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The Role of Sleep Deficiency in the Trajectory of Postconcussive Symptoms in Adolescents

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Abstract

Objective: To investigate the trajectory of sleep deficiency after concussion and examine its role as a predictor of postconcussive symptoms (PCS) over 3 weeks and at 3 months post-concussion.

Design: This was a prospective pilot study of 29 adolescents recruited from a pediatric Emergency Department (69% female, mean age = 14.0 years, SD = 1.8) following a concussion.

Methods: Adolescents completed questionnaire assessments at baseline, Weeks 1, 2, and 3 on PCS and sleep patterns. Concurrently, adolescents also completed a daily diary and wore an actigraph continuously to monitor sleep activity. At 3 months post-concussion, adolescents repeated questionnaire measures.

Results: At enrollment, 53.6% reported severe PCS, and 12% maintained severe symptoms at 3 months. Over the first three weeks, sleep duration and daytime sleepiness gradually declined; however, insomnia symptoms remained unchanged. After accounting for age, sex and time since concussion, greater insomnia symptoms at enrollment were associated with more severe PCS at 3 weeks and 3 months ($\beta = 1.17$, $p < .001$). In contrast, sleep duration, efficiency and waketime after sleep onset were not predictors.

Conclusions: Study findings suggest that insomnia symptoms after concussion may provide a target for early intervention to reduce prolonged severity and duration of PCS.

Keywords

Postconcussive symptoms; Sleep; Adolescents; Insomnia; Actigraphy

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Conflict of interest statement

The authors have no conflicts of interest related to this work.

Introduction

The reported rates of concussion amongst adolescents have increased dramatically over the last ten years (1), along with recognition that postconcussive symptoms (PCS) are common in pediatric populations. Over 30% of adolescents report cognitive, somatic, psychological, and behavioural symptoms after a concussion (2). For the majority of adolescents, PCS are expected to resolve within two weeks following injury (3, 4). However, up to one third will continue to experience symptoms at one month, with more than 10% of youth reporting persistent symptoms at 3 months after injury (5–7). These symptoms are associated with poorer quality of life (8), reflected by higher school absenteeism, poorer psychological functioning, and reduced participation in peer, community and school activities.

Concurrently, higher health care costs are incurred in the pursuit of treatment for persistent symptoms related to the head injury (9). Taken together, the high health care costs and negative impact on adolescent quality of life indicate that pediatric concussion is a public health priority (10).

To address the debilitating impact of persistent PCS, there has been extensive research on identifying clinical and individual factors to predict long-term outcomes. Injury characteristics, such as loss of consciousness and amnesia at time of concussion, older age, female sex, premorbid diagnoses of headaches and attention deficit hyperactivity disorder have been identified as potential predictors of worse outcomes (11–13). However, several demographic (e.g., age, sex) and clinical risk factors (loss of consciousness) are fixed and do not provide avenues for prevention or early intervention. More recently, there has been an increased interest in exploring modifiable behavioural factors that could be targeted for early intervention. Sleep problems are one such behavioural factor related to poorer health outcomes, and may represent a potentially modifiable factor of interest.

Deficits in sleep duration and sleep quality following traumatic head injuries are common (14–18). Children may experience shortened nighttime sleep duration, frequent night wakings, difficulty falling asleep and maintaining sleep after head injury (14, 17). In fact, these symptoms may continue for months to years beyond the injury. In adults, similar changes in sleep patterns have also been described (19, 20). In particular, there is evidence of long-term poor sleep quality being associated with greater postconcussion symptoms (21). Research has found that sleep deficiency negatively influences health and symptom expression in other medical conditions (such as chronic pain) (22–24). Moreover, sleep deficiency is linked to poorer neurocognitive and psychological outcomes, as well as reduced academic achievement in other clinical samples (25). Therefore, the presence of sleep deficiency during the period postconcussion poses significant concerns for the neurocognitive and functional outcomes of children and adolescents (26). Although a number of studies have linked poor sleep with poor clinical outcomes, there have been limited studies to understand the course of sleep trajectories following pediatric concussion and its influence on recovery outcomes (27).

The primary aim of this longitudinal study was to characterize the trajectory of sleep deficiency (reduced sleep duration, reduced sleep efficiency, increased insomnia symptoms,

increased daytime sleepiness) and PCS from early post injury to three months after concussion in adolescents. We hypothesized that the majority of adolescents would experience sleep deficiency immediately after injury that would resolve over the subsequent three weeks. However, a subgroup of adolescents was expected to demonstrate persistent PCS and sleep deficiency up to 3 months postconcussion. The secondary aim was to determine the influence of early sleep deficiency after concussion on the severity of PCS over time. We hypothesized that early presence of sleep deficiency would be associated with greater PCS up to 3 months later.

Methods

This study was approved by the institutional review board of the study institution.

Study design and participants.

This was a prospective study conducted between February and August 2016. After evaluation and management of concussion by medical providers at the Emergency Department (ED) of a northwest children's hospital, potential participants were approached by research coordinators. Recruitment took place in the ED by research coordinators who screened all ED patients for eligibility. Participants meeting inclusion criteria were enrolled following obtaining assent from adolescents and consent from their parents for study participation prior to discharge from the ED. Adolescents were eligible if they were between ages 11 to 18 years old, sustained a head injury in the past 7 days and were diagnosed with sports- or recreational-related concussion by ED clinicians, and were planning to be discharged home from the ED. Adolescents were excluded if they did not speak or read English, or were prescribed a new medication in the past month. At discharge, adolescents and families were provided with standard instructions for care after concussion, including rest, symptom observation, and restriction from sports until re-evaluation by primary care physician or at the hospital's concussion clinics.

Research coordinators approached 43 adolescents in the ED. Of those approached, 5 were not enrolled due to lack of interest ($n = 3$) or not meeting eligibility criteria ($n = 2$). There were 38 adolescents and parents enrolled, however 9 participants did not complete baseline assessments in a timely manner (i.e., questionnaire data or actigraphy data not collected in the first week after concussion), and therefore were not included in the study. Data from the final sample of 29 adolescents are included in final analyses.

Procedures and Measures

The prospective study design involved data collection at enrollment, Week 1, Week 2, Week 3, and 3 months after concussion. At enrollment, parents completed questionnaires to collect demographic information and medical history. Adolescents completed questionnaire assessments at Week 1, Week 2, and Week 3 following the injury. Adolescents also completed a daily sleep diary and wore an actigraph continuously for these 3 weeks to monitor sleep patterns. At 3 months after concussion, adolescents repeated the questionnaire measures.

Demographics.—Parents provided information on socio-demographic characteristics at the time of enrollment.

Concussion.—The mechanism of injury and the sport/activity during which the concussion occurred were abstracted from the medical record by the research coordinators.

Post-concussive symptoms (PCS).—Adolescents completed the Health and Behaviour Inventory (HBI) Child Version to assess PCS (28, 29). This 20-item measure asks youth to rate the severity of their symptoms from 0 = “never” to 3 = “often”. The items are totaled for a summary score with two subscales in cognitive and somatic domains. Higher scores indicate greater and more severe symptoms. Cronbach α for the summary score was from 0.93 to 0.96 across assessment occasions, demonstrating high internal consistency. Cronbach α ’s for the cognitive and somatic subscales were from 0.93 to 0.96 and from 0.73 to 0.90 respectively. The HBI was developed for children with moderate to severe TBI and has been validated for children with head injuries (29). Scores of > 21 are categorized as more severe (28).

Assessment of sleep patterns

Actigraphy.—Sleep patterns were monitored using the Actiwatch-2 (Phillips Respironics/MiniMitter Company Inc., Bend, OR). The Actiwatch-2 is an omni-directional motion sensor, which allows continuous monitoring of activity, as a proxy for sleep patterns. Adolescents were asked to wear the device on their non-dominant wrist for 24 hours a day for 3 weeks. Youth were asked to press an event marker button when going to bed in the evening and waking up in the morning and to complete a sleep log. The event markers and sleep logs aided scoring. Actiware Software version 6.0.6 was used which follows an algorithm to detect the frequency and amplitude of movements; counts were collected in 1-minute epochs. The sleep variables calculated from actigraphy data were: total nighttime sleep duration (SD), wake after sleep onset (WASO), and sleep efficiency (SE). Total sleep time is the number of minutes from sleep onset to sleep offset. WASO is the number of minutes awake from sleep onset to sleep offset. Finally, sleep efficiency is calculated by dividing sleep duration by the total time spent in bed and multiplying by 100. Percentages closer to 100 indicate better sleep efficiency. Studies have shown that actigraphy is sensitive in assessing actual sleep when compared to polysomnography, with 85 – 95% agreement on certain sleep variables (30–32).

Insomnia symptoms.—Adolescents completed the Insomnia Severity Index (33), a 7-item questionnaire that assesses insomnia severity and the impact of symptoms. The response choices are rated on a 5-point Likert scale (0 – 4). All items are summed to a total score to rate severity of insomnia symptoms (33). This has been validated in the pediatric population (34–36).

Daytime Sleepiness.—To assess daytime sleepiness, adolescents reported symptoms using the modified pediatric Epworth Sleepiness Scale (37). Adolescents completed this 8-item questionnaire on a 4-point Likert scale (0 = “would never doze or sleep”, 3 = “high

chance of dozing or sleeping”) about activities during the day. Higher scores indicate higher levels of daytime sleepiness. This has been validated in children and adolescents (38).

Data Analyses

Descriptive statistics were calculated for sociodemographic and clinical characteristics. For the first aim, we applied General Estimating Equation (GEE) to examine separate trajectories of PCS (somatic and cognitive subscales), insomnia symptoms, and daytime sleepiness during Weeks 1, 2, and 3 and 3 months post-injury. Trajectories for actigraphy data (sleep duration, sleep efficiency and wake time after sleep onset) were also examined in separate GEE models for the 3-week period after concussion. The use of GEE as a semiparametric technique can be applied to longitudinal data to estimate model parameters related to change in sample means over time. It relaxes distribution assumptions and includes all participant data even when missing time points, allowing for the assessment of change in sample means in longitudinal studies (39). To assess sleep deficiency as a predictor of PCS at 3 weeks postconcussion) and 3 months postconcussion, we applied GEE to determine the association of sleep variables to PCS. Separate models were created for total PCS, somatic and cognitive subscales. In all models, age, sex, and days between injury and enrollment were entered as covariates. Analyses were performed using SPSS (IBM SPSS Statistics for Windows, Version 19.0. Armonk, NY: IBM Corp.citation) and Stata (StataCorp. 2017 Stata Statistical Software: Release 15. College Station, TX: StatCorp LLC).

Results:

The sample included 29 adolescents (69% female; $M = 13.96$ years, $SD = 1.83$). Sociodemographic information is detailed in Table 1. On average, participants were seen in the Emergency Department and enrolled into the study at a mean of 4.8 days ($SD = 1.7$ days, range 2 – 8 days) after sustaining the concussion. Approximately half (44.8%) of the concussions were sustained during a contact sport, with the rest occurring during a limited contact or non-contact sport.

The rates of response for completion of questionnaires at Week 1 was 100% ($n = 29$), at Week 2 was 89.7% ($n = 26$), at Week 3 was 100% ($n = 29$), and at 3 months was 86.2% ($n = 25$). Actigraphy data were available for 28 adolescents, due to technical errors rendering one participant’s actigraphy data unusable. As actigraphy data were averaged over the course of one week, only participants who completed at least 7 days of actigraphy were included for analyses ($n = 28$). The average number of actigraphy days completed was 19.8 days.

Change in Postconcussive symptoms (PCS)

GEE models were used to examine change in PCS over time, while controlling for age, sex and number of days since sustaining the concussion. As hypothesized, the majority of adolescents reported PCS during the first week after concussion ($M = 25.50$, $SD 15.68$), with 53.6% ($n = 15$) reporting severe levels of PCS. Compared to Week 1, there was a significant reduction in PCS at Week 2 ($\beta = -8.56$, $p < 0.001$) and Week 3 ($\beta = -11.52$, $p < 0.001$). Further improvement was found at 3 months follow up ($\beta = -15.58$, $p < 0.001$;

Figure 1). Trajectories were similar for cognitive and somatic subscales. At Week 3, 17.2% of adolescents reported severe levels of PCS; at 3 months, 12.5% reported severe levels of PCS (Table 2).

Changes in sleep

Nighttime sleep duration on actigraphy decreased over the acute postconcussive period. Sleep duration was the longest at Week 1, averaging 7 hours 40 minutes (SD = 77 minutes). Compared to Week 1, there was a significant reduction in sleep duration at Week 2 to an average duration of 7 hours and 4 minutes (SD = 1 hour, 31 minutes; $\beta = -41$, $p = 0.04$). Comparing Week 3 to Week 1, there was a further reduction to 6 hours and 43 minutes (SD = 1 hour 38 minutes; $\beta = -61$, $p = 0.002$). However, there were no significant changes in WASO and sleep efficiency across the first 3 weeks following concussion.

As a group, adolescents reported low to moderate range of insomnia symptoms after injury (M = 8.23, SD = 6.67). There was no statistically significant change in levels of insomnia over the course of the first 3 weeks post-injury. However, at 3 months after concussion, there was a statistically significant reduction (M = 4.96, SD = 3.80, $\beta = -2.39$, $p = 0.031$) in insomnia symptoms compared to the first weeks after concussion.

Daytime sleepiness was highest during Week 1 after concussion and reduced over the recovery period with significant reduction at Week 2 ($\beta = -3.13$, $p < .001$), Week 3 ($\beta = -3.33$, $p = .001$) and 3 months ($\beta = -4.43$, $p = .004$).

Early insomnia symptoms, but not actigraphic sleep variables were associated with more severe PCS.

Greater insomnia symptoms at Week 1 ($\beta = .93$, $p < 0.001$) were associated with more severe total PCS at 3 weeks and 3 months after concussion (Table 4). Similarly, in separate models examining cognitive ($\beta = .555$, $p = 0.002$) and somatic ($\beta = .36$, $p = 0.001$) PCS symptoms, more severe insomnia symptoms were associated with worse symptoms in both domains at 3 months. Separate models tested actigraphic sleep duration and sleep efficiency as a predictor of PCS. Neither sleep duration nor efficiency were found to be significant predictors over time (Table 5).

Discussion

Over half of the adolescents in our study reported severe PCS during the acute recovery period. As expected, there was a gradual and steady reduction of PCS over the first 3 weeks across both cognitive and somatic functioning. In line with findings from prior research (7, 40), we found that 20% reported PCS at 3 weeks after concussion, and 12% of adolescents continued to experience PCS at 3 months after injury. We also examined trajectories of sleep deficiency (i.e., increased daytime sleepiness, reduced sleep duration, increased insomnia symptoms) finding variability over time. As expected, as adolescents recovered over the first 3 weeks after concussion, their level of daytime sleepiness and night time sleep duration decreased. We interpret this as a temporary change due to the concussion with a return to their baseline at 3 weeks. However, actigraphy measures of sleep efficiency and wakings at night time remained stable over time, suggesting that concussion did not alter the amount of

sleep fragmentation experienced by adolescents. This study extends our understanding of both subjective and objectively measured sleep following concussion, and sheds light on the trajectories of sleep and postconcussive symptoms in the acute and chronic phase after injury.

In contrast to daytime sleepiness and night time sleep duration, adolescents reported low to moderate range of insomnia symptoms after concussion, and the level of symptomatology remained unchanged over the subsequent weeks. It was on assessment at the 3-month follow up that adolescents reported a significant reduction in insomnia symptoms. This raises the question that insomnia symptoms may not be as transitory, compared to changes in sleep duration and daytime sleepiness after concussion. Additionally, we also found evidence that greater insomnia symptoms after concussion were associated with more severe post-concussive symptoms at 3 months, extending findings from retrospective studies. Retrospective studies have shown that insomnia may be associated with poorer outcomes (41, 42). In a study of over 400 adolescents after concussion, Bramley and colleagues found that sleep disturbance was associated with a 3- to 4-fold increase in recovery time (41). Similarly, using a national high school sports injury surveillance database Kerr and colleagues found that insomnia symptoms were associated with a 2.79 increase in odds of post-concussive symptoms (42).

Our study findings indicate that there is variability of adolescent sleep patterns and behaviors following a concussion. Further, insomnia symptoms immediately following concussion may predict the severity of post-concussive symptoms over time. Understanding the changes in the different etiologies of sleep disturbances can help to tailor specific treatment approaches. Adolescent insomnia has been effectively treated with melatonin, sleep hygiene education and cognitive behavioral therapy (43–46). Thus, as a modifiable target, insomnia symptoms has potential to be a powerful target of early intervention in youth following concussion. There have not yet been any studies specifically to treat insomnia in youth following concussion, although insomnia has been successfully treated in other pediatric populations and this will be an important avenue for future investigations (36, 47). Nonetheless, our data suggest that sleep should be part of a standard clinical assessment following concussion.

The neurobiological basis of sleep deficiency following head injury remains elusive. It is hypothesized that injury to the sleep modulation centre (housed in the forebrain and the midbrain) may result in altered neurotransmitter and neuropeptide levels responsible for the sleep-wake cycle, potentially contributing to changes in sleep patterns and behaviour. Insomnia may result from behavioural and affective changes following injury including changes in sleep habits (e.g., increased time in bed napping or resting) and increased arousal at bedtime. Further research is needed to understand changes in multiple aspects of sleep including sleep architecture and sleep behaviours.

One strength of this study is the comprehensive assessment of sleep patterns using both subjective and objective measures, and the evaluation of multiple domains of sleep functioning including sleep efficiency, sleep duration, insomnia symptoms, and daytime sleepiness. Second, we studied a clinical group of adolescents from the community who

presented to the Emergency Department after sustaining a concussion. As concussions are generally viewed to be self-limiting, patients may not present for medical evaluation; or often recommended to follow-up at primary care clinics as needed. However, for up to 30% of adolescents, the prolonged symptom duration highlights the need to identify at-risk individuals to target intervention, minimizing longer term negative consequences.

Study findings should be evaluated in the context of the following limitations. This is a pilot study of a small sample of adolescents. Therefore, the findings need to be replicated in a larger sample. The second limitation is that assessment of sleep was conducted after the injury event. It is unknown whether the sleep deficiency identified following concussion was a result of the concussion, or was present prior to injury. We also did not assess events occurring during recovery that may influence sleep such as return to school. Third, there was no control group for comparison of sleep patterns. Potentially, any injury may be associated with poorer sleep patterns and behaviours. Thus, future studies should include a comparison group. Fourth, this sample consisted of a higher proportion of females than has been noted in studies of the prevalence of concussion in adolescents, which may reflect the recruitment setting or participation patterns. Moreover, future studies should also examine for sex differences given possible differences in concussion symptoms and sleep patterns and behaviors. Finally, we did not assess for other types of sleep disturbances (abnormal movements and behaviours during sleep such as restless leg syndrome, pleiosomnia, circadian rhythm disorders) in this study. The rates of these sleep disturbances in individuals with concussion are reported to be higher compared to healthy peers (48). Future studies should include assessment of other etiologies of sleep disturbances.

In summary, post-concussive symptoms are common in adolescents during the acute recovery period. Sleep trajectories show some changes during recovery in both nighttime and daytime symptoms. Insomnia symptoms predict the severity and duration of post-concussive symptoms. Future research is needed to understand whether screening for insomnia symptoms after concussion may provide a target for early intervention to reduce PCS.

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Figure 1.
Trajectory of postconcussion symptoms at Weeks 1, 2, 3, and 3 months.

Table 1.

Demographic and clinical characteristics of adolescents.

	Mean (SD) / Frequency (%) (n = 29)
Age (years)	13.96 (1.83)
Sex (% female)	20 (69.0)
Race	
White	23 (79.3)
African American	2 (6.9)
Native Hawaiian/Pacific Islander	1 (3.4)
Asian	2 (6.9)
Other	1 (3.4)
Household annual income	
< \$10,000	1 (3.4)
\$30,000–49,999	4 (13.8)
\$50,000–69,999	5 (17.2)
\$70,000–100,000	6 (20.7)
> \$100,000	13 (44.8)
Mechanism of injury	
Fall	17 (58.62)
Struck by object	12 (41.37)
Sport played	
Contact	13 (44.8)
Limited contact	12 (41.4)
Non-contact	4 (13.8)
Days since injury (range)	4.83 (2 – 8)

Table 2.

Means and standard deviations of clinical factors at weeks 1, 2, 3, and 3 months after concussion.

	Week 1	Week 2	Week 3	3 month
	Mean (SD)			
PCS Total	25.5 (15.68)	15.04 (11.77)	12.64 (12.88)	7.5 (8.49)
PCS: Cognitive	15.14 (9.51)	8.64 (7.23)	8.04 (8.04)	5.13 (6.27)
PCS: Somatic	10.36 (7.08)	6.4 (5.55)	4.61 (5.60)	2.38 (2.72)
PCS > 21	14/29 (48.3%)	7/26 (26.9%)	5/29 (17.2%)	3/25 (12%)
Insomnia	8.23 (6.67)	7.50 (6.54)	6.18 (5.28)	4.96 (3.80)
Daytime sleepiness	8.66 (6.26)	5.04 (5.23)	5.07 (5.60)	3.74 (3.88)

Table 3.

Means and standard deviations of actigraphic sleep variables at Weeks 1, 2, and 3 after concussion.

	Week 1	Week2	Week 3
Sleep duration (minutes)	460.2 (78.6)	424.9 (92.9)	406.0 (99.9)
Sle e p Efficie ncy (%)	87.0 (4.6)	84.1 (17.1)	82.4 (18.3)
Wake time after Sleep Onset (minutes)	56.8 (24.1)	51.7 (22.3)	52.7 (23.7)

Table 4.

Multilevel linear mixed effects model demonstrating the associations of insomnia symptoms on postconcussive symptoms.

Independent Variables		PCS Total Beta Coefficient (Confidence intervals)	PCS Cognitive Beta Coefficient (Confidence intervals)	PCS Somatic Beta Coefficient (Confidence intervals)
Week	1	Ref	Ref	Ref
	2	-8.69 (-12.04, -5.33) ***	-5.58 (-7.65, -3.51) ***	-3.14 (-5.17, -1.10) **
	3	-11.53 (-15.93, -7.16) ***	-6.43 (-9.20, -3.66) ***	-5.16 (-7.19, -3.12) ***
	12	-15.27 (-21.03, -9.54) ***	-8.22 (-11.67, -4.77) ***	-7.00 (-9.47, -4.53) ***
Age		0.53 (-1.08, 2.14)	0.46 (-0.64, 1.56)	0.06 (-0.54, 0.66)
Sex		0.92 (-5.38, 7.22)	-0.37 (-5.28, 4.54)	1.31 (-0.95, 3.57)
Days following Injury		1.18 (-0.62, 2.98)	0.33 (-0.89, 1.55)	0.83 (0.14, 1.53)
Insomnia Symptoms (Week 1)		1.17 (0.68, 1.65) ***	0.69 (0.37, 1.01) **	0.47 (0.24, 0.69) ***

* =
p < .05;

** =
p < .01;

*** =
p < .001

PCS: Postconcussive symptoms

Table 5.

Multilevel linear mixed effects model demonstrating the associations of actigraphy sleep duration on postconcussive symptoms.

Independent Variables		PCS Total Beta Coefficient (Confidence intervals)	PCS Cognitive Beta Coefficient (Confidence intervals)	PCS Somatic Beta Coefficient (Confidence intervals)
Week	1	Ref	Ref	Ref
	2	-8.86 (-12.96, -4.76) ***	-5.26 (-7.65, -2.86) ***	-3.62 (-6.12, -1.13) ***
	3	-12.90 (-18.31, -7.49) ***	-6.94 (-10.37, -3.51) ***	-5.99 (-8.41, -3.56) ***
	12	-13.49 (-19.66, -7.32) ***	-6.89 (-10.45, -3.34) ***	-6.61 (-9.40, -3.82) ***
Age		0.99 (-0.98, 2.96)	0.64 (-0.74, 2.02)	0.35 (-0.31, 1.00)
Sex		3.23 (-5.36, 11.82)	1.00 (-5.73, 7.82)	2.26* (0.03, 4.49)
Days following Injury		-0.16 (-3.20, 2.89)	-0.20 (-2.43, 2.04)	0.03 (-0.90, 0.97)
Sleep duration (Week 1)		0.01 (-0.02, 0.05)	0.001 (-0.02, 0.03)	0.008 (-0.003, 0.02)

* =
p < .05;

** =
p < .01;

*** =
p < .001

PCS: Postconcussive symptoms