JOURNAL OF NEUROTRAUMA 32:847–852 (June 1, 2015)

DOI: 10.1089/neu.2014.3559

Subjective and Objective Assessment of Sleep in Adolescents with Mild Traumatic Brain Injury

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Abstract

There is increased recognition that sleep problems may develop in children and adolescents after mild traumatic brain injury (mTBI). However, few studies have utilized both subjective and objective measures to comprehensively assess sleep problems in the pediatric population following the acute post-TBI period. The aims of this study were to compare sleep in adolescents with mTBI to healthy adolescents using subjective and objective measures, and to identify the clinical correlates associated with sleep problems. One hundred adolescents (50 adolescents with mTBI recruited from three to twelve months post-injury and 50 healthy adolescents) completed questionnaires assessing sleep quality, depression, and pain symptoms, and underwent 10 day actigraphic assessment of sleep patterns. Adolescents with mTBI reported poorer sleep quality and demonstrated significantly shorter actigraphic-measured sleep duration, poorer sleep efficiency, and more wake time after onset of sleep, compared with healthy adolescents (all, $p < 0.05$). For both groups of adolescents, poorer self-reported sleep quality was predicted by greater depressive symptoms. Poorer actigraphic sleep efficiency was predicted by membership in the mTBI group after controlling for age, sex, depressive symptoms, and presence of pain. Our findings suggest that adolescents may experience subjective and objective sleep disturbances up to one year following mTBI. These findings require further replication in larger samples. Additionally, research is needed to identify possible mechanisms for poor sleep in youth with mTBI.

Key words: actigraphy; adolescents; pain; sleep; traumatic brain injury

Introduction

Mild traumatic brain injury (mTBI) accounts for the vast majority of traumatic brain injuries sustained in the pediatric population.[1] Annually in the United States, as many as 75% of TBIs are classified as mild severity. For the majority of children and adolescents, it is generally accepted that the negative impact of mTBI is largely resolved by two to three months after injury[2]; however, there is increasing recognition that a significant proportion of youth may continue to experience impairments across different domains of functioning, including neurocognitive, psychological, and sleep functioning.[3]

Historically, problems with sleep have been conceptualized within the framework of post-concussive syndrome and have been included in the evaluation during the acute post-injury period. Surveys have found that up to 38% of children with TBI were reported to have poorer sleep during the first month post-injury.[4,5] Research has been less clear regarding the rates of sleep problems beyond the acute period following mTBI. For example, on subjective sleep measures, up to 28% of adolescents who sustained an mTBI were found to have greater levels of sleep disturbances, compared with healthy peers up to six years post-injury.[6] Using objective assessment of sleep, Kaufman and colleagues identified short sleep duration and frequent night time awakenings in adolescents three years after mTBI.[7] In contrast, Milroy and colleagues did not find differences in objectively-assessed sleep patterns comparing children with mTBI to children with orthopedic injuries.[8] These inconsistent findings may be due to the small sample sizes, variation in the range of assessment time after injury, and the inclusion of samples that span wide age ranges. Thus, the presence and persistence of sleep disturbances in adolescents in the longer-term recovery course following mild TBI is not clear.

Understanding sleep disturbances following mTBI is important for several reasons. First, sleep disturbances can result in broad and pervasive negative impact across domains of cognitive, emotional, and physical functioning.[9,10] Second, although problems with sleep may initially be precipitated by the injurious event, over time, sleep problems may become persistent, associated with other functional problems, and refractory to treatment. Management of sleep disturbances requires sleep-focused treatment that is independent from the management and rehabilitation process following mTBI. Therefore, identification of sleep disturbances during the early course following mTBI may lead to effective delivery of sleep-focused intervention and reduction in the experience of chronic sleep problems.

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In the pediatric population, adolescents are particularly vulnerable to a higher incidence of mTBI, as demonstrated by data from the Center for Disease Control and Prevention.[11,12] The incidence of head trauma peaks at age 15 years, with up to 20% of adolescents sustaining a TBI during this developmental period. At the same time, adolescence also presents as a period with increased risk for the development of sleep problems due to the physiologic changes that result in delayed sleep phase combined with behavioral changes from academic and social demands that compete with sleep. During adolescence, many changes in sleep wake patterns occur. Most commonly, there is shortened sleep duration, delayed sleep timing and reduced sleep quality.[13–15] Thus, when adolescents sustain an mTBI, they are likely to incur greater risk of developing chronic sleep problems, compared with younger children. Further, studies have found that persistent sleep problems following TBI are associated with decreased participation in normative roles and poorer quality of life.[16]

Sleep may be affected by TBI through several mechanisms. The impact of a head injury may result in damage to the sleep modulation centers in the brain, such as the hypothalamus and mid and basal forebrain. Findings of altered neurotransmitter levels that play a role in sleep–wake modulation lend support to this theory.[17,18] Other precipitating causes for sleep problems include post-injury psychological factors (e.g., increases in depressive and anxiety symptoms), clinical factors (e.g., onset of headaches), and the use of medications that may interfere with the sleep–wake cycle. Once present, sleep problems are often perpetuated by ongoing psychological, clinical and behavioral factors.[16] We have previously found that higher levels of psychological distress and frequent pain were associated with greater sleep disturbances at 24 months after TBI. Other associated risk factors include female sex and mild severity TBI.[5,6,16] Over the early course following mTBI, multiple factors, in addition to the inciting injury, may contribute to the development and persistence of sleep problems.

The primary objective of this study was to compare the sleep patterns of adolescents following mTBI with a comparator sample of healthy adolescents using multidimensional assessment of sleep (subjective and objective assessments) after the acute period post-injury. This study extends the current literature by using a more comprehensive sleep assessment and an intermediate timeframe of assessment (at three to twelve months after injury), during which there is limited data regarding the persistence of sleep problems. We hypothesized that adolescents with mTBI would report poorer sleep quality and higher levels of pre-sleep arousal, and display shorter actigraphic-measured sleep duration, poorer sleep efficiency, and more frequent night awakenings, compared with healthy peers. The second objective was to identify the multivariate clinical and behavioral risk factors associated with sleep disturbances in adolescents with TBI, compared with healthy adolescents. We hypothesized that controlling for older age, female sex, presence of pain, and greater depressive symptoms, presence of mTBI would be associated with higher rates of sleep disturbances.

Methods

This study was approved by the institutional review boards of the participating hospitals.

Study sample

Participants were adolescents with mTBI ($n = 50$) and healthy adolescents ($n = 50$). For the cohort of adolescents with mTBI, we identified eligible participants from a hospital trauma registry in the northwest United States. This registry is composed of patients who were admitted to the area's Level I trauma hospital after sustaining trauma to any anatomical region. The inclusion criteria for the cohort of adolescents with mTBI were: 1) ages 12 to 18 years old; 2) sustained mTBI in the preceding three to 12 months; 3) able to understand and speak English proficiently; 4) able to mobilize independently; and 5) no serious co-morbid health condition (e.g., cancer, diabetes) or documented developmental delay. Mild TBI was defined as any period of transient confusion, disorientation, impaired consciousness, or amnesia lasting less than 24 h, or signs of other neurological or neuropsychological dysfunction, with the worse Glasgow Coma Scale (GCS) score of 13–15 in the 24 h post-injury or at hospital discharge if discharged prior to 24 hours from admission,[19] abstracted from the hospital trauma registry records. Adolescents with moderate and severe TBI were excluded from this study as the recovery course was more likely to be complicated by neurocognitive problems. The cohort of healthy adolescents was recruited from the community through flyers and word of mouth. Inclusion criteria were: 1) ages 12 to 18 years old; 2) able to understand and speak English proficiently; 3) able to mobilize independently; and 4) absence of a serious co-morbid health condition (e.g., cancer, diabetes) or documented developmental delay. Adolescents were excluded if there was a previous history of head injury.

For the cohort of adolescents with mTBI, letters of invitation with a brief study description were mailed to the potential participant's family residence. The adolescents and families were subsequently contacted via telephone by study staff for further study description and screened for eligibility. Adolescents were enrolled in the study after obtaining written consent from the parent/caregiver and written assent from the adolescent. Recruitment and enrollment occurred between September 2011 and December 2012. A total of 200 eligible participants were sent letters of invitation. Of the 200 eligible participants, 88 did not respond to the mailing or telephone contact for unknown reasons, 44 declined participation due to lack of interest or time constraints, and six were not eligible after initial screening due to decreased proficiency in English. Two were excluded as they were classified as moderate TBI. Participants and non-participants did not differ by sex or age. Post-enrollment, a total of 10 adolescents dropped out, of which five did not return study equipment, and five did not complete the study due to time constraints. The total analyzed cohort included 50 adolescents with complete study data. For the cohort of healthy adolescents, 32 adolescents were part of a larger study examining sleep–wake disturbances.[20] An additional 18 healthy adolescents were recruited between June 2012 and December 2012, for a total sample size of 50 in the healthy cohort. There were four adolescents who did not meet eligibility criteria due to a previous history of head injury and thus were not included in the study.

Procedures

Following enrollment, study materials (written instructions, questionnaire measures, Actiwatch, electronic sleep diary) were mailed to the participants' homes. Study staff then contacted families to instruct parents and adolescents to independently complete questionnaires, and for the adolescent to wear the Actiwatch and complete the electronic sleep diary twice daily for 10 days. This phone call provided an opportunity to answer any questions. Upon completion of the questionnaires and 10-day sleep assessment, families returned all study materials via mail. Families were reimbursed with gift cards to local stores.

Demographics

Information regarding demographics (age, sex, race, ethnicity, and socio-economic status) was provided by the parents.

Depressive symptoms

The Center for Epidemiological Studies Depression Scale (CES-D) is a 20-item questionnaire that assesses symptoms of depression

over the past week.[21] The total score was used in analyses, with higher scores indicating greater depressive symptomatology. The CES-D is one of the most widely used screening tools for depression and has demonstrated adequate reliability and validity in adolescents.[21–23] Consistent with prior research, a CES-D score of 16 or higher was used to indicate the presence of depressive illness.[24] Internal reliability for the present study was good (Cronbach's $\alpha = 0.81$).

Pain

Adolescents rated their usual pain intensity over the past three months using the 11-point Numerical Rating Scale (NRS), with anchors of 0 (''no pain'') to 10 (''worst pain possible''). The NRS is a widely accepted and well validated tool, and has demonstrated good reliability and validity in adolescent populations.[25] Frequency of pain was measured on a 6-point Likert scale from ''less than once a month'' to ''daily.'' The location(s) of pain was assessed using a body outline. Adequate validity of these pain dimensions in clinical and community samples of adolescents has been demonstrated.[26]

Sleep quality

Adolescents completed the Adolescent Sleep Wake Scale (ASWS), a 28-item measure that assesses the quality of sleep along five subscales over the past month: going to bed, falling asleep, maintaining sleep, reinitiating sleep, and return to wakefulness.[27] A total score is calculated from the mean of all items, with higher scores indicating better sleep quality. This scale has been shown to have adequate reliability and validity.[27] Internal reliability for the present study was excellent (Cronbachs $\alpha = 0.90$).

Pre-sleep arousal

Adolescents completed the Pre-Sleep Arousal (PSA)[28] questionnaire, which is a 16-item measure that assesses cognitive and somatic symptoms associated with sleep onset.[28] A slightly modified version for adolescent samples was used in the present study.[20,29] Examples of cognitive arousal symptoms include racing thoughts, worry and anxiety at bedtime; somatic arousal symptoms are tense muscles and racing heart prior to falling asleep. A total score is calculated, with subscales for cognitive PSA and somatic PSA. This scale has shown good reliability and validity in adolescents.[30] Internal consistency in the present study was good (Cronbach's $\alpha = 0.88$).

Actigraphy sleep assessment

Sleep patterns were assessed using the Actiwatch 64 (Phillips Respironics/MiniMitter Company Inc., Bend, OR), a watch-like device that estimates sleep–wake patterns via activity monitoring. Participants were instructed to wear the Actiwatch on their non-dominant wrist for 10 d continuously. In addition, participants were instructed to maintain a corresponding 10 d electronic sleep diary, which was used to assist with actigraphy scoring. Following the 10 d assessment period, data were abstracted and scored by trained research staff using the Actiware Sleep version 5.5.[31] The scoring algorithm is based on the amplitude and frequency of detected movements, using the one-minute epoch and medium sensitivity/wake threshold. The actigraphic variables used in analyses were sleep duration, wake time after sleep onset (WASO) and sleep efficiency. Sleep duration is the total number of minutes from sleep onset to sleep offset. WASO is the number of minutes that the adolescents were awake from sleep onset to offset. Sleep efficiency is the ratio of sleep duration and the total time spend in bed reported as a percentage. Actigraphy sleep variables were averaged over the participants' total assessment period. On average, adolescents with mTBI had 9.5 valid nights of actigraphy data and healthy adolescents had 9.3 valid nights of data available for analysis. Studies suggest that actigraphy provides a reasonably good estimate of sleep–wake patterns, compared with polysomnography in healthy adolescents.[32,33]

Data analysis

Group differences between youth with mTBI and healthy youth were computed on demographic (age, sex, race, ethnicity, family income) and predictor variables (pain intensity, depressive symptoms) using chi-square tests for categorical variables and *t* tests for continuous variables. To address the first aim, independent samples *t* tests were used to examine between group differences on subjective sleep measures (sleep quality and PSA) and actigraphic sleep variables (sleep duration, WASO, and sleep efficiency). To evaluate our second aim, we constructed two separate multivariate regression models evaluating the contribution of demographic and clinical factors to subjective (sleep quality) and objective sleep disturbances (SE). Predictors that were included in all models were age, sex, pain intensity, depressive symptoms, and group (mTBI vs. healthy). All analyses were conducted using Statistical Package for Social Science version 19.0 (IBM Corp., Armonk, NY).

Results

The total sample included 100 youth (50 adolescents with mTBI and 50 healthy adolescents) ages 12 to 18 years old (mean [M] = 15.6 years; standard deviation [SD] = 2.0; 72.1% male). The majority of the participants (84.4%) were Caucasian and non-Hispanic/Latino. There were no significant differences between groups on demographic variables (age, sex, race, ethnicity, or family income; Table 1).

Table 1. Demographics and Clinical Characteristics of Adolescents with TBI and Healthy Adolescents

Variables	*Adolescents with TBI M (SD* n *(%)*	*Healthy adolescents M (SD)* n *(%)*	p
Age	15.9 (2.0)	15.3 (1.9)	0.10
Sex (male)	41 (66.1)	34 (54.8)	0.09
Race			0.99
Caucasian	41 (78.8)	41 (78.8)	
African American	4 (7.7)	5 (9.6)	
Asian	3 (5.8)	2 (3.8)	
Native American/ Alaskan native	1 (1.9)	1 (1.9)	
Other	3 (5.8)	3 (5.8)	
Ethnicity			0.82
Hispanic/Latino	8 (15.4)	7 (13.5)	
Non-Hispanic/Latino	43(82.7)	42 (80.8)	
Unknown	1 (1.9)	2 (3.8)	
Household income			0.10
< $10,000	4 (7.7)	0 (0)	
$10,000 – 19,000	2 (3.8)	1 (1.9)	
$20,000 – 29,000	5 (9.6)	2 (3.8)	
$30,000 – 39,000	2 (3.8)	4 (7.7)	
$40,000 – 49,000	6 (11.5)	5 (9.6)	
$50,000 – 59,000	2 (3.8)	8 (15.4)	
$60,000 – 69,000	4 (7.7)	4 (7.7)	
> $70,000	24 (46.2)	28 (53.8)	
Missing	3 (5.8)	0 (0)	
Pain intensity	4.0 (2.2)	3.2 (2.0)	0.05
Depressive symptoms	13.3 (10.6)	8.3 (6.0)	0.004

TBI, traumatic brain injury; M, mean; SD, standard deviation.

Adolescents with mTBI were assessed at a mean duration of 8.7 months post-injury (SD = 1.7), with a range of five to 12 months after TBI. Within this clinical sample, 37 had a worst Glasgow Coma Scale (GCS) score of 15, 11 had a worst GCS score of 14, and two adolescents had a worst GCS score of 13 at 24 h after TBI. The causes of mTBI were due to motor vehicle crashes (38%), sports-related injuries (20%), falls (18%), being struck by/against an object (14%), and assault (10%). Analyses indicated that adolescents with mTBI experienced significantly higher levels of pain intensity, compared with healthy adolescents (M = 4.1, SD = 2.2 vs. M = 3.2, SD = 2.0; $p = 0.05$). Weekly pain was experienced by more than half of the adolescents with mTBI (58%), with the majority of pain being in the lower limbs (40.4%) and the head (34.6%). A higher proportion of adolescents with mTBI also met the criteria on the CES-D measure for depressive illness, compared with healthy peers (36% vs. 12%; $p = 0.005$).

Group comparison on subjective sleep disturbances

As hypothesized, adolescents with mTBI reported significantly poorer overall sleep quality on the ASWS, compared with healthy adolescents ($p = 0.02$; Table 2). With respect to the individual subscales, compared with healthy peers, adolescents with mTBI reported more difficulties with reinitiating sleep ($p = 0.03$) and trends in difficulties with falling asleep ($p = 0.07$) and returning to wakefulness ($p = 0.07$).

Adolescents with mTBI also reported significantly higher levels of PSA ($p = 0.01$), which was largely driven by significantly higher levels of cognitive symptoms, compared with the healthy peers ($p = 0.006$). There was no difference in the somatic arousal scale scores between groups.

Group differences on actigraphic sleep patterns

As hypothesized, there were significant differences on actigraphic assessment of sleep patterns between adolescents with mTBI and healthy adolescents. Adolescents with mTBI displayed significantly shorter sleep duration at 5.90 hours per night (SD = 0.95), compared with 6.52 h per night in healthy adolescents (SD = 0.92, $p = 0.002$). Fifty-one percent of adolescents with mTBI had a sleep duration of 6 h or less, compared with 24% of healthy adolescents ($p = 0.005$). In addition, adolescents with mTBI experienced more wake minutes during the night, averaging 112 min (SD = 54), compared with 79 min in healthy adolescents (SD = 49; $p = 0.002$). Consequently, adolescents with TBI had significantly poorer sleep efficiency (M = 74.6.0; SD = 10.6) compared with healthy adolescents (M = 81.1, SD = 9.7; $p = 0.002$). Table 3 presents the means and standard deviations of subjective and actigraphy sleep measures for both groups.

Table 2. Between-Group Comparison on Subjective Sleep

	Adolescents with TBI N = *50* *M (SD)* n *(%)*	*Healthy adolescents* *N = 50* *M (SD)* n *(%)*	p
Sleep Quality Total Score	4.11 (0.71)	4.46 (0.83)	0.02
Going to bed	3.89 (1.05)	4.04 (0.95)	0.46
Falling asleep	4.27 (0.97)	4.95 (2.48)	0.07
Maintaining sleep	4.48 (1.10)	4.80 (0.79)	0.11
Reinitiating sleep	4.93 (0.89)	5.28 (0.59)	0.03
Return to wakefulness	2.96 (1.05)	3.32 (0.94)	0.07
Pre-sleep Arousal Total Score	29.66 (9.70)	25.48 (5.86)	0.01
Somatic Scale	11.64 (3.72)	10.60 (2.46)	0.10
Cognitive Scale	18.02 (6.69)	14.88 (4.26)	0.006

TBI, traumatic brain injury; M, mean; SD, standard deviation.

Table 3. Between Group Comparison on Actigraphic Sleep

	Adolescents with TBI N = *50* *M (SD)* n *(%)*	*Healthy Adolescents* N = *50* *M (SD)* n *(%)*	p
Sleep duration (minutes)	353 (58)	389 (55)	0.002
Wake time after sleep onset (minutes)	112 (54)	79 (49)	0.002
Sleep efficiency (percent)	74.6 (10.6)	81.1 (9.7)	0.002

TBI, traumatic brain injury; M, mean; SD, standard deviation.

Multivariate predictors of subjective and objective sleep

Multiple linear regression models were used to identify predictors of subjective and objective sleep. For subjective sleep, we used the ASWS total sleep quality score as the dependent variable. The independent variables included in the model were age, sex, pain intensity, depressive symptoms, and group (mTBI vs. healthy). Depressive symptoms ($\beta = -0.405$; $p \leq 0.001$) were identified as the only significant predictor of poorer subjective sleep quality ($F [5, 94] = 6.86$; $p < 0.001$), with the full model accounting for 26.7% of the variance. Age, sex, and pain intensity were not significant predictors. Contrary to our hypothesis, after controlling for these variables, membership in the mTBI group did not predict subjective sleep quality.

In the second regression model, we evaluated predictors of objective sleep using the actigraphic variable of sleep efficiency. Age, sex, pain intensity, depressive symptoms, and group membership (mTBI vs. healthy) were included as predictors in the model, which overall accounted for 19.4% of the variance in actigraphic sleep efficiency ($F [5, 93] = 4.48$; $p = 0.001$). As hypothesized, after controlling for age, sex, pain intensity, and depressive symptoms, membership in the mTBI group ($\beta = 0.229$; $p = 0.02$) predicted actigraphic measured sleep efficiency. Male sex ($\beta = 0.33$; $p = 0.002$) also was a significant predictor of sleep efficiency.

Discussion

Our findings extend the limited data available on sleep disturbances in adolescents with mTBI. In the short-term recovery period, we found that adolescents who experienced mTBI had higher rates of sleep disturbances, compared with healthy peers, at five to 12 months post-injury. As hypothesized, adolescents with mTBI reported poorer sleep quality and demonstrated poorer objectively measured sleep on actigraphy. Specifically, adolescents with mTBI demonstrated shorter sleep duration, increased wake time during the night, and poorer sleep efficiency, compared with healthy adolescents. Our findings also extend the understanding of potential risk factors for sleep disturbances. Greater depressive symptoms were associated with poorer subjective sleep quality in both groups of adolescents. In contrast, controlling for depression, having a mild TBI and male sex were associated with poorer actigraphic sleep efficiency. These findings suggest that ongoing evaluation of sleep past the immediate period after a head injury is warranted in the

adolescent population. Moreover, screening of depression symptoms also should be a standard part of the care of mTBI patients.

We found that adolescents with mTBI demonstrated markedly short sleep duration, with an average duration of less than 6 h per night. Moreover, these adolescents showed a high number of wake minutes during the night, suggestive of poor sleep quality. The National Sleep Foundation has made the recommendation that optimal sleep requirements for adolescents are approximately 9 h a night, making the total sleep duration displayed by adolescents with mTBI in our sample particularly concerning.[34] Sleep is an important part of the development and growth during childhood, such that poor sleep patterns and behaviors are associated with poorer functioning across multiple domains.[9,35,36] In the context of mTBI, good quality and quantity sleep may be critical for the rehabilitation process. Further, in the present study, sleep disturbances were identified at a mean assessment of eight months after mTBI. This is a point beyond the acute post-injury period when the resolution of post-concussive symptoms is expected. These findings highlight the need for further research to examine the longitudinal course and consequences of sleep disturbances after TBI in the adolescent population, and whether treatment can mitigate these sleep disturbances.

The mechanisms underlying the development of sleep disturbances after TBI are unclear. In a previous study, we found that emotional distress and pain complaints were longitudinally associated with parent-reported sleep problems in children two years after TBI.[16] Similarly, our data obtained from adolescent self-report also show a relationship between greater depressive symptoms and poorer subjectively reported sleep quality following mTBI. The relationship between depressive symptoms and sleep quality has been found in other published clinical samples of adolescents, such as adolescents with major depression and with chronic pain conditions.[37,38] However, contrary to our hypotheses, pain intensity was not associated with subjective sleep disturbances or objective sleep patterns. Adolescents with mTBI reported mild to moderate pain intensity, and it is possible that the pain may not be severe enough to present as a risk factor for disturbed sleep. In studies in which the participants have more severe TBI, pain has been shown to be associated with sleep problems.[16,39]

In contrast, a different pattern of predictors emerged for analyses examining objectively measured sleep (actigraphic sleep efficiency). Our findings demonstrated that after controlling for depressive symptoms, having an mTBI and being male were associated with poorer sleep efficiency. Sex differences in adolescent sleep have been found in several prior studies. Specifically, girls were found to spend more time asleep, with decreased frequency of night time movements.[40] However, depressive symptoms were not associated with actigraphic sleep efficiency, showing a differing pattern from the subjective assessment of sleep quality. It is possible that youth self-report is biased toward overly negative ratings (on both depressive symptoms and sleep) or that the dimensions of sleep affected by mTBI have differing mechanisms. It may be that TBI represents a unique clinical presentation with factors that may contribute to sleep problems that were not evaluated here. We were not able to assess the presence of post-traumatic stress disorder symptoms and the use of medications. We also did not assess sleep architecture using polysomnography and thus cannot speak to possible differences in sleep staging. Further research is needed to investigate multifactorial mechanisms that may contribute to the alterations in the sleep wake cycle that, in turn, contribute to the precipitation and persistence of the range of sleep disturbances in this population.

The findings of the study should be interpreted in light of several limitations. First, the gold standard for physiological assessment of sleep is the use of polysomnography. However, sleep assessment based on actigraphy measurements have been shown to be a valid and reliable method of assessing sleep–wake patterns in adolescents.[33] Further, there is good correlation between sleep and wake time on actigraphy variables and polysomnography.[41] Actigraphy was used in this study due to the advantages of lower cost, and the ability to conduct this study in the home environment. Further studies may extend understanding of sleep by also incorporating polysomnography. Second, we are limited in our ability to understand premorbid sleep problems as we did not have a baseline assessment of sleep problems that may be present prior to TBI. It is possible that a subgroup of youth had pre-existing sleep disturbances. Unfortunately, pre-injury sleep was not examined in this study, and thus, the causal relationships cannot be determined in this study. Third, as mentioned, we did not assess post-traumatic stress disorder symptoms that may play a role in the experience of sleep disturbances in TBI. The experience of trauma itself may be a precipitant for sleep disturbances. Further studies are needed to examine whether trauma symptoms are associated with sleep disturbances in the pediatric population. Fourth, our clinical sample included adolescents with mTBI, which can include a range of severity of head injury that can be further subcategorized based on imaging studies. Future studies may benefit from including an assessment of severity of mTBI. Last, our participation rate in the study was relatively low (although in line with other studies in pediatric TBI), and it was not clear whether there were any systematic sources of sampling bias by sleep concerns between participants and non-participants. Larger, more representative samples are needed in future studies in this area.

The findings in this study support the need to conduct clinical assessment of sleep disturbances in adolescents who suffer from mTBI. A number of sleep screening tools are available (e.g., the BEARS; B = Bedtime Issues, E = Excessive Daytime Sleepiness, A = Night Awakenings, R = Regularity and Duration of Sleep, S = Snoring) that can be used in the clinical assessment to identify sleep problems and alert the practitioner to treat the existing problem.[42] Effective early treatment of sleep disturbances may reduce the likelihood of sleep problems becoming a long-term comorbidity. Minimizing the long-term impact of mTBI bears special relevance for adolescents, as injuries that occur during this developmental period (and their associated complications) may reverberate through to adulthood. In healthy adolescents, poorer sleep quantity and quality have been associated with daytime sleepiness, poorer academic performance, and attentional difficulties.[9,36] The negative sequelae may be compounded in adolescents following mTBI given their increased risk for deficits in attention and concentration and cognitive fatigue secondary to their injury. The relationship between neurocognitive symptoms and sleep disturbances remains an area that warrants further investigation, with research needed to identify the temporal development, causality, and mechanisms underlying this complex set of symptomatology. In particular, longitudinal study designs are needed to examine trajectories of symptoms and potential demographic and clinical factors that predict trajectories. Therefore, improved understanding of the course of sleep disturbances following mTBI and factors that predict increased risk of poor sleep are an important part of the clinical management of this population.

Acknowledgments

This work was funded by NIH Grant P30 NR 011400 (Heitkemper & Vitiello, PI) and 5T32GM086270-03 (PI: Palermo, TM).

We sincerely thank the participants and their families for their interest and willingness to take part in this research.

Author Disclosure Statement

No competing financial interests exist.

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