battista, Soo, Catroppa, & Anderson, 2012; Rivara et al., 2012), and psychological health (Max et al., 2011; Max et al., 2012; Max, 2014). In addition to such sequelae, a growing base of research suggests sleep disturbances (SD) are a prevalent and debilitating problem after pediatric TBI (Beebe et al., 2007; Milroy, Dorris, & McMillian, 2008; Pillar et al., 2003; Shay et al., 2014; Sumpter, Dorris, Kelly, & McMillan, 2013, Tham et al., 2012). Although a greater number of studies have been published focusing on SD in adults with TBI, there is

emerging evidence children with TBI may also develop such disturbances (Stores & Stores, 2013; Wiseman-Hakes, Colantonio, Gargaro, 2009). The aims of this study were 1) to further investigate the characteristics and development of SD occurring in the chronic stage of recovery in children and adolescents with closed-head injury or other bodily injury, 2) to determine risk factors for the post-injury development of SD, 3) and to examine the relationship between psychological health and SD after pediatric injury.

Pediatric TBI

Epidemiology. Of the 1.36 million individuals admitted to the ED for TBI, over 500,000 are ages 0-19 (Faul et al., 2010; Keenan & Bratton, 2006). A child who sustains a TBI may endure severe disability, and the cost to society is also high. Pediatric TBI inpatient costs are estimated to be over $1 billion a year, while overall hospital charges rise over $1 billion as well (Keenan & Bratton, 2006; Schneier, Shields, Hostetler, Xiang and Smith, 2006). In a recent epidemiological study, the most common mechanism of injury was due to falls, followed by sport-related injury (Barlow, 2013). In adolescence, the number of injuries occurring from motor vehicle accidents increased as well. Substantial improvement in the management and treatment of children with TBI may not only help injured children but also may mitigate the economic burden on families and society.

Pathophysiology. Pathophysiology after TBI involves a wide range of neuroanatomical sites and mechanisms. Rotational acceleration and deceleration forces have been associated with increases in the severity of injury (Ommaya, Fass, & Yarnell, 1968). In such animal models, the lowest rotational forces lead to subdural and subarachnoid hemorrhages, while axonal injury and eventually midbrain and brainstem pathology occurred as rotational force increased (Ommaya, Goldsmith, & Thibault, 2002). TBI results in primary

and secondary injuries that complicate recovery and treatment. In a recent study of children with TBI, common acute pathology from head injuries included epidural hematoma, subdural hematoma, and diffuse axonal injury. Secondary insults consist of cerebral edema, mass effect, hypoxia, hypotension, decreased cerebral perfusion, and hypercarbia (Barlow, 2013). In addition, animal models have illustrated the resulting cascade of metabolic dysfunction and ionic flux after TBI that contribute to widespread neuronal injury (Giza & Hovda, 2014). TBI research often includes injury comparison groups to control for the specific mechanisms of head injury. Comparison groups such as orthopedic injury (OI; bodily injury to regions other than the head) are often used. For the current study, the injury control group will be classified as the extracranial injury (EI) group, which also includes internal non-head injuries (e.g., laceration of spleen).

Classification of injury severity. TBI severity is most often clinically assessed using acute injury characteristics. The Glasgow Coma Scale (GCS; see Appendix A) is currently the most frequently used measure of acute TBI severity, assessing eye opening, verbal response, and motor response (Teasdale & Jennett, 1974). Additionally, clinical findings such as loss of consciousness, posttraumatic amnesia, pupillary reactivity, hypoxia, hypotension and/or positive neuroimaging results are often used for classification (Adelson et al., 2012). Severity has been traditionally measured as mild (GCS = 13-15), moderate (GCS = 9-12), and severe (GCS = 3-8), with the more recent "complicated mild" classification supported in clinical research. Complicated mild is commonly defined as a GCS of 13-15 with positive CT imaging, excluding linear skull fractures. Despite the distinct labels used, observed sequelae of children with complicated mild TBI appear to resemble those associated with moderate TBI (Levin et al., 2008; Williams, Levin, & Eisenberg, 1990).

Though the GCS has been widely used, outcomes of patients within severity groups differ substantially.

Additional acute variables are often used for assessing severity of bodily-injury. The Abbreviated Injury Scale (AIS) has been used to classify overall bodily injury, and the National Institute of Neurological Disorders and Stroke currently includes the AIS as a Common Data Element for measuring TBI severity (Adelson et al., 2012). The AIS is an anatomically based global severity measure that classifies each injury by body region according to its relative importance (Gennarelli & Wodzin, 2006). This scale is not only utilized for the assessment of bodily injury, but also to control for bodily injury when studying children with TBI.

Post-TBI and -bodily injury sequelae. Both acute and chronic deficits in neuropsychological skills and psychological health are associated with pediatric TBI. Long-term impaired academic achievement has been demonstrated in children with TBI, in both functional and specific academic skill areas (Ewing-Cobbs et al., 1997; Ewing-Cobbs et al., 1998; Ewing-Cobbs et al., 2004; Ewing-Cobbs et al., 2006). Seriously injured patients are more likely to have occupational and educational problems over 10 years post-injury, and behavioral and arithmetic skill deficits remain, regardless of severity of TBI (Anderson et al., 2011; Catroppa, Godfrey, Rosenfield, Hearps, & Anderson, 2012). Catroppa and Anderson (2005) indicated some attentional deficits remain 24 months after injury, which may play a role in the development of behavioral problems prevalent after TBI. A recent report of 6-14 year olds demonstrated children with more severe TBI were at increased risk for internalizing but not externalizing behavioral symptoms 6 months after injury (Anderson et al., 2012). Further supporting to such concerns for emotional adjustment, studies have also reported the

development of novel internalizing and externalizing disorders and post-injury personality changes in children after TBI (Max et al., 2006; Max et al., 2012; Peterson et al., 2013). However, specific injury and non-injury related factors determining such longer-term emotional adjustment outcomes have not been consistently corroborated in research thus far.

Though typically applied as an injury comparison group, children with OI have demonstrated psychological and functional problems as well. Fay et al. (2009) examined functional outcomes including neuropsychological, behavioral, adaptive, and academic domains in children with TBI and OI. Results at 12 months after injury revealed one or more deficits in 25% of children with OI, 45% children with moderate TBI, and 57% of children with severe TBI. Additionally, children hospitalized with serious fractures displayed initial psychosocial and functional impairment (Ding et al., 2006; Stancin et al., 2001). However, most showed positive recovery one year after injury, suggesting that although children with bodily injury are at risk for psychological and functional problems, the timeline of recovery is unclear.

Posttraumatic stress develops in children following TBI, possibly playing a role in chronic impairment. For instance, children with severe TBI reported higher rates of posttraumatic stress at 12 months than those with moderate TBI or OI (Levi, Drotar, Yeates, & Taylor, 1999). This relationship was also confirmed by parent report. Furthermore, one study showed that13% of children with severe TBI developed a diagnosis of posttraumatic stress disorder (PTSD) by 12 months post-injury (Gerring et al., 2002). Higher posttraumatic stress has also been associated with poorer health outcomes. In another study of hospitalized children with TBI, post-injury stress levels and severity of TBI significantly predicted new onset mood and/or anxiety disorders 6 months after injury (Luis & Mittenberg, 2002).

Demonstrating additional support for such processes, Zatzick et al. (2008a) studied adolescents recruited from surgical inpatient units, and showed those who suffered from early PTSD symptoms reported more health problems and psychological dysfunction during the year after injury.

For studies comparing posttraumatic stress after TBI versus other bodily injury, results vary yet still suggest more psychological impairment and posttraumatic stress after TBI. In contrast to the findings showing children with severe TBI were at increased risk for posttraumatic stress (Levi et al., 1999), a recent report revealed adolescents with mild TBI had significantly higher posttraumatic stress symptoms than those with more severe TBI and those with arm injuries (O'Connor et al., 2012). Additionally, an earlier multisite study also showed children with more severe TBI exhibited diminished risks of PTSD when compared to children hospitalized with non-TBI and mild TBI (Zatzick et al., 2010). This report is consistent with some research suggesting longer posttraumatic amnesia after injury may be associated with worse encoding of traumatic memories, thus decreasing risk for posttraumatic stress (Bryant et al., 2009; Gil, Caspi, Ben-Ari, Koren, & Klein, 2005). However, linking severity of injury with posttraumatic amnesia and posttraumatic stress has not been extensively studied in children, and findings remain inconsistent. Prevalence of PTSD after TBI and bodily injury is supported, but results and methodological design vary (Harvey & Bryant 2000, Bryant, Marosszeky, Corrks, & Gurka, 2000; Hickling, Blanchard, Silverman, & Schwarz, 1992; Levin et al., 2001; Mellman, David, Bustamante, Fins, Esposito, 2001; Ohry, Rattok, & Solomon, 1996). For instance, over 20% of a sample of 2707 surgical inpatients reported posttraumatic stress 12 months after injury, but this study included those with TBI and non-TBI injuries (Zatzick et al., 2008b). Considering the

inconsistencies of research thus far, details of the relationship between TBI, bodily injury, and PTSD diagnosis remains unsettled.

A primary aim of research in TBI has been to determine risk factors for impaired functional recovery. In the pediatric population, academic, behavioral, social functioning, and quality of life concerns seem to be influenced by multiple pathways. Moreover, SD after TBI may result from or lead to the manifestation of difficulties in such domains.

Pediatric SD and associated impairment

Child and adolescent SD. Sleep patterns of healthy children typically change throughout development. As a child grows into adolescence and eventually young adulthood, there is a general decrease in the proportion of slow wave sleep, REM, and sleep duration (Dahl & Carskadon, 1995; Dahl & Lewin, 2002). Past studies demonstrate 20-30% of children exhibit some type of bedtime problems or night wakings (Mindell & Owens, 2003), and lifetime presence of insomnia in 13-16 year olds may be greater than 10% (Owens, 2009). SD in children and adolescents are associated with widespread deficits and developmental problems, reaching cognitive, behavioral, social and physical domains. In other words, children who have persistent sleep problems are also at risk for developing additional psychopathology (e.g., Gregory et al., 2005). Such psychopathology includes significantly reduced sustained attention and psychomotor vigilance (Sadeh, Raviv, & Gruber, 2000), higher rates of behavioral problems including oppositionality (Beebe, Fallone, Godiwala, & Flanigan, 2008), decreased ability to deal with complex cognitive tasks (Sadeh, Gruber, & Raviv, 2002), and physical/cardiovascular health problems (Amin et al., 2002; Gozal & Kheirandish-Gozal, 2008). These findings help illustrate the broad and powerful impact of sleep on the developing child's quality of life.

SD and mental health. Current research demonstrates a complex relationship exists between SD and mental health. A number of studies indicate sleep problems can independently predict development of emotional and behavioral problems later in life. For instance, Johnson, Chilcoat, and Breslau (2000) showed that children with trouble sleeping had significantly higher parent-reported rates of anxiety and depression years later. In 2002, Gregory and O'Connor demonstrated a significant predictive relationship between sleep problems at age 4 and behavioral and emotional problems, such as anxiety and depression, in middle-adolescence. In contrast, several studies demonstrate children with increased psychological problems are at risk for SD (Alfano, Beidel, Turner, & Lewin, 2006; Alfano, Ginsburg, & Kingery, 2007; Gregory, Eley, O'Connor, & Plomin, 2004; Ong, Wickramaratne, Min, & Weissman, 2006). As a whole, the evidence suggests there is a bi-directional relationship between sleep and mental health.

Posttraumatic stress and SD. Individuals who experience trauma are at a high risk for developing sleep problems, while SD following trauma also may predict posttraumatic stress. Insomnia, commonly defined as difficulty initiating or maintaining sleep, has been reported as a frequent disturbance after trauma (Green 1993). Glod and colleagues (1994) reported children who experience abusive trauma have higher rates of SD than both healthy and depressed controls. Sleep arousal disturbances, or "nighttime awakenings," typically manifesting as nightmares and sleep terrors, are also prevalent after trauma. Additionally, SD such as nightmares occurring in the immediate aftermath of a trauma are predictive of later PTSD (Mellman, & Hipolito, 2006). When investigating long-term impairment, SD at 24 months in children and adolescent survivors of Hurricane Katrina was associated with severity of posttraumatic stress at 30 months post-trauma (Brown, Mellman, Alfano, &

Weems, 2011). This highlights the chronic impact that trauma may have on both stress and sleep, but additional research is needed to more fully understand the course of functioning after trauma in children.

SD and TBI

Epidemiology of SD after TBI. Accurate assessment of prevalence for pediatric SD after TBI is difficult, given the paucity of rigorous research in the area. Epidemiological studies targeting sleep quality in children with TBI have not been published yet. Clinically oriented studies have varied substantially in terms of methods used to characterize SD. Thus far only a small number of peer-reviewed studies have included either a healthy or other injury comparison group in their study design. Studies also vary in terms of the breadth of coverage of SD behaviors afforded by the questionnaires used; coverage ranges from single items rating SD to validated, multidimensional questionnaires assessing multiple dimensions of sleep quality. Studies also differ in the timing of assessment of SD, whether preinjury SD was evaluated, and the respondent. Despite the relative dearth of empirical investigations and variability in methods, several studies have shown higher rates of SD in children with TBI than in typically developing or injured comparison groups (Beebe et al., 2007; Gosselin et al., 2009, Milroy et al., 2008, Shay et al., 2014, Sumpter et al., 2013, Tham et al., 2012). These studies demonstrated disturbances in the acute and chronic stage of recovery, as well as across the range of injury severity.

Consensus has not been reached as to whether children with bodily injury, often utilized as comparison groups in TBI research, also endure higher levels of SD. However, a recent study compared SD in children with TBI versus OI, finding over 17% of children in each group to have sleep problems 12 months after injury (Ewing-Cobbs et al., 2014), which

is similar to community rates (Lipscomb, Satin, & Neutra, 1992; Ohayon, 2002). Though the research is sparse, there are signs indicating that bodily injury may be related to SD. Studies have reported sleep difficulties in severe burn patients and children with chronic pain, although mechanism and pathology of injury is more specific in such cases (Kravitz et al., 1993; Mayes et al., 2013; Roth-Isigkeit, Thyen, Stöven, Schwarzenberger, & Schmucker, 2005). The influence of injury and non-injury related factors impacting SD over the course of child development has yet to be comprehensively studied. There is also a stark need for more research in school-aged children and adolescents. The most methodologically rigorous study to date included a sample of 3 to 6 year old children (Shay et al., 2014), but similar studies are warranted for older children.

Common forms of SD associated with TBI. Investigations of sleep and TBI often include an overall measure of SD, either as a total score of specific subscales or a general stand-alone marker. Tham et al. (2012) used prospective parent-report questionnaires of children with complicated mild and moderate/severe TBI, finding higher SD in children with TBI than children with OI at 12 and 24 months after injury. However, the sleep measure consisted of only one item from the Pediatric Quality of Life Inventory (Varni, Seid, & Kurtin, 2001). Pillar et al. (2003) matched ninety-eight 7-15 year olds admitted to a tertiary care hospital for mild head injury (MHI) (GCS of 13 or higher) with 98 healthy controls. Authors utilized an 8-item child report questionnaire of SD, assessing difficulties falling asleep, frequent and early-morning awakenings, movements in sleep, anxiety/fear on arousals from sleep, non-restorative sleep, nightmares, and daytime somnolence. A higher percentage of the MHI group (28%) complained of total SD than did controls (11%), as well as higher rates of bruxism (10% and 4%, respectively). A caveat to these findings is that the

disturbances were reported anywhere from 6 months to 6 years post injury, while impairment likely fluctuates in the years following injury.

SD in research is often categorized into specific types that suggest potential sleep disorders. Mathias and Alvaro (2012) found insomnia, hypersomnia, and sleep apnea to be the most commonly diagnosed disorders in adults after injury, respectively. However, symptoms of insomnia have been reported less frequently in pediatric populations. Hypersomnia, typically defined by symptoms of excessive daytime sleepiness (EDS), has been reported more commonly (Hooper et al., 2004). EDS can be caused by poor sleep quality and poor sleep efficiency, in addition to sleep disorders. In addition, the presence of SD such as sleep breathing disorders and disorders of sleep hyperhidrosis has not been established in children with TBI. The mechanisms of SD after injury may not influence such disorders, but further investigation is needed to clarify such questions.

More recently, Shay et al. (2014) revealed parent-reported shorter sleep duration and increased bedtime resistance in 3-6 year olds after TBI. The prepubertal age range of this cohort may have some specific impact on the presence of bedtime resistance, but the TBI group was compared to an OI comparison group, strengthening such findings. Kaufman et al. (2001) studied 19 adolescents reporting SD 3 years post MHI. Five-day actigraphy and whole-night polysomnography (PSG) confirmed the MHI group's sleep difficulties, showing decreased sleep efficiency and more minutes of wake time compared to healthy controls. This study was limited by a small sample size, selective recruitment methods, and not controlling for premorbid or post-injury functioning. Sumpter et al. (2013) reported increased trouble sleeping and higher overall sleep problems pre-to-post TBI at 6-12 months post-injury. Children with TBI also had longer sleep latency and poorer sleep efficiency on

actigraphy. The consistency between results on objective and subjective sleep measures in these studies contrasts from the more common discrepancies found between such measures of sleep after TBI.

Objective and subjective measures of sleep after TBI. Findings among objective PSG and actigraphy are often inconsistent with subjective parent- and self-report measures of SD after TBI, however this is found in non-injured children as well**.** For instance, actigraphy and child self-report 2 years after mild TBI failed to confirm parent-reported overall SD (Milroy et al., 2008). In contrast, Aurora et al. (2007) found normal PSG sleep architecture in adults with TBI up to 7 years post-injury, despite self-reported complaints of SD. In addition, EEG findings failed to corroborate subjective SD ratings of children 4-6 weeks after injury (Korinthenberg, Schreck, Weser, & Lehmkuhl, 2004). The cause for discrepancies between objective measures, parent, and self-report is ambiguous. Potentially, children and adolescents themselves might be less aware of, and thus have difficulty self-reporting, the SD parents may observe (Forbes et al., 2008). Methodological variability between studies and individual sleep variability may play a role in such inconsistencies as well.

A well-validated parent-report measure of childhood SD is the Sleep Disturbance Scale for Children (SDSC) (Bruni et al., 1996; Spruyt, Cluydts, & Verleye, 2004). Subscales of the SDSC include: sleep initiation and maintenance (insomnia), sleep-related breathing (e.g. sleep apnea), sleep arousal/nightmares (nighttime awakening), sleep-wake transition, excessive somnolence (daytime sleepiness or fatigue), and hyperhidrosis (bed sweating). The SDSC has been effectively used as a routine measure of sleep in children with neurological disorders as well (Cohen, Halevy, & Shuper, 2013). The subscales and total scores of the

SDSC identify children with SD and potential areas of clinical concern within such disturbances.

Injury-related factors associated with the development of SD after TBI

Severity of TBI. Surprisingly, a majority of published literature demonstrates a higher prevalence of SD after mild TBI, though there is evidence of sleep problems across all severities (Ayalon, Borodkin, Dishon, Kanety, & Dagen, 2007; Kraus et al., 2009; Mahmood, Rapport, Hanks, & Fichtenberg, 2004; Tham et al., 2012). While this trend is prominent in the adult literature, research on children with TBI has not consistently shown this pattern. For instance, Shay et al. (2014) demonstrated both mild and moderate/severe TBI groups were associated with SD in distinct ways. While those with complicated mild and moderate TBI demonstrated more total sleep problems than children with OI at 6 months, children with severe TBI reported more bedtime resistance than children with OI at this time point. Children with severe TBI also had shorter sleep duration than those with complicated mild TBI 18 months after injury.

Children with severe TBI and SD also demonstrated more cognitive issues at 6 and 12 months when compared to children with severe TBI who did not have SD. Additionally, Beebe et al. (2007) reported worse SD after severe TBI than after mild TBI or OI, while parent-report in another study reported SD after mild TBI (Milroy et al., 2008). Because of the many factors that influence recovery after injury, the relationship between TBI severity and sleep problems is not yet well understood. Children with mild TBI might be more cognitively aware of sleep difficulties, while those with more severe injury may have greater deficits of concern. However, more studies are needed to investigate the relationship between injury type, severity, and SD.

Posttraumatic stress. Although posttraumatic stress is clearly associated with SD, only a small number of studies have investigated the impact that posttraumatic stress may have on sleep difficulties after TBI. Two studies of the same cohort of children with GCS of 10 or higher after motor vehicle accidents illustrated a link between child posttraumatic stress levels after injury, presence of nightmares, and prolonged sleep latency (Wittmann, Zehnder, Schredl, Jenni, & Landolt, 2010; Wittmann, Zehnder, Jenni, & Landolt, 2012). Sleep latency was defined as the subjective amount of time needed to fall asleep. In the initial study, early posttraumatic nightmares predicted posttraumatic stress symptoms 2 and 6 months later (Wittmann et al., 2010), in line with previous research demonstrating early SD predicts PTSD after trauma (Mellman & Hipolito, 2006). Additionally, posttraumatic stress symptom severity predicted increased sleep onset and maintenance problems (Wittmann et al., 2012). Though the presence of SD was demonstrated in this sample, these reports did not include a control or comparison group in their design, and did not separate children with head injury versus other bodily injury. Thus, additional research on the complex relationship between TBI, posttraumatic stress, and SD is needed.

Non-injury factors associated with the development of SD after TBI. Although a host of non-injury related factors have been associated with recovery after TBI, those factors specifically contributing to SD have yet to be fully understood. In addition, the continuously moving target of development presents a challenge for comparing functioning across pediatric age groups. Still, preliminary findings indicate possible risk factors for SD after TBI include female gender, presence of psychosocial problems, and frequent pain (Tham et al., 2012). In the study by Pillar et al. (2003), children with SD after TBI had higher average BMI than those without SD, which has also been implicated in sleep-related breathing

disorders. In pediatric TBI research, those with higher premorbid risk factors are at higher risk for impairment after injury (Babikian & Asarnow, (2009); Babikian et al., 2012). Along similar lines, it is possible that premorbid functioning before TBI may be associated with SD after TBI. Future studies including premorbid risk factors of TBI and SD as covariates will help distinguish injury from non-injury related factors.

Impairment associated with SD after TBI. A wide range of deficits has been observed in patients with SD after TBI. Such impairment often mirrors general problems seen after TBI and after SD alone, as previously described above. In pediatric populations, few studies investigating SD in children after TBI have also targeted other domains of functioning. In one study, sleep problems were associated with an increased risk for externalizing and internalizing problems (Shay et al., 2014). Additionally, Tham et al. (2012) reported frequency of SD in moderate and severe TBI predicted poorer behavioral adjustment and participation levels. In another report on SD after TBI, children with TBI had poorer quality of life and more behavioral problems than sibling controls, but this was not statistically related to SD (Sumpter et al., 2003). More work is needed to shed light on details of these complex injury sequelae.

Rationale

The purpose of this study was to further characterize the long-term course and relation of SD in children with TBI and EI in comparison to a healthy comparison group. Few studies have targeted the association between severity of injury and SD, and only a small number have included an injury comparison group. Additionally, there are limited and varied findings as to how TBI or bodily injury relate to specific types of SD. SD in children has been associated with psychological health difficulties such as internalizing problems. The

presence of a bidirectional relationship between SD and psychopathology warrants further investigation of such processes in injured populations. However, literature on the relation of SD to chronic psychological health outcomes following pediatric injury remains sparse.

Posttraumatic stress occurs frequently after traumatic injury, and literature suggests this impairment may be more prevalent after TBI than after bodily injury. However, SD in such populations has not been well studied. The few studies investigating posttraumatic stress and SD in children after injury were retrospective and did not separate injury type and severity, and there (Wittmann et al., 2010; Wittmann et al., 2012). To address these gaps in the literature, the purpose of the current study was to investigate the relationship between injury type and severity, psychological health, and SD following pediatric injury.

Hypotheses

1) The severity of TBI and EI were predicted to have a significant positive association with SD at 6 and 12 months post injury.

2) At 6 and 12 months after injury, children with TBI were expected to have significantly higher levels of overall SD than children with EI, while those with traumatic injury were expected to have significantly higher levels of overall SD than typically developing (TD) children.

3) The type of SD was expected to differ significantly across groups. In the areas of initiating/maintaining sleep, excessive somnolence, and nighttime awakenings, children with TBI were predicted to have significantly more SD than children with EI, and children with traumatic injury were expected to have significantly more SD than TD children at 6 and 12 months. In addition, the TBI, EI and TD groups were

hypothesized to have similar rates of sleep hyperhidrosis and sleep breathing disorders at 6 and 12 months.

4) Internalizing behavior problems were expected to vary by group. Children with traumatic injury, including those with TBI and those with EI would have significantly higher levels of internalizing behaviors than TD children at 6 and 12 months. In addition, internalizing behavior problems in children TBI would be higher than problems in the children with EI, and this pattern was expected across time.

5a) Bidirectional relationships have been found between SD and psychopathology. SD has been shown to predict the development of anxiety and depression; conversely, internalizing symptoms have been shown to predict SD. Therefore, bidirectional relations among chronic injury groups, SD, and internalizing symptoms were examined. The effect of pediatric injury on internalizing problems at 6 and 12 months after injury was predicted to be mediated significantly by the presence of SD and that SD at 6 months similarly was expected to mediate the relation between group and internalizing problems at 12 months.

5b) Due to limited research in the area, exploratory hypotheses were examined also. The effect of pediatric injury on SD at 6 and 12 months after injury was hypothesized to be mediated significantly by the presence of internalizing problems and internalizing problems at 6 months similarly were predicted to mediate the relation between group and SD at 12 months.

6) Posttraumatic stress levels were expected to vary by group. Children with TBI were hypothesized to have significantly higher levels of posttraumatic stress than children with EI at 12 months, but not at 6 months post-injury.

7a) Though posttraumatic stress is associated with SD, the influence of type of injury and chronic posttraumatic stress on SD has not been well studied. The effect of injury group on SD at 6 and 12 months was predicted to be mediated significantly by the presence of posttraumatic stress, and alternatively, posttraumatic stress at 6 months was expected to mediate the relation between group and SD at 12 months.

7b) The effect of injury group on posttraumatic stress at 6 and 12 months was predicted to be mediated by the presence of SD, and SD at 6 months was expected to mediate the relation between group and posttraumatic stress at 12 months.

Methods

Participants

This study was submitted for Committee for the Protection of Human Subjects (CPHS) approval by the University of Houston (UH) Institutional Review Board (IRB). Data for this archival study were collected originally in compliance with regulations mandated by the IRB of the University of Texas – Health Science Center at Houston (UT-HSCH), and were part of a National Institutes of Health grant. UH CPHS approval and permission from Linda Ewing-Cobbs, PhD, to use the data collected were obtained for this study before any collected data were viewed or obtained. Archival data from approximately 36 children and adolescents with varying severity of closed head injury, as well as 24 with EI who were admitted to a Level 1 Trauma hospital in Houston, Texas were used for the current study. Additionally, data from 27 TD children also recruited previously through posting of flyers throughout Texas Medical Center were used. Under the original protocol, children with TBI and those with EI hospitalized or treated in the emergency department were between the ages of 8 and 15, and the TD children were first assessed between the ages of 8 and 15.

In the original study from which these data were taken, inclusion criteria for children with TBI were: mild TBI, defined as a post-resuscitation Glasgow Coma Scale (GCS; Teasdale & Jennett, 1974) score of 13-15, complicated mild, defined as a post-resuscitation GCS of 13-15 with evidence of parenchymal injury on computed tomography (CT) scan, moderate TBI, defined as a post-resuscitation GCS score of 9-12, and severe TBI, defined as a post-resuscitation GCS score of 3-8 (Levin et al., 2008; Williams, Levin, & Eisenberg, 1990). For children with EI, an AIS score of 2, 3, or 4 was required, as well as no head or facial injuries to minimize the possibility of co-occurring mild TBI. The Injury Severity Score will be used for assessing EI severity, and is calculated by the sum of the squares of the 3 most severely injured body regions rated by the AIS (Buckley et al., 1994).

Under the initial protocol, exclusionary criteria were: previous history of brain injury, severe developmental disability, hospitalization for a psychological disorder, history of child protective services intervention (except in the case of family placement with no abuse or serious neglect), or if they sustained a gunshot wound to the head or other penetrating head injury. Additionally, all children included in the original study were either bilingual or primarily English speaking.

Procedure

In the original study informed written consent was obtained from the child's guardian, in accordance with guidelines established by the UT-HSCH IRB. Children who were ages 11 and older signed the written adolescent consent while those under 11 signed the written child assent at the 6 week visit.

When data were collected at that time, participants were examined individually at the UT-HSCH, in a quiet testing room. A trained research assistant conducted evaluations at 6

and 12-month time points. Parents and children were each given a separate large battery assessing various domains of the participant's functioning.

Measures

Sleep disturbance

Sleep Disturbance Scale for Children (SDSC). The SDSC is a well-validated parent-report measure of childhood SD, demonstrating internal consistency (0.71) and test-retest reliability (.71) (Bruni et al., 1996; Spruyt, Cluydts, & Verleye, 2004). The 26-item measure uses a 5-point Likert-type scale (0 = least severe and 5 = most severe) and contains 6 subscales previously categorized using factor analysis (see Appendix B). The sleep disturbance subscales are: sleep initiation and maintenance (insomnia), sleep-related breathing, sleep arousal/nightmares, sleep-wake transition, excessive somnolence (daytime sleepiness or fatigue), and hyperhidrosis (bed sweating). The SDSC has been effectively used as a routine measure of sleep in children with neurological disorders as well (Cohen, Halevy, & Shuper, 2013). The current study utilized a total score, representing overall SD, and subscale scores, which represent specific type of SD. Internal consistency in the current study was high for preinjury ($\alpha = 0.87$), 6 month ($\alpha = 0.89$), and 12 month time points ($\alpha = 0.89$).

Internalizing behavior problems

Child Behavior Checklist (CBCL). The CBCL is a widely accepted parent-reported measure consisting of 112 items targeting various domains of behavioral functioning. The measure has demonstrated high test-retest reliability and validity for 6-18 year olds (Achenbach & Rescorla, 2001). Intraclass correlations in the .90s have been demonstrated for

inter-parent agreement, 1-week test-retest reliability, and inter-interviewer reliability. The current study utilized the scale for internalizing behavior problems.

Posttraumatic stress

Child PTSD Symptom Scale (CPSS). The CPSS is a 24-item self-report measure assessing the severity of posttraumatic symptoms and screens for diagnosis of PTSD in children exposed to trauma (see Appendix C). The scale has two parts, the first portion is a 17- item "Likert" scale corresponding to the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) symptom criteria. The second portion contains 7 items assessing functional impairment from the first 17 items. The scale has demonstrated good total symptom score internal consistency ($\alpha = .89$), test-retest reliability for symptom severity ($\alpha = .84$), and has demonstrated discriminant and convergent validity (Foa, Johnson, Feeny, & Treadwill, 2001).

Premorbid functioning

Premorbid data reflecting the child's level of functioning just prior to the injury were collected 6 weeks after injury via parent and child interview and questionnaires. Participants' history of internalizing and posttraumatic stress symptoms was obtained via clinical interviews in line with DSM-IV criteria. Level of functioning in sleep and internalizing behavior problems are provided for descriptive purposes (Table 2).

Statistical Analyses

Hypothesis 1 sought to investigate the association between injury severity and SD. The GCS was used to rate TBI severity, while the Injury Severity Score represented EI severity. Spearman's rho correlational analyses were used due to the ordinal nature of the GCS. These analyses were repeated at 6 and 12-month time points for each injury group.

For hypotheses 2 and 3, key demographic variables that may vary with sleep, such as age and socioeconomic status, were examined to determine if groups were comparable and, if necessary, they were to be added as covariates. Regarding hypothesis 2, in order to determine if the children with TBI and EI had higher levels of SD than the TD group at 6 and 12 months, a two factor mixed model analysis of variance (ANOVA) was conducted with group (TBI, EI, TD) as the between subjects factor and time (6 and 12 months) as the repeated measures factor. This analysis allowed an evaluation of whether SD was stable over time and group. This analysis sought to determine if a main effect of group existed, with planned comparisons of SD means, such that children with TBI would demonstrate significantly more SD than the EI group and children with traumatic injury would demonstrate significantly more SD than the TD children at 6 and 12 months.

To characterize specific SD types that may develop in children with TBI or EI, hypothesis 3 utilized a two factor mixed model ANOVA, with group (TBI, EI, TD) as the between subjects factor and time (6 and 12 months) as the repeated measures factor. This analysis was performed for each SD type of interest, allowing for an a priori evaluation of SD types between group and time. Considering the small sample size and exploratory nature of the hypothesis, multiple mixed model ANOVAs were preferable to multivariate analyses in order to conserve power. This analysis sought to determine if disturbances in initiating/maintaining sleep, excessive somnolence, and sleep arousal/night time awakenings were higher in children with TBI than children with EI, and higher in children with traumatic injury than TD children at 6 and 12 months. This analysis sought to determine if a main effect of group existed, with planned comparisons of SD means for each subtype. A group by time interaction was not expected for this hypothesis. In addition, a significant effect of

group among disorders of sleep hyperhidrosis, and sleep breathing disorders at both time points was not expected.

Hypothesis 4 examined the relationship between type of injury and internalizing behavior problems. This hypothesis sought to determine if both injury groups demonstrated significantly more internalizing behavior problems than TD children across both time points. In addition, this hypothesis examined whether internalizing behavior problems in children with EI would decrease by 12 months post-injury, such that these problems in children with TBI would be significantly higher at this time point. In order to investigate how TBI, EI and TD groups were associated with internalizing behavior problems over time, hypothesis 4 utilized a two factor mixed model ANOVA, with group (TBI, EI, TD) as the between subjects factor and time (6 and 12 months) as the repeated measures factor. Therefore, this analysis sought to determine if a significant group by time interaction was demonstrated, with planned comparisons of internalizing behavior problems to explore the hypothesized group differences.

Due to the study design employing multiple groups**,** hypotheses 5a and 5b called for multicategorical mediation models, allowing for the use of a multicategorical independent variable (IV; Hayes & Preacher, 2014). This model reflects a causal sequence in which an IV affects a dependent variable (DV) indirectly through a mediator variable. This method considers 4 paths (i.e., *a*, *b*, *c*, and *c'* paths) in addition to the indirect effect (*ab*). The *a* path is the effect of the predictor (X) on the proposed explanatory variable (M). The *b* path is the effect of the proposed explanatory variable (M) on the outcome (Y) while controlling for the predictor (X). The *c* path is the effect of the predictor on the outcome without controlling for the proposed explanatory variable; this is the total effect path. The *c'* path is the effect of the

predictor on the outcome while controlling for the proposed explanatory variable; this is the direct effect path. An indirect effect is the product of path *a* and path *b.* As modeled in Hayes & Preacher (2014), the multicategorical model called for k – 1 groups to be dummy coded via indicator coding, where k was equal to the number of IV categories. The TD group was not explicitly coded, meaning all k – 1 dummy variables were set to 0 for cases in that group. Thus the TD group functioned as the reference category in the analysis, and parameters in the model pertinent to group differences are quantifications relative to this reference group. Coefficients for hypothesis 5a and 5b are included in Table 5, including constant coefficient values for outcome (Y) variables in the TD group. This model allowed an analysis of SD as a potential mediator of the relation of groups to internalizing behavior problems and vice versa. Mediation models were assumed to be significant if the confidence intervals around their product did not include zero (Preacher & Hayes, 2008).

For hypothesis 5a, TBI, EI, and TD groups served as the multicategorical IV, internalizing behavior problems as the DV, and SD was entered as the mediator for 6 and 12-month time points. To investigate time precedence, another mediation model utilized group as the IV, internalizing behavior problems at 6 months as the DV, SD at 12 months as the mediator. Hypothesis 5b included TBI, EI, and TD groups as the IV, SD as the DV, and internalizing behavior problems as the mediator at 6 and 12-month time points. Additionally, another mediation model examined whether the effect of group (IV) on SD at 12 months (DV) was mediated by internalizing behavior problems at 6 months. The TD group served as a reference group in the mediational models.

To investigate posttraumatic stress sequelae of children with TBI and bodily injury, hypothesis 6 necessitated a two factor mixed model ANOVA, with group (TBI, EI) as the

between subjects factor and time (6 and 12 months) as the repeated measures factor. This analysis sought to examine whether posttraumatic stress levels were significantly higher in children with TBI than children with EI at 12 months, but not at 6 months after injury, demonstrating group by time interaction.

For hypothesis 7a and 7b, simple mediation models were used to examine chronic posttraumatic stress and SD as possible mechanisms of impairment after injury. For hypothesis 7a, TBI and EI groups served as the IV, SD was the DV, and posttraumatic stress was entered as the potential mediator at 6 and 12 months after injury. To investigate time precedence, another mediation model utilized group as the IV, SD at 12 months as the DV, and posttraumatic stress at 6 months as the mediator. This allowed an illustration of whether posttraumatic stress symptoms at 6 months account for variability in SD 12 months after injury. Hypothesis 7b included TBI and EI groups as the IV, posttraumatic stress as the DV, and SD as the mediator at 6 and 12-month time points. Additionally, another mediation model examined whether the effect of group (IV) on posttraumatic stress at 12 months (DV) was mediated by SD at 6 months.

Results

Data analyses were run using IBM SPSS Version 22 (2013). Data screening strategies and goodness-of-fit checks were conducted before analyses were run (Tabachnick, & Fidell, 2001). The dataset was screened for missing data points, and outliers were examined for inclusion or exclusion from the current study. Approximately 1 percent of the data were missing from the 12-month internalizing behavior problem scores, and such cases were omitted from the relevant analyses. The correct range of data points for each scale used in the current study was noted and there were no data entered which exceeded each scale's range. A

Winsorized mean of the preinjury SDSC values was utilized in consideration of outliers 4-8 standard deviations above previously published normative means (Bruni et al., 1996). No other data were determined to be outliers.

The distribution of the data was tested for normality to determine whether skewness and/or kurtosis were demonstrated. Skewness and kurtosis were found to be within acceptable limits for all variables. Homoscedasticity, multicollinearity, and linearity assumptions were examined. As demonstrated by Levene's Test of Equality of Error Variances and Box's Test of Equality of Covariance Matrices, the levels of reported sleep disturbances and 12-month internalizing behavior problems displayed unequal variances between groups. Therefore, Log-10 transformations were performed, and analyses were conducted before and after transformations. Because results were unchanged by the transformations, untransformed data were used for the current study.

Demographic variables of interest were examined for comparability across groups. Preinjury CBCL and SDSC scores are provided for descriptive purposes (Table 1-2). Age at initial evaluation did not differ significantly by group $F(1,84) = 0.27$, $p = .766$ and was not correlated significantly with outcomes. However, because extensive literature has demonstrated changes in the current study's outcome variables with age, mixed models were also conducted using age as a covariate. ANOVA results were unchanged by the addition of age as covariate, therefore the original mixed model ANOVA data were used for the current study. Socioeconomic status, as measured by the Hollingshead-Redlich Index, did not vary significantly by group $F(1,84) = 0.76$, $p = .470$ (Weinberg, Dietz, Penick, McAlister, 1974). Additionally, a Chi-Square Test of Independence demonstrated gender varied significantly by group, such that injury groups included more males and the TD group included more

females χ^2 (2, N = 87) = 6.97, p <.05. SD at 6 and 12 months trended towards varying by gender, with a trend toward higher SD in females $F(1,85) = 2.99$, $p = .087$; $F(1,85) = 3.78$, $p = .055$.

Hypothesis 1 sought to investigate the association between injury severity and SD. The GCS was used to rate TBI severity, while the Injury Severity Score represented EI severity. Spearman's rho correlational analysis was used, due to the ordinal nature of the GCS. These analyses were repeated at 6 and 12-month time points for each injury group. This hypothesis was only partially supported, as severity of injury was not positively associated with SD in children with TBI. Here, EI injury severity was significantly positively associated with 6 month SD but not 12 month SD (Table 3).

Group differences in SD

For all linear models, key demographic variables that may vary with sleep, such as age and socioeconomic status, were examined. Such variables were not associated significantly with outcomes; therefore, they were not added to the analyses as covariates. Regarding hypothesis 2, in order to determine SD levels across time in those with traumatic injury and those in the TD group, a two factor mixed model analysis of variance (ANOVA) was conducted with group as the between subjects factor and time (6 and 12 months) as the repeated measures factor. Hypothesis 2 was not supported, as only a significant main effect of time was shown $F(2,84) = 5.09$, $p = .027$, $\eta^2 = .06$. Here, SD in children improved between 6 and 12 months but did not differ by group (Figure 1). There was no significant effect of group $F(2,84) = 2.01$, $p = .141$, $\eta^2 = .05$ or interaction between group and time $F(2,84) = .12$, $p = .89$, $\eta^2 = .00$. Planned comparisons demonstrated the SD in the TBI group and EI group did not significantly differ $F(1,57) = .05$, $p = .820$, $\eta^2 < .00$. Given the

inconsistency in the existing evidence comparing SD in TBI and bodily injured populations, it is possible both injury groups exhibit similar disturbances in sleep. In order to investigate SD after traumatic injury during childhood more broadly, planned comparisons were performed in which both injury groups were combined. Controlling for age, children with bodily injury or head injury experienced significantly higher levels of SD than typically developing children $F(1,84) = 4.03$, $p = .048$, $\eta^2 = .05$.

To characterize specific SD types that may develop in children with TBI or EI, hypothesis 3 utilized a two factor mixed model ANOVA, with group (TBI, EI, TD) as the between subjects factor and time (6 and 12 months) as the repeated measures factor. This analysis was performed for each SD type of interest, allowing for an a priori evaluation of SD types between group and time. Disturbances of initiating/maintaining sleep, excessive somnolence, and sleep arousal/night time awakenings did not result in a significant main effect of group. Significant main effects of time were reported for disturbances of sleep transition, sleep hyperhidrosis, and a trend for disturbance of initiating/maintaining sleep, but no significant group or group by time effects were demonstrated for any of the SD subtypes (Table 4). Additionally, planned comparisons demonstrated SD subtypes did not significantly differ between TBI and EI groups, and SD subtypes in those with traumatic injury compared to those in the TD group did not demonstrate significant differences (Appendix D). Therefore this hypothesis was not supported.

Internalizing behavior problems and SD

Regarding hypothesis 4, internalizing behavior problems were expected to be elevated across time in children with TBI and were expected to decline over time in children with EI. A two factor mixed model ANOVA demonstrated this hypothesis was partially

supported, with significant main effects of group $F(2,83) = 4.36$, $p = .016$, $\eta^2 = .10$, and time $F(2,83) = 4.40$, $p = .039$, $\eta^2 = .05$ (Figure 2). Planned comparisons revealed that internalizing problems were higher in the combined TBI and EI group when compared to the TD group at both time points $F(1,83) = 8.75$, $p = .004$, $\eta^2 = .10$**.** However, the level of internalizing problems between TBI and EI groups was not significantly different at each time point $F(1,56) = .12$, $p = .730$, $\eta^2 = .00$. There was also no significant interaction between group and time in this analysis $F(2,83) = .03$, $p = .973$, $\eta^2 = .00$.

Due to the study design employing multiple groups**,** hypotheses 5a and 5b called for multicategorical mediation models, allowing for the use of a multicategorical independent variable (IV; Hayes & Preacher, 2014). This model reflects a causal sequence in which an IV affects a dependent variable (DV) indirectly through a mediator variable. This model allowed an analysis of SD as a potential mediator of the relation of groups to internalizing behavior problems and vice versa. For hypothesis 5a, TBI, EI, and TD groups served as the multicategorical IV, internalizing behavior problems as the DV, and SD as the mediator for 6 and 12-month time points. The TD group was used as a reference group, such that EI and TBI group *ab* coefficients represent value relative to the constant coefficient of the outcome variable (Y) in the TD group (Table 5). SD significantly mediated the relation between group and internalizing behaviors for the TBI group at 6 months (Figure 3). In addition, at 12 months, SD significantly mediated the relation between each injury group and internalizing behavior problems (Figure 4). To investigate time precedence, another mediation analysis demonstrated that SD at 6 months significantly mediated the relation between TBI and internalizing behavior at 12 months (Figure 5). The time precedence mediation model for the EI group was not significant. Results for hypothesis 5b demonstrated that at 6 and 12 months,

the level of internalizing behavior problems significantly mediated the relation between group and SD for children in either injury group, such that an increase in internalizing was associated with an increase in SD (Figure 6-7). Similarly, internalizing behavior problems at 6 months significantly mediated the relation between group and SD at 12 months for both injury groups (Figure 8).

Posttraumatic stress and SD

To investigate posttraumatic stress sequelae of children with TBI and bodily injury, hypothesis 6 necessitated use of a two factor mixed model ANOVA, with group (TBI, EI) as the between subjects factor and time (6 and 12 months) as the repeated measures factor. Hypothesis 6, however, was not supported, as posttraumatic stress levels did not differ significantly by group $F(2,83) = 1.01$, $p = .319$, $\eta^2 = .02$ or across time $F(2,83) = 1.44$, $p = .236$, $\eta^2 = .02$ (Table 6).

For hypothesis 7a and 7b, simple mediation models were used to examine relations between chronic posttraumatic stress and SD. For hypothesis 7a, posttraumatic stress did not significantly mediate the relation between group and SD at 6 or 12 months (Table 7). In addition, the posttraumatic stress at 6 months did not significantly mediate the relation between group and chronic SD at 12 months. Additionally, hypothesis 7b was not supported, as SD did not significantly mediate the relationship between group and posttraumatic stress significantly at 6 or 12 months. In analyzing time precedence, SD at 6 months did not significantly mediate the relation between group and posttraumatic stress significantly at 12 months.

Discussion

The aim of the current study was to compare SD in children and adolescents with TBI to those with bodily injury and typically developing children, and to better understand the relation between sleep, internalizing behavior problems, and posttraumatic stress symptoms in these groups. While limited research has compared SD among children with TBI, bodily injury, and typically developing children, no study has investigated the SD and internalizing as mechanisms of outcomes across groups. Findings suggest SD is associated with traumatic injury when compared to typically developing children. Internalizing behavior problems were significantly higher in both of our injury groups when compared to typically developing children, at both 6 and 12 month time points. In addition, sleep quality improved from 6 to 12 months.

Our results build on a previous study by Shay and colleagues (2014) investigating SD and internalizing problems following traumatic injury in early childhood. However, the current study expands on these findings by providing novel evidence pointing to a bidirectional relation between SD and internalizing problems in school-aged children and adolescents following traumatic injury. The association between TBI and internalizing problems was mediated by SD at both subacute and chronic stages of recovery. This association was further supported by the finding that SD identified 6 months after TBI predicted internalizing problems one year after injury. The links between SD and internalizing behavior problems were weaker in children with EI, in whom SD at 6 month predicted internalizing only at the one year time point. In the other direction, for both injury groups, internalizing behavior problems were associated with the level of SD across both time points. Additionally, both injury groups demonstrated linkage of internalizing problems

and SD over time, as internalizing problems at 6 months post-injury was significantly related to SD in the chronic phase.

The effect of posttraumatic stress on SD and the effect of SD on posttraumatic stress between injury groups across time were also examined. Results of the current study did not demonstrate that posttraumatic stress varied by injury type, which fits the findings of a recent review of the literature, showing that subjective factors (e.g., degree of subjective threat) rather than objective factors (e.g., injury type or severity) significantly influence the development of posttraumatic stress (Donlon & Jones, 2015). Results also indicate that the presence of SD did not significantly mediate posttraumatic stress across time, while posttraumatic stress failed to mediate SD at 6 or 12 months as well.

Taken together, the current findings expand on past work indicating the multifaceted relation between TBI and bodily injury on psychological health outcomes. Specifically, this study demonstrated a persistent bidirectional relation after injury between SD and internalizing behavior problems, particularly in children after TBI but also after bodily injury. Given the direct role SD and emotional/behavioral problems play in physical, psychological, and academic functioning of children and adolescents, current findings have strong implications for clinical management of traumatic injury populations where a growing knowledge base can help inform clinical intervention.

Group differences and SD

The relation between SD, TBI, and body injury is complex. When comparing TBI, bodily injury, and TD groups, significant group differences in SD were not demonstrated, and SD significantly decreased in all groups from 6 to 12 months. However, post-hoc analyses combining bodily injury and TBI groups evidenced significantly higher levels of SD

in the combined injury group compared to typically developing children. This supports existing research illustrating the range of functional difficulties children experience after both TBI and bodily injury, such that both injury types can result in chronic impairment (Ding et al., 2006; Ewing-Cobbs, et al., 2014; Stancin et al., 2001). For instance, negative impact on quality of life has been shown to persist one-year after bodily injury in those with lower extremity fractures (Ding et al., 2006). In regards to SD specifically, parent-reported SD was reported in 16.7% of a sample of children one to five years post-TBI; however, this was not significantly different from SD in the bodily injury group (Necajauskaite, et al., 2005). In a recent report, Shay and colleagues (2014) demonstrated group differences between bodily injury and TBI groups at 6 months but did not demonstrate differences at 12 or 18 months. Although Milroy et al. (2008) showed group differences in SD between children with TBI and with bodily injury, over half of those with bodily injury still reported SD levels above a commonly used cutoff score on their sleep measure. Thus, even in cases where significant injury group differences emerge, a substantial proportion of those in bodily injury group still report clinically relevant SD.

Multiple factors may impact whether reliable differences in SD are noted between TD, TBI and EI groups. These factors include developmental components related to sleep quality, preinjury levels of SD, method of assessment of SD, and severity of both TBI and EI. For instance, lifetime prevalence of insomnia in typically developing adolescents is greater than 10% (Owens, 2009). Thus, SD in the TD group and preinjury SD across groups may be naturally elevated, making higher SD more difficult to distinguish between groups. In regards to injury groups, based on one item from a parent-report questionnaire, Tham et al. (2012) demonstrated higher severity of and more prolonged SD in children with TBI than in a

comparison group of children with isolated fractures. Still, bodily injury and TBI groups showed SD at 3 and 12 months that increased from pre-injury baseline levels, adding evidence of SD following both types of injury. However, the use of a multiple-item scale would also help interpretation of these results. The bodily injury group in Shay et al. (2014) consisted of children with a fracture requiring one night of hospitalization via any mechanism of injury. Injury Severity Scores in the bodily injury group were generally lower than ratings in the current study, with an average of 7.05 and 10.3, respectively. Tham and colleagues (2012) did not present severity of the bodily injury group, but included only isolated fractures mostly via falls. In contrast, the current study's bodily injury group consisted of children with polytrauma, including those with multiple fractures and internal blunt-trauma injuries. It is not unreasonable to think that children with polytrauma would recover less well (Abdelgawad & Kanlic, 2011; Niedzielski, Guzikiewicz, Malecki, & Golański, 2013). Thus, variability of clinical characteristics in comparison groups may influence the characterization of SD across studies.

The relation between duration of sleep and risk-taking behaviors, including risk of injury may have also played a role in SD in the current study. For example, Koulouglioti, Cole, and Kitzman, (2008) demonstrated that young children aged 1.5-4 years old who had lower sleep duration were at higher risk for medically attended injuries. Owens, Fernando, and McGuinn (2005) reported children ages 3-7 with more frequent injuries and risk-taking behaviors had significantly more SD via parent-report than those with less frequent injuries and risk-taking behaviors. Thus, the current study may have included children with higher levels of preinjury SD or lower sleep duration, placing them at a higher risk of injury. This

research highlights the importance of considering premorbid SD, and sleep duration specifically, when analyzing relations between injury characteristics and SD.

Even though several studies have identified SD after pediatric TBI (Beebe et al., 2007; Gosselin et al., 2009, Milroy et al., 2008, Shay et al., 2014, Sumpter et al., 2013, Tham et al., 2012), there is a paucity of information regarding the aspects of sleep that are affected. Previous investigations of SD types have not used injury comparison groups with children in the 8-15 year age range, but Shay et al., (2014) did find 3-6 year olds with TBI to have higher bedtime resistance (sleep transition problems) than children after bodily injury. The current study, along with the report by Shay and colleagues (2014), is the only investigation of this area to include a validated sleep measure that assesses multiple types of sleep quality while using an injury comparison group. In the present study, significant group differences among each SD type were not observed. Disturbances of sleep hyperhidrosis and sleep transition across the sample significantly improved from 6 to 12 months, while difficulty initiating and maintaining sleep trended towards improving over time. Thus, positive change among all three groups illustrates the complex pattern of SD type after injury in school-aged children and warrants further research.

Findings in other developmental populations may inform future exploration of SD types in children after traumatic injury. Romeo and colleagues (2014) studied SD in children with cerebral palsy, who exhibited disorders of initiating and maintain sleep, sleep transition, breathing, and excessive somnolence. A recent review of neurodevelopmental disorders reported a high prevalence of decreased sleep and difficulties initiating and maintaining sleep in that population (Picchioni, Reith, Nadel, & Smith, 2014). Disorders of sleep arousal and excessive somnolence are also prevalent in children with juvenile rheumatoid arthritis or

migraines (Bruni et al., 1997; Wolfson & Carskadon, 1998). Similarly, these SD types may be of interest in future studies of children following traumatic injury.

The current study is the first to investigate SD in TBI, bodily injury, and typically developing comparison groups using a validated sleep questionnaire examining multiple dimensions of sleep behaviors. Results partially support existing literature, showing higher SD in children following traumatic injury than in typically developing children. In contrast to the limited extant research utilizing injured comparison groups, the current sample did not show that children with TBI experience higher SD than those with EI. Given the sparse number of published studies using injury and typically developing comparison groups, one reason for the current findings may simply be that children with bodily injury develop a similar course of SD as children with TBI. Another reason for this finding may be that a majority of this sample's TBI group suffered more mild head injuries (Table 1). Although findings vary, more recent and methodologically rigorous work has supported the idea that more severe TBI is associated with higher SD in children (Beebe et al., 2007; Shay et al., 2014, Sumpter et al., 2013). This emphasizes the importance of investigating both type of injury along with severity of injury when studying SD in children.

Injury severity and SD

The relation between severity of injury to the body or brain and SD is complex, and positive association was partially supported in the current study. Injury severity in the EI group was positively associated with SD at 6-months but not at 12 months post-injury. In contrast to previous studies, SD was not associated with level of TBI severity (Beebe et al., 2007; Osorio et. al., 2013; Shay et al., 2014; Tham et al., 2012). Inconsistencies in the literature regarding injury severity and sleep quality may be due to several factors, including

methods of assessing TBI severity and SD, as well as the time interval after injury when sleep behaviors are assessed. For example, measurement of severity in the current study relied on the GCS score. Though commonly used, the GCS can be limited by other factors, such as patient sedation or hours assessed post-injury (Teasdale et al., 2014). Including additional clinical markers of severity, such as neuroimaging findings, may help better assess sleep outcomes in relation to TBI severity.

Past studies on SD after TBI in children have used various methods to categorize injury severity, and results have been inconsistent. For instance, Shay et al (2014) separated TBI severity into complicated mild/moderate and severe groups, reporting higher SD in the severe TBI group at 18 months while only the combined mild/moderate TBI group reported significantly more at 6 months. It may be that children with severe TBI and their families are more concerned with other significant areas of impairment in functioning early on, while the mild/moderate TBI group is less impaired overall at 6 months and thus better able to attend to sleep disturbances if they occur. In a study following school-aged children and adolescents, Tham and colleagues (2012) grouped their sample into three levels of mild TBI and used GCS motor scores to group moderate and severe TBI, finding a complex relation where mild TBI was most predictive of SD over time, yet moderate or severe TBI was associated with higher SD 24 months post injury. A report of SD within the first 6 months of TBI revealed that parents rated 51% of adolescents with moderate-to-severe TBI and 22% of those with complicated mild TBI to have significant daytime somnolence (Osorio et al., 2013). The differential associations between level of injury severity and amount of SD demonstrated by Tham et al. (2012), Shay et al. (2014), and others, highlight the current lack of consensus and the need for more research. The variety of classifications used and variable time points

children have been evaluated post-injury in past research has complicated comparisons across studies. Given the multifaceted impact SD demonstrates particularly across development, the profile of injury type, TBI severity, and associated SD in children warrants further research.

Group differences in internalizing behavior problems

Children in both TBI and EI groups reported reliably more internalizing problems than TD children (Figure 2). This finding is in line with past work demonstrating greater internalizing problems following TBI when compared to preinjury functioning (Anderson et al., 2012; Max et al, 2011; Peterson et al., 2013). However, children with TBI and those with EI in the current study did not differ in their level of internalizing problems. Interesting, internalizing T-scores were non-significantly higher in the EI group than the TBI group at 12 months, and T-scores across time and group remained in the average clinical range, suggesting elevations may be subclinical in severity (Table 2). Our findings contrast from prior literature reporting that children with TBI suffer from higher levels of internalizing behavior problems than children with bodily injury. For instance, higher parent-reported internalizing problems were found in children with TBI than bodily injury at 6 and 12 months (Kirkwood et al., 2000). However, the study's TBI group consisted of children with moderate and severe TBI only, whereas the current study's sample also included children with mild TBI. Investigating severity and internalizing relations further, Luis & Mittenberg (2002) reported that children with a new-onset mood or anxiety disorder following TBI tended to have lower GCS, indicating more severe injury, than children without a new-onset disorder. Despite these findings, few studies have utilized injury comparison groups when investigating internalizing behavior problems after pediatric TBI. Thus, future investigations

would benefit from comparing internalizing problems across injury type, considering TBI severity level, and including multiple time points following injury.

Overall, the current study demonstrates that following traumatic injury, children demonstrate higher levels of internalizing behavior problems in early and chronic stages when compared to problems in typically developing children. Though internalizing behaviors in the sample decreased significantly over time, they still remained higher in injured groups when compared to typically developing children. Such findings reinforce previous literature demonstrating internalizing behavior problems in children after TBI and bodily injury, yet premorbid internalizing problems should also be considered in future analyses (Peterson et al., 2013; Zatzick et al., 2008). Importantly, factors contributing to elevated internalizing problems, including SD, were not investigated in these studies, and remain understudied.

Mechanisms of action for SD and internalizing behavior problems

In regards to exploration of mechanisms of action between the effects of SD and internalizing behavior problems, multiple models were utilized. Results demonstrated that the effect of internalizing behavior problems at 6 and 12 months was fully mediated by SD in the TBI group (Figures 3 & 4). Additionally, SD in the EI group partially mediated the effect of internalizing problems at 12 but not 6 months, suggesting SD is associated with internalizing problems in the chronic phase after bodily injury. Investigating the pathway in reverse, the effect of SD was fully mediated by internalizing behavior problems at 6 and 12 months for both injury groups (Figures 6 & 7). This supports the idea that children with internalizing behavior problems after traumatic injury are at risk for developing SD.

Investigating time precedence, the TBI group showed SD at 6 months fully mediated internalizing behaviors at 12 months (Figure 8). Thus, in comparison to typically developing

children, those with TBI suffering from early SD may be at risk for developing chronic internalizing behavior problems (Figure 5). Conversely, both injury groups displayed time precedence in the other direction, with the effect of SD at 12 months being fully mediated by internalizing at 6 months (Figure 8). In sum, results provide evidence that internalizing behavior problems occur across injury type, persist over time, and that injured children with such problems are more likely to develop SD than typically developing children.

The current study provides evidence that in the early and chronic stages, SD plays a role in the development of internalizing behavior problems in children after TBI when compared to typically developing children. These findings advance recent evidence provided by Shay and colleagues (2014), illustrating that children with higher SD, across healthy, bodily injury, and TBI groups, had higher internalizing behavior problems. Notably, the study by Shay et al. (2014) is the only published study at this time that has examined the relation of SD and internalizing behavior problems in children after TBI. However, that study did not explore the reverse pathway of whether those with internalizing problems were at risk for SD. When investigating that pathway, our study suggests internalizing behavior problems in children with TBI and children with bodily injury put them at risk for SD, in comparison to typically developing children.

These findings support the presence of a bidirectional relation between SD and internalizing behavior problems after traumatic injury. Moreover, results showed that although this relation persists over time in both injury groups, it was more consistently demonstrated in children following TBI. Specifically, internalizing problems presented as a mechanism of action for SD across both injury groups, while SD was primarily a mechanism of action in children with TBI. Existing evidence demonstrates a predictive impact of

internalizing disorders on SD, although this link has not yet been extensively studied in children following traumatic injury (Alfano, Ginsburg, & Kingery, 2007; McMakin & Alfano, 2015; Gregory, Eley, O'Connor, & Plomin, 2004; Ong, Wickramaratne, Min, & Weissman, 2006). Results indicate that in children with TBI, SD may lead to or worsen internalizing behavior problems, while internalizing in those with TBI or bodily injury may lead to SD. Findings support the current body of literature suggesting early adolescence to be a critical developmental period regarding the interaction between sleep and anxiety (McMakin & Alfano, 2015).

Current research does not present a clear basis that would explain why SD after bodily injury was a significant mechanism of internalizing behavior problems at 12 months but not at 6 months. It may be that a lower level of parental concern for, or focus on, internalizing problems after bodily injury is present at 6 months, with functional limitations being a primary parent-reported source of distress at that time period (Stancin et al, 2001). Those with persisting SD at 12 months after bodily injury may also be a more high-risk group with differing premorbid internalizing or higher subjective reactions to stressors. Such characteristics have been shown to predict internalizing behavior problems and other negative outcomes after injury and traumatic events (Marsac et al., 2014; McMakin & Alfano, 2015; Yeates et al., 1997; Zatzick et al., 2008a; Zatzick et al., 2008b).

Posttraumatic stress and SD

Although a growing number of studies have been published investigating SD and posttraumatic stress in children, research has not extensively explored this relation in children with traumatic injury. The current study compared posttraumatic stress symptoms between children with TBI and those with bodily injury, but did not find significant

differences between groups or changes over time (Table 6). Past research has shown posttraumatic stress symptoms occur after TBI and after bodily injury (O'Connor et al., 2012; Zatzick, et al., 2006; Zatzick et al., 2008b). It may be that posttraumatic symptoms follow a similar course among injury types at early and chronic stages, such that injury types can present a similar course of posttraumatic stress. Current literature demonstrates subjective level of distress and fear to be associated with the development of posttraumatic stress, yet less evidence shows objective factors such as injury type and severity to significantly impact the level of posttraumatic stress (Donlon & Jones, 2015; Kassam-Adams & Winston, 2004; Langeland & Olff, 2008; Nugent, Christopher, and Delahanty, 2006; Trickey, Siddaway, Meiser-Stedman, Serpell, & Field, 2012). Thus, investigating posttraumatic stress in children may be most informative via subjective markers of distress rather than injury type.

Investigation of mechanisms of action called for exploration of mediating factors between injury groups. As shown in Table 7, however, all mediation pathways of posttraumatic stress were non-significant. Results showed the effects of SD at 6 and 12 months were not significantly mediated by posttraumatic stress symptoms, while the effect of posttraumatic stress at 6 and 12 months was not mediated by SD. In regards to time precedence, the effect of SD at 12 months was not mediated by posttraumatic stress at 6 months. In contrast to these results, two studies of the same sample of children following motor vehicle accidents reported that nightmares and time to sleep onset 10 days after injury predicted self-reported posttraumatic stress 2-6 months later (Wittmann, et al., 2010; Wittmann et al., 2012). However, these studies did not include a control group and did not separate children with head injury and bodily injury in their sample. Similarly, a brief report

of adults who suffered from motor vehicle accidents showed sleep complaints at 1 month predicted posttraumatic stress disorder diagnosis at 12 months (Koren, Arnon, Lavie & Klein, 2002). Although such literature shows earlier SD predicts later posttraumatic stress, SD was assessed much earlier in these investigations than in the current study. It is possible that acute status of SD, rather than SD at 6 months, is a better marker for the development of later posttraumatic stress.

In addition to linking early SD to posttraumatic stress, past research has also shown early distress, including internalizing symptoms, to predict later posttraumatic stress in children and adolescents following injury (Marsac et al., 2014; Kassam-Adams & Winston, 2004; Zatzick et al., 2006). For example, a study of hospitalized adolescents showed higher posttraumatic and depressive symptoms at the time of injury to be associated with increased posttraumatic stress at 5 and 12 month follow up (Zatzick et al., 2006). Physiological arousal, assessed via heart rate and cortisol levels, has also been linked to the persistence of posttraumatic stress in injured children, adding to the biopsychosocial framework of these mechanisms (Kassam-Adams, Garcia-España, Fein, & Winston, 2005; Ostrowski, Christopher, van Dulmen, & Delahanty, 2007). Published research linking the physiological pathways to posttraumatic stress after injury is not extensive. Thus, future work can investigate the acute psychological and physiological reaction following injury as possible mediators of later posttraumatic stress and SD.

Factors such as the method of SD measurement likely contribute to the variability in current literature. Recently, Spilsbury et al., (2014) found no relation between parent-reported sleep and posttraumatic stress symptoms within the first 3 months. However, child-reported increase in SD was associated with posttraumatic stress levels in the same study,

and change in posttraumatic stress was also associated with child-reported SD. To optimize validity of results, future work can include parent and self-report measures, and consider mechanism of injury when comparing research across studies. However, considering the sparse number of studies and variability in methods used, there is a strong need for additional multi-method studies comparing SD and posttraumatic stress in children.

Limitations and Future Directions

The current study is limited by several factors. Utilizing three groups as a methodological strength for group comparisons also hindered obtaining necessary power for the study hypotheses. A larger sample size would also help to preserve more statistical power, especially in regards to investigations of posttraumatic stress, which excluded the typically developing, non-trauma exposed, comparison group. Given that preinjury characteristics have been associated with post-injury outcomes, their consideration would strengthen our interpretations and allow for longitudinal methods of analysis. However, this also can introduce retrospective bias, as data on injury groups' premorbid status rely on backdated parent-report while children are in the acute recovery stage. Still, SD and sleep duration levels have been associated with higher risk of injury as well as internalizing behavior problems, which highlights the importance of considering preinjury functioning (Koulouglioti et al., 2008; Owens et al., 2005).

Measures utilized in the current study are not without limitations. Regarding outcome measures used, gold standard sleep assessment is typically considered to be both objective and subjective measures. Therefore, including polysomnography and/or actigraphy assessment, along with parent and child report would provide a more comprehensive view of SD (Willis & Gregory, 2015). Measurement of TBI severity would benefit from the use of

other clinical markers in addition to GCS scores, such as neuroimaging findings. Internalizing problems were assessed using parent-report questionnaires, while ideal assessment of psychological functioning would include a clinical diagnostic interview as well. Developmental changes also present added challenges to research on children and adolescents. For instance, Dahl and colleagues (2009) found that pubertal changes in sleep precede physical changes in puberty, therefore future studies would benefit from accounting for current pubertal stage of children. In addition to age at injury, stratifying samples by pubertal stage can help control for developmental changes that have been shown to impact psychological and behavioral outcomes.

Conclusion

The current study broadens a growing base of literature on SD following pediatric TBI and bodily injury. Importantly, study design included injury and non-injury comparison groups and investigated potential mechanisms of relevant outcomes. Results indicated children who have suffered from TBI or bodily injury report higher levels of SD than typically developing children, and this pattern persists in the chronic stage. This study reinforces evidence that children with TBI and bodily injury have higher levels of internalizing behavior problems, and expands on past work by showing such problems mediate SD in the subacute and chronic stages. Importantly, results show that primarily in children with TBI, but also in those with bodily injury, SD mediates the level of internalizing problems at subacute and chronic stages. The current study also provides support that early internalizing problems across both injury groups serve as a mediator for chronic SD, and in the other direction, early SD mediates chronic internalizing problems in children with TBI.

This temporal relation among outcomes reinforces the clinical need for early screening and intervention after traumatic injury.

It is well-established that SD in children negatively impacts a range of mental and physical health domains, including: declining cardiovascular health, dysregulation of metabolism contributing to obesity, dysregulation of stress and arousal systems, impaired cognitive/academic functioning, and difficulties in socio-emotional development (Alfano, Zakem, Costa, Taylor, & Weems, 2009; Amin et al., 2002; Dahl, 1996; El-Sheikh & Buckhalt, 2015). This evidence highlights the public health risk that SD and related psychological problems pose. In turn, the findings of the current study demonstrate those with traumatic injury suffer from sleep and behavioral problems. Notably, more than 9.2 million children are treated in emergency departments for nonfatal injuries per year, while 1.36 million are admitted due to TBI. Investigating chronic problems occurring after TBI and bodily injury remains a critical public health issue (Borse et al, 2008, Faul et al., 2010; Keenan & Bratton, 2006). By investigating this substantial health concern in a high-risk post-traumatic injury population, the current study expands upon limited evidence and demonstrates a stark need for further methodologically rigorous research. Results have strong implications for early and chronic clinical management of TBI and bodily injury, such that a comprehensive acute and post-acute evaluation should include questions screening for SD and internalizing problems. By improving early identification of SD and psychological problems after injury, evidence-based treatment methods stand a better chance at successful intervention for an at-risk population.

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Table 1

Demographics and Clinical Characteristics

	TBI (n = 36)	EI (n = 24)	TD (n = 27)
Age at baseline in years, M*(SD)*	12.2 *(2.4)*	11.7 (2.4)	11.9 (2.7)
Months from injury to baseline, M *(SD)*	1.5 *(0.7)*	1.7 *(0.8)*	
Males, *n (*%)	63.9%	70.8%	37.0%
Hollingshead, M *(SD)*	38.8 *(14.8)*	34.6 *(14.6)*	38.8 *(12.9)*
Race/Ethnicity			
African American	11.1%	20.8%	25.9%
Asian	2.8%	0.0%	3.7%
Caucasian	30.6%	25.0%	14.8%
Hispanic/Latino	50.0%	54.2%	40.7%
Mixed ethnicity	5.6%	0.0%	14.8%
Injury characteristics			
GCS score distribution			
3-8	30.6%		
9-12	8.3%		
13-15	61.1%		
ISS score, M (*SD*)	17.7 *(11.8)*	10.3 *(6.8)*	

Note. TBI=traumatic brain injury, EI=extracranial injury, TD=typically developing, M = mean, *SD* = standard deviation, GCS = Glasgow Coma Scale, ISS = Injury Severity Score. *$p < .05$

Table 2

Premorbid and chronic SD and internalizing outcomes

Measure	Typically developing	Extracranial injury	Traumatic brain injury
Premorbid SDSC M(*SD*)	37.7(6.6)	39.3(9.3)	41.9(10.4)
6M SDSC M(*SD*)	37.7(8.4)	43.3(15.8)	43.0(14.1)
12M SDSC M(*SD*)	35.4(6.3)	41.1(16.5)	39.7(10.6)
Premorbid Int M(*SD*)	50.9(9.2)	53.0(12.9)	52.7(12.6)
6M Int M(*SD*)	47.3(9.6)	54.8(12.9)	54.6(13.8)
12M Int M(*SD*)	45.5(7.5)	54.1(13.3)	52.7(13.0)

Note. Preinjury, 6, and 12 month outcomes are provided. 6M = 6 month, 12M = 12 month, M = Mean, *SD* = Standard deviation, CBCL = Child Behavior Checklist, Int = Internalizing behavior problems, SDSC = Sleep Disturbances Scale for Children.

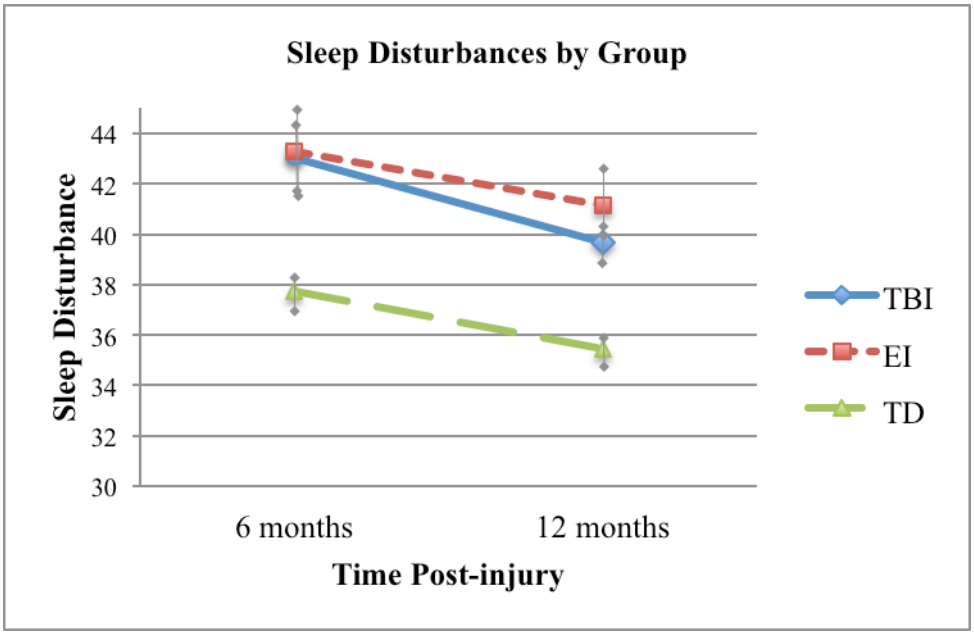
Table 3

Correlations between Sleep Disturbance and Injury Severity

Measure	1	2	3	4
1. GCS Score	-			
2. ISS Score	-.560	-	.445*	.272
3. 6 month SD	.066	.181	-	.746**
4. 12 month SD	.164	-.010	.839**	-

Note. Correlation coefficients below the diagonal denote traumatic brain injury group, while those above the diagonal denote those with extracranial injury. * = $p<.05$, ** = $p<.01$, GCS = Glasgow Coma Scale, ISS = Injury Severity Score, SD = sleep disturbance.

Figure 1



Note. A two factor mixed model analysis of variance was conducted with group as the between subjects factor and time (6 and 12 months) as the repeated measures factor. Only a main effect of time was shown, with sleep disturbances in the sample improving between 6 and 12 months. There was no significant effect of group or interaction between group and time, and sleep disturbances between TBI and EI groups were not significantly different from each other. Sleep disturbances in the combined injury group were significantly higher than the typically developing group. TBI = traumatic brain injury, EI = extracranial injury, TD = typically developing.

Table 4

Sleep disturbance type by group

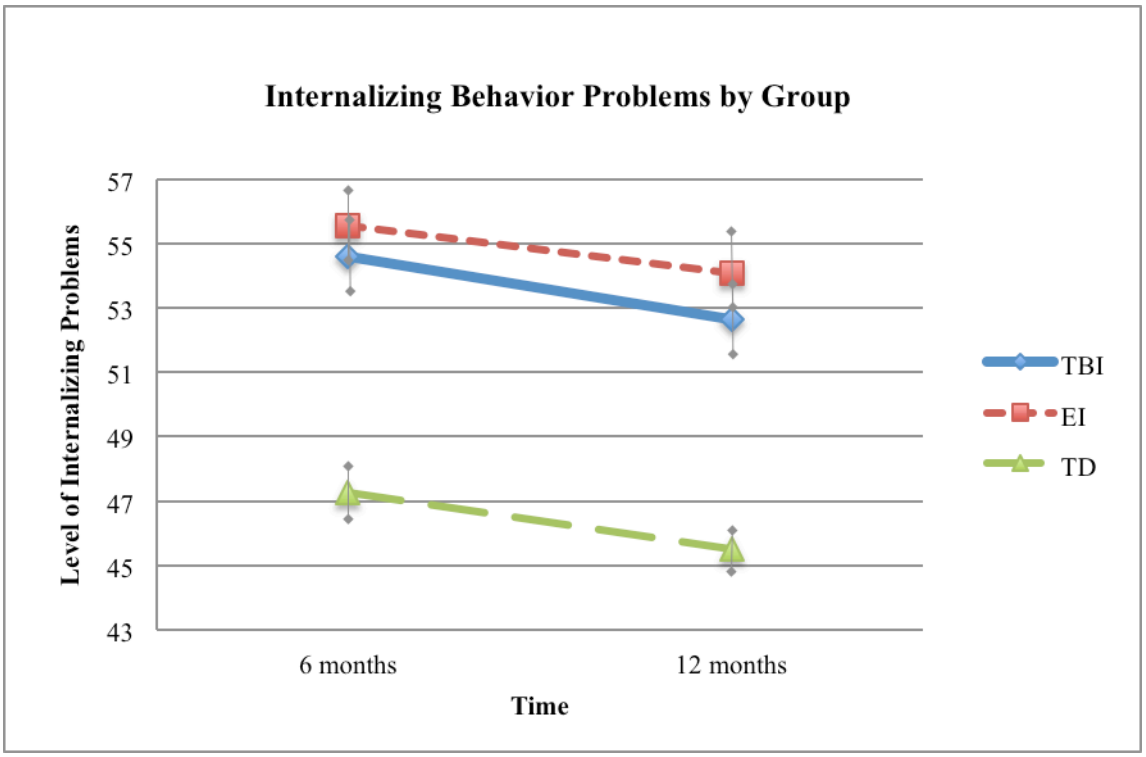
	Traumatic brain injury		Extracranial injury		Typically developing	
M*(SD)*	6 month	12 month	6 month	12 month	6 month	12 month
Insomnia	13.78*(5.82)*	12.83*(4.75)*	13.13*(5.18)*	12.29*(4.91)*	11.7*(3.72)*	11.07*(2.60)*
Hypersomnia	8.14*(4.04)*	7.81*(3.15)*	8.67*(4.18)*	8.5*(4.24)*	7.11*(2.11)*	6.78*(1.63)*
Night time awakening	3.83*(1.95)*	3.25*(.65)*	3.71*(1.71)*	3.67*(2.06)*	3.33*(.92)*	3.37*(1.36)*
Sleep breathing	4.36*(1.71)*	4.19*(2.05)*	4.33*(1.95)*	4.33*(2.65)*	4.22*(1.05)*	3.89*(1.05)*
Sleep transition	10.14*(4.45)*	9.06*(3.70)*	10.12*(4.92)*	9.63*(4.29)*	8.85*(3.39)*	8.00*(2.39)*
Sleep hyperhydrosis	2.78 (*1.62)*	2.53 *(1.06)*	3.33*(2.41)*	2.71*(1.71)*	2.48*(1.01)*	2.3 *(.61)*

	Group F*(p)*	Time F*(p)*	Group by time F*(p)*
Insomnia	1.59*(.211)*	3.61*(.061)*	.05*(.950)*
Hypersomnia	1.76*(.179)*	1.18*(.280)*	.04*(.957)*
Night time awakening	.50*(.611)*	1.00*(.320)*	1.12*(.332)*
Sleep breathing	.24*(.786)*	.64*(.425)*	.19*(.825)*

Sleep transition	1.22*(.300)*	5.65*(.020)*	.25*(.781)*
Sleep hyperhydrosis	1.55*(.219)*	4.86*(.030)*	.66*(.517)*

Note. Mixed model analysis of variance results for each type of sleep disturbance, demonstrating mean sleep scores and standard deviations for each type, as well as F-value and p-values for each model. A main effect of time was reported for disturbances of sleep transition, sleep hyperhidrosis, and a trend for insomnia, but no group or group by time effects were demonstrated for any of the subtypes.

Figure 2



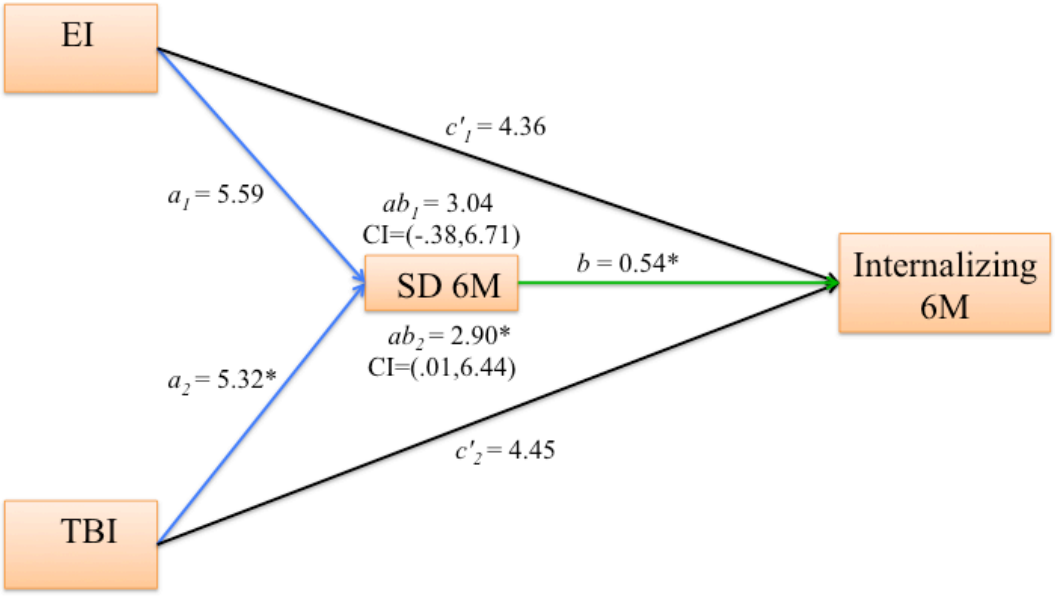
Note. A mixed model analysis of variance demonstrated significant effects of group and time. Specifically, internalizing behavior problems in the sample decreased between 6 and 12 months, while problems in the traumatic brain injury and extracranial injury group were significantly higher than those in the typically developing group at each time point. Internalizing problems between TBI and EI groups were not significantly different from each other. TBI = traumatic brain injury, EI = extracranial injury, TD = typically developing.

Table 5
Multicategorical mediation models of sleep disturbance and internalizing behavior problems

Y	Model	Y Constant for TD Group	Indirect Effect *ab*(LLCI,ULCI)	Direct Effect c'(LLCI,ULCI)
1	*a* = Group→6M SD *b* = 6M SD→6M Int *c′* = Group→6M Int	6M Int = 47.26	EI group *3.04*(-.38,6.71) TBI group *2.90*(.01,6.44)	EI group *4.36*(-1.38,10.10) TBI group *4.45*(-.76,9.67)
2	*a* = Group→12M SD *b* = *12*M SD→12M Int *c′* = Group→12M Int	12M Int = 45.52	EI group *3.47*(.19,8.26) TBI group *2.32*(.23,5.20)	EI group *5.10*(-.61,10.81) TBI group *4.83*(-.24,9.90)
3	*a* = Group→6M SD *b* = 6M SD→12M Int *c′* = Group→12M Int	12M Int = 45.52	EI group *2.51*(-.18,5.73) TBI group *2.26*(.05,5.18)	EI group *6.06*(.18,11.94) TBI group *4.89*(-.39,10.16)
4	*a* = Group→6M Int *b* = 6M Int→6M SD *c′* = Group→6M SD	6M SD = 37.7	EI group *4.57*(.95,9.39) TBI group *4.53*(1.13,9.37)	EI group *1.02*(-5.16,7.21) TBI group *.79*(-4.85,6.43)
5	*a* = Group→12M Int *b* = 12M Int→12M SD *c′* = Group→12M SD	12M SD = 35.4	EI group *4.53*(1.25,10.18) TBI group *3.78*(1.11,8.32)	EI group *1.84*(-3.88,7.56) TBI group *.48*(-4.62,5.58)
6	*a* = Group→6M Int *b* = 6M Int→12M SD *c′* = Group→12M SD	12M SD = 35.4	EI group *3.53*(.70,7.81) TBI group *3.51*(.88,7.83)	EI group *2.19*(-3.52,7.90) TBI group *.75*(-4.45,5.96)

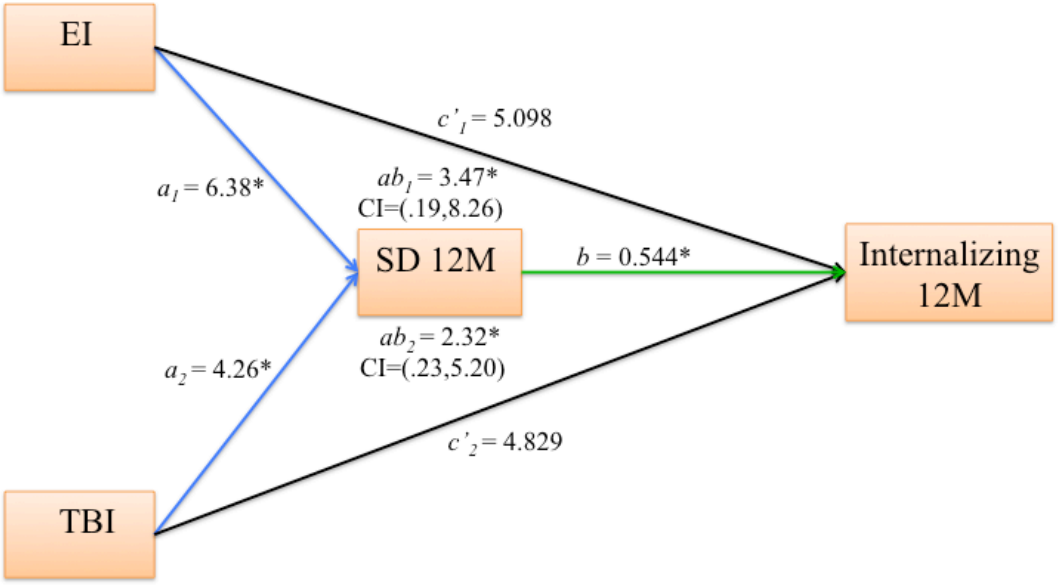
Note. Mediation model coefficients and confidence intervals for direct (*c'*) and indirect effects (*ab*) are shown, examining the relation between sleep disturbance and internalizing behavior problems. TBI = traumatic brain injury, EI = extracranial injury, SD = sleep disturbance, Int = Internalizing behavior problems, 6M = 6 month, 12 = 12 month, ULCI = upper bound, LLCI = lower bound of a 95% confidence interval. The 95% confidence interval for the indirect effects are obtained through bootstrapping with 10,000 re-samples.

Figure 3



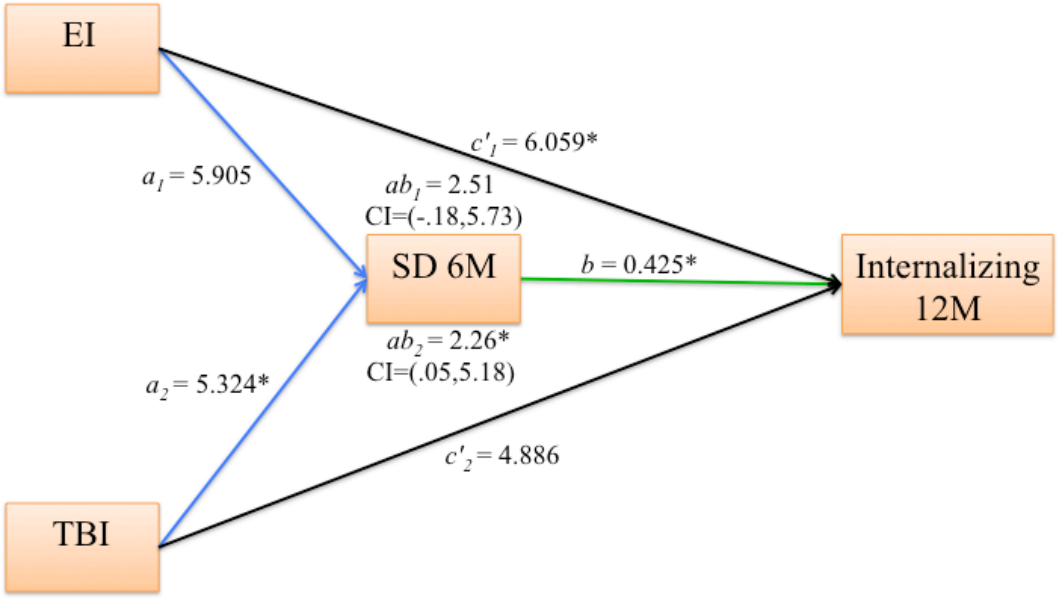
Note. Multicategorical mediation model showing the direct (*c'*) and indirect effects (*ab*) of traumatic brain injury and extracranial injury on internalizing behavior problems at 6 months. Results showed sleep disturbance at 6 months mediated the relation between traumatic brain injury and internalizing problems at 6 months. Sleep disturbance did not mediate the relations between extracranial injury and internalizing behavior problems. EI = extracranial injury, TBI = traumatic brain injury, SD = sleep disturbance, 6M = 6 month, 12 = 12 month, CI = confidence interval, path ab_1 = indirect effect of EI on internalizing, path ab_2 = indirect effect of TBI on internalizing. Confidence intervals for both indirect effects are shown. The 95% confidence interval for the indirect effects were obtained through bootstrapping with 10,000 re-samples. * $p<.05$.

Figure 4



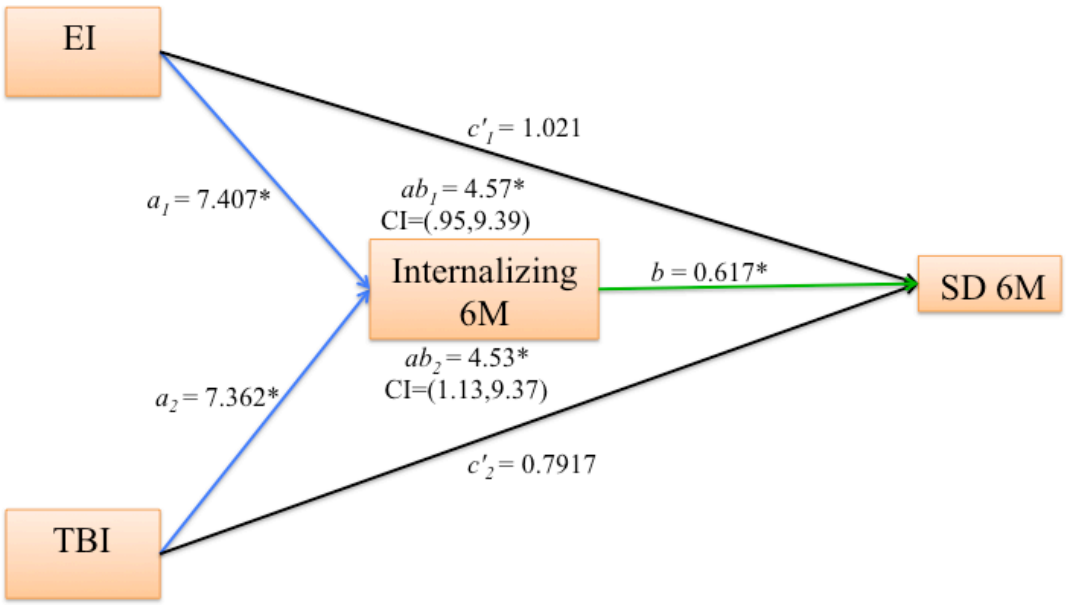
Note. Multicategorical mediation model showing the direct (*c'*) and indirect effects (*ab*) of traumatic brain injury and extracranial injury on internalizing behavior problems at 12 months. Results showed sleep disturbance at 12 months mediated the relation between group and internalizing problems at 12 months for each injury group. EI = extracranial injury, TBI = traumatic brain injury, SD = sleep disturbance, 12 = 12 month, CI = confidence interval, path ab_1 = indirect effect of EI on internalizing, path ab_2 = indirect effect of TBI on internalizing. Confidence intervals for both indirect effects are shown. The 95% confidence interval for the indirect effects were obtained through bootstrapping with 10,000 re-samples. * $p<.05$.

Figure 5



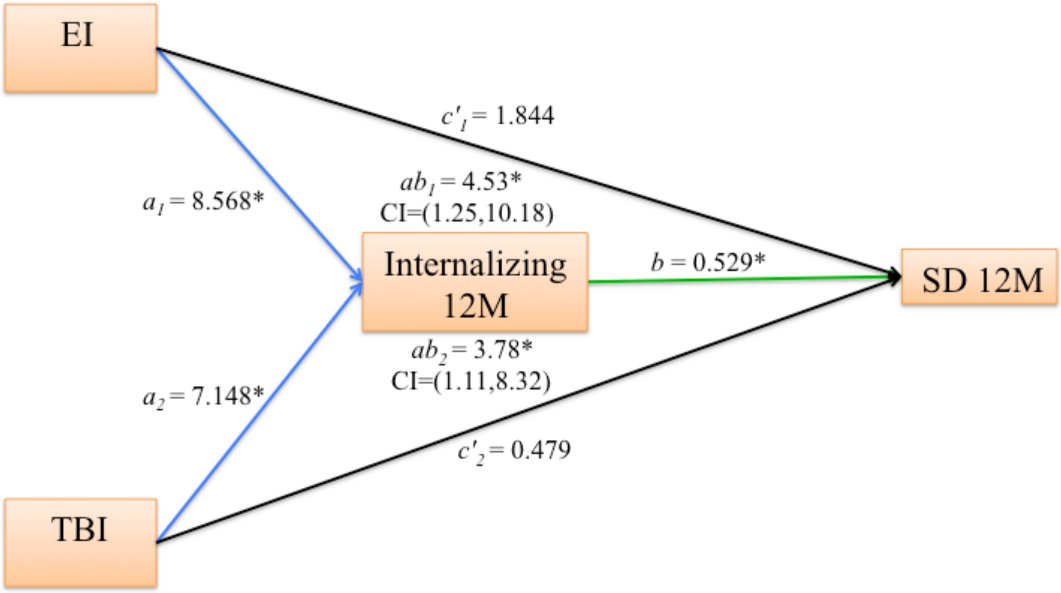
Note. Multicategorical mediation model showing the direct (*c'*) and indirect effects (*ab*) of traumatic brain injury and extracranial injury on internalizing behavior problems at 12 months. Results showed sleep disturbance at 6 months mediated the relation between traumatic brain injury and internalizing problems at 12 months. Sleep disturbance did not mediate the relation between extracranial injury and internalizing behavior problems. EI = extracranial injury, TBI = traumatic brain injury, SD = sleep disturbance, 6M = 6 month, 12M = 12 month, CI = confidence interval, path ab_1 = indirect effect of EI on internalizing, path ab_2 = indirect effect of TBI on internalizing. Confidence intervals for both indirect effects are shown. The 95% confidence interval for the indirect effects were obtained through bootstrapping with 10,000 re-samples. * $p<.05$.

Figure 6



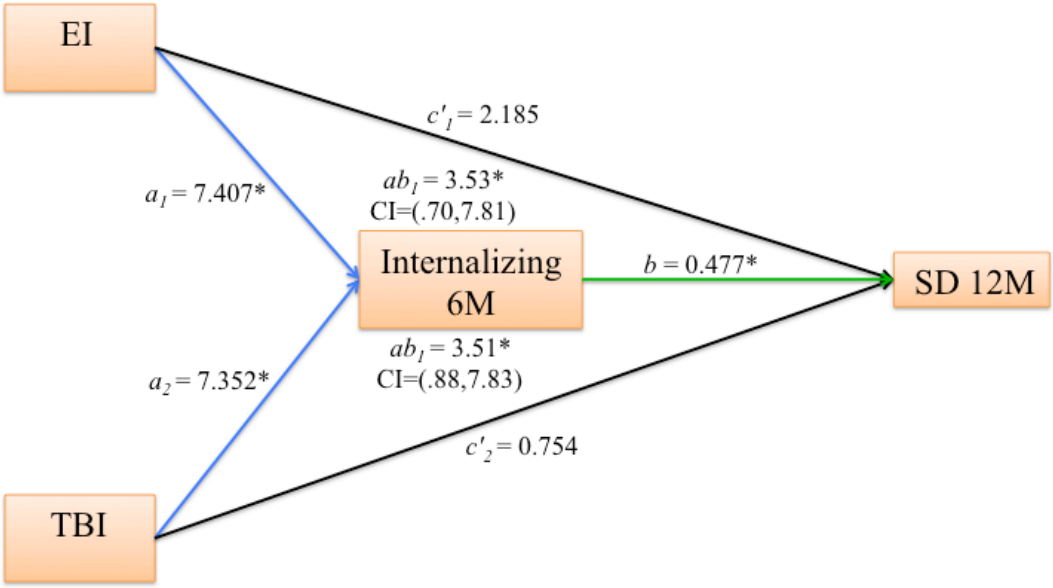
Note. Multicategorical mediation model showing the direct (*c'*) and indirect effects (*ab*) of traumatic brain injury and extracranial injury on sleep disturbance at 6 months. Results showed internalizing behavior problems at 6 months mediated the relation between group and sleep disturbances at 6 months for both injury groups. EI = extracranial injury, TBI = traumatic brain injury, SD = sleep disturbance, 6M = 6 month, CI = confidence interval, path ab_1 = indirect effect of EI on internalizing, path ab_2 = indirect effect of TBI on internalizing. Confidence intervals for both indirect effects are shown. The 95% confidence interval for the indirect effects were obtained through bootstrapping with 10,000 re-samples.
* $p<.05$.

Figure 7



Note. Multicategorical mediation model showing the direct (*c'*) and indirect effects (*ab*) of traumatic brain injury and extracranial injury on sleep disturbance at 6 months. Results showed internalizing behavior problems at 12 months mediated the relation between group and sleep disturbances at 12 months for both injury groups. EI = extracranial injury, TBI = traumatic brain injury, SD = sleep disturbance, 12M = 12 month, CI = confidence interval, path ab_1 = indirect effect of EI on internalizing, path ab_2 = indirect effect of TBI on sleep disturbance. Confidence intervals for both indirect effects are shown. The 95% confidence interval for the indirect effects were obtained through bootstrapping with 10,000 re-samples. * $p<.05$.

Figure 8



Note. Multicategorical mediation model showing the direct (*c'*) and indirect effects (*ab*) of traumatic brain injury and extracranial injury on sleep disturbances at 12 months. Results showed internalizing behavior problems at 6 months mediated the relation between traumatic brain injury and sleep disturbance at 12 months. EI = extracranial injury, TBI = traumatic brain injury, SD = sleep disturbance, 6M = 6 month, 12 = 12 month, CI = confidence interval, path ab_1 = indirect effect of EI on internalizing, path ab_2 = indirect effect of TBI on internalizing. Confidence intervals for both indirect effects are shown. The 95% confidence interval for the indirect effects were obtained through bootstrapping with 10,000 re-samples. * p<.05.

Table 6

Posttraumatic stress after injury

	CPSS scores, M*(SD)*		Statistics, F*(p)*		
	6 month	12 month	Group	Time	Group by time
Traumatic brain injury	10.50*(10.83)*	10.67*(10.34)*	1.01*(.319)*	1.44*(.236)*	1.86*(.178)*
Extracranial injury	9.50(*8.51)*	6.92*(8.10)*			

Note. Mixed model analysis of variance results comparing posttraumatic stress scores between groups at 6 and 12 months. Results did not demonstrate a main effect of group, time, or group by time interaction. CPSS = Child PTSD Symptom Scale, M = mean, SD = standard deviation, F = F-value, *p* = p-value.

Table 7

Mediation models of posttraumatic stress and sleep disturbance

Y	Model	Indirect Effect *B*(LLCI,ULCI)	Direct Effect *B*(LLCI,ULCI)
1	*a* = Group-->6M PTS *b* = 6M PTS-->6M SD *c′* = Group-->6M SD	*.599*(-2.51,3.81)	-*.863*(-8.10,6.37)
2	*a* = Group-->12M PTS *b* = *12*M PTS-->12M SD *c′* = Group-->12M SD	*.1.77*(-.09,5.54)	*3.22*(-9.97,3.53)
3	*a* = Group-->6M PTS *b* = 6M PTS-->12M SD *c′* = Group-->12M SD	*.404*(-1.39,3.30)	*1.863*(-8.57,4.85)
4	*a* = Group-->6M SD *b* = 6M SD-->6M PTS *c′* = Group-->6M PTS	-*.072*(-3.06,1.64)	*1.071*(-3.79,5.93)
5	*a* = Group-->12M SD *b* = 12M SD-->12M PTS *c′* = Group-->12M PTS	-*3.56*(-2.66,1.46)	*4.11*(-.67,8.88)
6	*a* = Group-->6M SD *b* = 6M SD-->12M PTS *c′* = Group-->12M SD	-*.070*(-2.93,1.58)	*3.82*(-.82,8.46)

Note. Mediation model coefficients and confidence intervals for direct (*c'*) and indirect effects (*ab*) are shown, examining the relation between chronic posttraumatic stress and sleep disturbance. Indirect and direct effects were not statistically significant for any of the models presented. SD = sleep disturbance, PTS = posttraumatic stress, 6M = 6 month, 12 = 12 month, ULCI = upper bound, LLCI = lower bound of a 95% confidence interval. The 95% confidence interval for the indirect effects are obtained through bootstrapping with 10,000 re-samples.

Appendix A

Glasgow Coma Scale (GCS) Acute Assessment
Eye Opening
4 Opens eyes spontaneously
3 Opens eyes on command
2 Opens eyes to painful stimuli
1 No eye response
Verbal Response
5 Oriented conversation
4 Confused speech
3 Inappropriate words
2 Incomprehensible sounds (e.g., groans)
1 None
Motor response
6 Obeys simple commands
5 Localizes to pain
4 Normal flexion/withdraw to pain
3 Abnormal flexion to pain
2 Extension to pain
1 None

Appendix B

Sleep Disturbance Scale for Children (SDSC)

This questionnaire will allow us to have a better understanding of the sleep-wake rhythm of your child and of any problems in his/her sleep behavior. Try to answer every question. When answering, please think about your child's behavior over the last six months.

1.	How many hours of sleep does your child get on most nights?	9-11 hours	8-9 hours	7-8 hours	5-7 hours	less than 5 hours
2.	How long after going to bed does your child usually fall asleep?	less than 15 minutes	15 - 30 minutes	30 - 45 minutes	45 - 60 minutes	more than 60 minutes

Please answer the following items by marking a number from 1 to 5.

		Never	Occasionally *Once/twice per month*	Sometimes *Once/twice per week*	Often *3 to 5 per week*	Always *Daily*
3.	The child goes to bed reluctantly.	1	2	3	4	5
4.	The child has difficulty getting to sleep at night.	1	2	3	4	5
5.	The child feels anxious or afraid when falling asleep.	1	2	3	4	5
6.	The child startles or jerks parts of the body while falling asleep.	1	2	3	4	5
7.	The child shows repetitive actions such as rocking or head banging while falling asleep.	1	2	3	4	5
8.	The child experiences vivid dream-like scenes while falling asleep.	1	2	3	4	5
9.	The child sweats excessively while falling asleep.	1	2	3	4	5
10.	The child wakes up more than twice per night.	1	2	3	4	5
11.	After waking up in the night, the child has difficulty falling asleep again.	1	2	3	4	5
12.	The child has frequent twitching or jerking of legs while asleep or often changes position during the night or kicks the covers off.	1	2	3	4	5

Appendix B

	Never	Occasionally *Once/twice per month*	Sometimes *Once/twice per week*	Often *3 to 5 per week*	Always *Daily*
13. The child has difficulty in breathing during the night.	1	2	3	4	5
14. The child gasps for breath or is unable to breathe during sleep.	1	2	3	4	5
15. The child snores.	1	2	3	4	5
16. The child sweats excessively during the night.	1	2	3	4	5
17. You have observed the child sleepwalking.	1	2	3	4	5
18. You have observed the child talking in his/her sleep.	1	2	3	4	5
19. The child grinds teeth during sleep.	1	2	3	4	5
20. The child wakes from sleep screaming or confused so that you cannot seem to get through to him/her, but has no memory of these events the next morning.	1	2	3	4	5
21. The child has nightmares which he/she doesn't remember the next day.	1	2	3	4	5
22. The child is unusually difficult to wake up in the morning.	1	2	3	4	5
23. The child awakes in the morning feeling tired.	1	2	3	4	5
24. The child feels unable to move when waking up in the morning.	1	2	3	4	5
25. The child experiences daytime somnolence.	1	2	3	4	5
26. The child falls asleep suddenly in inappropriate situations.	1	2	3	4	5

Appendix C

Child PTSD Symptom Scale (CPSS)

Below is a list of problems that kids sometimes have after experiencing an upsetting event. Read each one carefully and check the box that best describes how often that problem has bothered you IN THE LAST MONTH. Please use your injury as the upsetting event when answering the questions.

Length of time since the injury: _____

	Question	Not at all or only one time 0	Once a week or less/ once in a while 1	2-4 times a week/ half the time 2	5 or more times a week/ almost always 3
1.	Having upsetting thoughts or images about the event that came into your head when you didn't want them to	☐	☐	☐	☐
2.	Having bad dreams or nightmares	☐	☐	☐	☐
3.	Acting or feeling as if the event was happening again (hearing something or seeing a picture about it and feeling as if I am there again)	☐	☐	☐	☐
4.	Feeling upset when you think about it or hear about the event (for example, feeling scared, angry, sad, guilty, etc.)	☐	☐	☐	☐
5.	Having feelings in your body when you think about or hear about the event (for example, breaking out into a sweat, heart beating fast)	☐	☐	☐	☐
6.	Trying not to think about, talk about, or have feelings about the event	☐	☐	☐	☐
7.	Trying to avoid activities, people, or places that remind you of the traumatic event	☐	☐	☐	☐
8.	Not being able to remember an important part of the upsetting event	☐	☐	☐	☐
9.	Having much less interest in doing things you used to do	☐	☐	☐	☐
10.	Not feeling close to people around you	☐	☐	☐	☐
11.	Not being able to have strong feelings (for example, being unable to cry or unable to feel happy)	☐	☐	☐	☐
12.	Feeling as if your future plans or hopes will not come true (for example, you will not have a job or get married or have kids)	☐	☐	☐	☐
13.	Having trouble falling or staying asleep	☐	☐	☐	☐
14.	Feeling irritable or having fits of anger	☐	☐	☐	☐
15.	Having trouble concentrating (for example, losing track of a story on the television, forgetting what you read, not paying attention in class)	☐	☐	☐	☐
16.	Being overly careful (for example, checking to see who is around you and what is around you)	☐	☐	☐	☐
17.	Being jumpy or easily startled (for example, when someone walks up behind you)	☐	☐	☐	☐

Appendix C

CPSS Part 2

Indicate below if the problems you rated in Part 1 have gotten in the way with any of the following areas of your life DURING THE PAST MONTH.

		Yes 1	No 0
18.	Doing your prayers	☐	☒
19.	Chores and duties at home	☐	☐
20.	Relationships with friends	☐	☐
21.	Fun and hobby activities	☐	☐
22.	Schoolwork	☐	☐
23.	Relationships with your family	☐	☐
24.	General happiness with your life	☐	☐

Appendix D

Sleep disturbance subtype planned comparisons

	TBI-EI vs TD		TBI vs EI	
Sleep disturbance subtype	F*(p)*	η^2	F*(p)*	η^2
Insomnia	2.85*(.095)*	.03	.17*(.680)*	< .00
Hypersomnia	2.91*(.091)*	.03	.57*(.453)*	.01
Night time awakening	.83*(.364)*	.01	.18*(.675)*	< .00
Sleep breathing	.51*(.478)*	< .00	.00*(.989)*	< .00
Sleep transition	2.79*(.099)*	.03	.01*(.918)*	< .00
Sleep hyperhydrosis	2.09*(.152)*	.02	.67*(.416)*	.01

Note. Mixed model analysis of variance results for each type of sleep disturbance, demonstrating F-value, p-values, and effect size for each model. Sleep disturbance type did not vary among planned comparisons, such that sleep disturbance levels in the combined traumatic injury group did not significantly differ from levels in the typically developing group, and sleep disturbance levels in the traumatic brain injury group did not significantly differ from levels in the extracranial injury group for any of the subtypes. TBI = traumatic brain injury, EI = extracranial injury, TD = typically developing.