Journal of Athletic Training 2023;58(5):414–422
doi: 10.4085/1062-6050-0078.22

www.natajournals.org

Prospective Implications of Insufficient Sleep for Athletes

Kaitlin Elizabeth Riegler, MS; Erin T. Guty, MS; Garrett A. Thomas, MS; Megan L. Bradson, MS; Peter A. Arnett, PhD

Pennsylvania State University, State College

Context: Poor sleep is common in collegiate student-athletes and is associated with heterogeneous self-reported complaints at baseline. However, the long-term implications of poor sleep at baseline have been less well studied.

Objective: To examine the implications of insufficient sleep at baseline, as well as factors such as symptom reporting and neurocognitive performance at baseline associated with insufficient sleep, for the risk of sport-related concussion (SRC).

Design: Cross-sectional study.

Setting: Undergraduate institution.

Patients or Other Participants: Student-athletes (N = 614) were divided into 2 groups based on the hours slept the night before baseline testing: sufficient (>7.07 hours) or insufficient (≤5.78 hours) sleepers. Athletes who went on to sustain an SRC during their athletic careers at our university were identified.

Main Outcome Measure(s): Four symptom clusters (cognitive, physical, affective, and sleep) and headache were examined as self-reported outcomes. Four neurocognitive outcome measures were explored: mean composite of memory, mean composite of attention/processing speed, memory intraindividual variability (IIV), and attention/processing speed IIV.

Results: Insufficient sleepers at baseline were nearly twice as likely (15.69%) as sufficient sleepers (8.79%) to go on to sustain an SRC. Insufficient sleepers at baseline, whether or not they went on to sustain an SRC, reported a higher number of baseline symptoms than did sufficient sleepers. When compared with either insufficient sleepers at baseline who did not go on to incur an SRC or with sufficient sleepers who did go on to sustain an SRC, the insufficient sleep group that went on to incur an SRC performed worse at baseline on mean attention/processing speed.

Conclusions: The combination of insufficient sleep and worse attention/processing speed performance at baseline may increase the risk of sustaining a future SRC.

Key Words: cognitive functioning, postconcussion symptoms, concussion, sports injuries, self-report

Key Points

- Insufficient sleep at baseline was associated with an increased risk of prospectively sustaining a sport-related concussion.
- Athletes who reported insufficient sleep at baseline and went on to experience a sport-related concussion performed worse on baseline attention/processing speed tasks than either sufficient sleepers who went on to experience a concussion or insufficient sleepers who did not.
- Screening for sleep difficulties at baseline can aid in early identification and referral to treatment for athletes experiencing sleep difficulties, thereby potentially reducing the risk of sustaining a sport-related concussion.

An estimated 500 000 players participate in US collegiate athletics annually, and about 6% will sustain a concussion.[1] The identification of characteristics that make an athlete more susceptible to sustaining a sport-related concussion (SRC) or will affect recovery from SRC has become a major focus of research.[2,3] The neuropsychological study of SRC is rooted in the biopsychosocial model, whereby biological, psychological, and socioenvironmental factors interact to affect the risk for injury in the first place and subsequent recovery after SRC.[4] These risk factors may be fixed preinjury characteristics, such as an athlete's biological sex or the presence of certain genetic polymorphisms, or modifiable characteristics, such as playing style, psychological and mood disorders, or insufficient sleep. It may be particularly important to focus on this second category because these can be points of intervention.

Growing attention has been focused on the importance of sleep and the effect of sleep on athletic performance as well as the overall quality of physical and mental health.[5] The most recent consensus statement[2] on concussion in sport identified sleep as a potential modifier. Poor sleep quality and insufficient sleep are common among collegiate athletes.[6] In a study at a National Collegiate Athletic Association Division I university, 39.1% described sleeping fewer than 7 hours per night, and 42.4% reported poor sleep quality as measured using the Pittsburgh Sleep Quality Index.[6,7] Contributors to poor sleep may be sport or nonsport related. Sport-related factors include travel for competition or early morning practices, and it has been estimated that collegiate athletes spend 27 to 41 hours per

week on athletic activities.[5,6] In addition to the time spent on athletics, student-athletes are purportedly students first and thus subject to the same non–sport-related sleep disruptions as other college students, including academic stress and loud shared environments for sleep. Given the unique time demands on athletes and the potential to disrupt sleep, examining the effect of sleep quantity in this population is warranted.

Prior researchers have explored the effects of poor sleep on baseline performance in nonconcussed athletes. Most[8–10] have found that nonconcussed athletes with poor sleep endorsed more subjective complaints on self-reported symptom inventories than nonconcussed athletes without poor sleep, but they did not perform significantly worse on neuropsychological assessments. Despite the lack of evidence for objective performance differences at baseline due to poor sleep, some investigators have demonstrated longer-term effects of poor sleep at baseline, suggesting that poor sleep may be linked to more distant consequences.[11]

More generally, poor sleep has been associated with sport injuries,[12] yet few authors have looked specifically at the association of concussion with it. These injuries may be more common for several reasons, including degraded sport performance, reduced body control, increased difficulty responding on the field, and poor visual tracking and reaction time during sport activities. Taken together, these theories indicate that sleep difficulties may increase the vulnerability to injury via performance decrements and that insufficient sleep may disrupt optimal injury recovery.

Whereas research on the remote consequences of baseline sleep disruption is limited, a few studies are worth mentioning. One group[8] determined that preinjury sleep difficulties were a risk factor for postconcussion syndrome. In addition, preinjury sleep disturbances predicted the postinjury symptom total and worse performance on visual memory and reaction time tests than a control group (no sleep disturbances).[13] Other authors[14] found that daytime sleepiness on 2 or more days per month and moderate to severe self-reported insomnia were associated with a 3 to 5 times higher risk of sustaining a future SRC. Therefore, the consequences of insufficient sleep at baseline may emerge later, and these consequences may manifest as increased difficulties when coupled with an SRC. Whereas the effects of sleep disruption at baseline may be restricted to self-reported complaints, these disturbances may meaningfully affect the risk for SRC or for worse outcomes after SRC. However, limited research has addressed other factors that may co-occur with sleep difficulties at baseline to confer a risk for future concussion, such as patterns of symptom reporting or cognitive functioning.

Prior investigators have identified that certain characteristics, or so-called *modifiers*, at baseline may affect symptom reporting and cognitive performance in subsets of athletes. For instance, a previous psychological diagnosis, such as depression and anxiety or a history of concussion or attention-deficit/hyperactivity disorder, may influence baseline performance.[15–17] Thus, understanding the factors associated with these characteristics at baseline is important in concussion management. Insufficient sleep is an additional factor that may be associated with these characteristics at baseline. Hence, we aimed to examine symptom reporting and cognitive functioning at baseline in the presence of insufficient sleep and explore whether certain patterns were associated with a risk for future SRC. To our knowledge, no earlier authors have explored these factors simultaneously to understand the remote consequences of insufficient sleep at baseline: the risk for SRC, subjective self-report symptoms, and cognitive performance.

Aim 1

Aim 1 was to examine whether *insufficient sleep*, defined as sleeping $\leq$5.78 hours the night before a baseline assessment, was a risk factor for prospectively sustaining an SRC.

Hypothesis 1

We predicted that a greater proportion of athletes getting insufficient sleep ($\leq$5.78 hours) at baseline would sustain an SRC than athletes getting sufficient sleep ($>$7.07 hours) at baseline.

Aim 2

Aim 2 was to identify other baseline factors associated with insufficient sleep at baseline and the likelihood of sustaining an SRC in the future. Two group comparisons were conducted: (1) insufficient sleepers with SRC versus insufficient sleepers without SRC and (2) sufficient sleepers with SRC versus insufficient sleepers with SRC. We compared baseline symptom reporting (symptom clusters and headache) and neuropsychological test performance (mean cognitive performance and intraindividual variability [IIV] in cognitive performance) in the specific domains of memory and attention/processing speed (APS).

Hypothesis 2

We proposed that insufficient sleepers who went on to sustain an SRC would report more symptoms and demonstrate worse cognitive performance at baseline than insufficient sleepers who did not go on to sustain an SRC. We also predicted that the insufficient sleepers who sustained an SRC would report more symptoms and demonstrate worse cognitive performance at baseline than the sufficient sleepers who sustained an SRC.

METHODS

Participants

The data for the current study are mostly cross-sectional and based on a secondary analysis from a concussion-management program at a Division I university; symptom reporting, cognitive functioning, and sleep were assessed at baseline. One component of the study is longitudinal in nature, given that athletes were prospectively followed to determine which ones went on to sustain a future SRC. This "parent study" consisted of all athletes who participated in the sports concussion program from 2002 to 2019. The athletes were administered baseline testing before their participation in collegiate athletics. For both aims, we divided participants into 2 groups based on the hours slept the night before the baseline test. Consistent with prior authors,[9] we derived these groups empirically based on the mean and SD of hours slept the night before the baseline of all participants who responded to that question (n = 772,

mean = 7.07 ± 1.29 hours). The sufficient sleep group consisted of those obtaining more hours of sleep than the mean in our sample (>7.07 hours), and the insufficient sleep group consisted of those obtaining fewer or equal hours of sleep to 1 SD below the mean in our sample (≤5.78 hours). The empirical derivation of these cutoffs was predicated on the perceived limitation in the existing literature from reliance on theoretically derived cutoffs. Specifically, given the unique patterns of sleep difficulties in both college students and student-athletes, we believed it was beneficial to use values from this normative sample to understand sleep difficulties that were experienced over and above those of their peers.

For aim 1, athletes were selected from the larger sample of 1057 collegiate athletes who completed baseline testing from 2002 to 2019. Recruits were excluded from the current study for the following reasons: did not complete the Immediate Post-Concussion Assessment and Cognitive Testing (ImPACT) question related to the prior night's sleep (n = 278) or reported sleep in the middle range (5.77–7.06 hours) the night before baseline (n = 165). Thus, the final sample for aim 1 consisted of 614 (455 men, 159 women) student-athletes. For aim 1, 102 athletes were in the insufficient sleep group and 512 were in the sufficient sleep group.

For aim 2, analyses related to the cognitive outcomes, athletes with *invalid performance on effort testing*, defined as ImPACT impulse control composite (ICC) scores ≥ 30 (n = 22), were also excluded.[18] The final sample for this set of analyses consisted of 592 athletes (437 men, 155 women). After removing athletes with invalid baseline neuropsychological performance, 100 insufficient sleepers and 492 sufficient sleepers remained. Demographics information for the aim 1 and aim 2 participants are located in Tables 1 and 2, respectively.

Procedures

Baseline testing was completed as part of the sports concussion program that is based on the "Sports as a Laboratory" model in which athletes are referred for baseline testing and then, if they sustain a concussion, they are referred for repeat testing. Referrals are made by an athletic trainer or team physician. *Concussion* was defined as an injury to the head resulting from a trauma or biomechanical force wherein brain function was disrupted, as evidenced by any alteration in mental status or postconcussion signs or symptoms at the time of injury, posttraumatic amnesia lasting <24 hours, or loss of consciousness lasting ≤30 minutes.[22]

The neuropsychological test battery was administered by trained undergraduate research assistants or graduate students who were supervised by a doctoral-level clinical neuropsychologist. Athletes provided informed consent before the study, and the study was approved by the university's institutional review board.

Measures

Sleep. The ImPACT consists of 3 main sections: demographics and background information, symptom reporting, and 6 testing modules.[18] One of the background questions is "How many hours did you sleep last night?" Reponses to this question were used to create the 2 sleep groups (sufficient sleepers and insufficient sleepers). Self-report of athletes' sleep has been widely used in prior research in this area, and evidence indicates that self-report is an appropriate cost-effective and time-effective alternative to objective sleep measurements in an uninjured athlete population. Self-report of a single night's sleep has also been shown[19] to be moderately to highly correlated with sleep measured using actigraphy, with Pearson product moment correlations ranging from 0.31 to 0.65.

Symptom Reporting. The ImPACT has a self-report symptom section called the Post-Concussion Symptom Scale (PCSS). The PCSS consists of 22 items rated on a 7-point severity scale ranging from *no symptoms* (0) to *severe symptoms* (6). These 22 items can be grouped into 4 common symptom clusters (cognitive, physical, affective, and sleep), with headache being a standalone symptom.[23] The clusters consist of the following items: cognitive (feeling slowed down, feeling mentally foggy, difficulty concentrating, difficulty remembering), physical (nausea, vomiting, balance problems, dizziness, sensitivity to light, sensitivity to noise, visual problems), affective (irritability, sadness, nervousness, feeling more emotional), and sleep (fatigue, trouble falling asleep, sleeping less than usual, drowsiness) factors.

Neurocognitive Performance. Athletes completed a hybrid neuropsychological testing battery of traditional paper-and-pencil and computerized measures. We chose the tests for this battery to measure performance across domains of verbal and visual learning and memory, executive functioning, attention, and processing speed as they are known to be sensitive to cognitive deficits after SRC. Similar batteries are commonly used in concussion-management programs.[21,24–26] The computerized measures were the ImPACT and the Vigil/W continuous performance task. The paper-and-pencil tests were the Brief Visuospatial Memory Test–Revised (BVMT-R), the Comprehensive Trail-Making Test (CTMT), a modified version of the Digit Span Test, the Hopkins Verbal Learning Test–Revised (HVLT-R), the Penn State University Cancellation Test, the Stroop Color-Word Test, and the Symbol-Digit Modalities Test (SDMT). Performance on several indices from each of these neuropsychological tests was measured. This test battery has previously been described in detail.[20]

Statistical Analyses

Data Transformations. Scores on all neuropsychological test measures were standardized (mean = 100 ± 15) using published baseline norms from a large sample of collegiate athletes at a Division I university.[21] These norms were used for all test measures except the ImPACT test, which was not included in this normative data set. Norms used for the ImPACT were from an equivalent sample of collegiate athletes at the same Division I university as described earlier.[20] Higher scores always indicated better performance.

Principal component analyses (PCAs) were used to identify and compute composite scores for conceptually related test indices (APS and memory tests). Tests loading above 0.40 were retained based on guidelines from Costello and Osborne.[27] For the PCA of the APS tests, the following indices were included: ImPACT Visual Motor Speed, ImPACT Reaction Time, Vigil Average Delay, CTMT "Simple," CTMT "Executive," Penn

Table 1. Participant Demographics for Aim 1[a]

Variable	Sleep Group		*P* Value
	Sufficient (n = 512)	Insufficient (n = 102)	
	Mean ± SD		
Age, y	18.53 ± 1.10	18.52 ± 0.95	.91
Wechsler Test of Adult Reading Full Scale-IQ estimate	103.36 ± 6.06	103.24 ± 6.08	.86
	No. (%)		
Sex			.12
Male	373 (72.9)	82 (80.4)	
Female	139 (27.1)	20 (19.6)	
Previous concussions, No.			.12
0	287 (56.1)	46 (45.1)	
1	150 (29.3)	38 (37.3)	
2+	72 (14.1)	18 (17.6)	
History of learning disability			.80
Yes	16 (3.1)	3 (3.0)	
No	468 (91.4)	98 (97.0)	
Maybe	2 (0.4)	0 (0.0)	
History of attention-deficit/hyperactivity disorder			.97
Yes	27 (5.3)	6 (5.9)	
No	453 (88.5)	94 (93.1)	
Maybe	6 (1.2)	1 (1.0)	
History of headache treatment			.34
Yes	47 (9.2)	6 (5.9)	
No	433 (54.6)	85 (83.3)	
History of migraine treatment			.80
Yes	23 (4.5)	5 (4.9)	
No	450 (87.9)	86 (83.3)	
History of substance or alcohol use treatment			.42
Yes	2 (0.4)	1 (1.0)	
No	471 (92.0)	90 (88.2)	
Ethnicity			
African American	69 (13.5)	26 (25.5)	
Asian American	4 (0.8)	0 (0.0)	
Biracial or multiracial	10 (2.0)	5 (4.9)	
Caucasian	415 (81.1)	67 (65.7)	
Hispanic American	3 (0.6)	2 (2.0)	
Latin American	3 (0.6)	0 (0.0)	
Other	8 (1.6)	2 (2.0)	
Sport			
Baseball	1 (0.2)	0 (0.0)	
Crew	0 (0.0)	1 (1.0)	
Football	100 (19.5)	39 (38.2)	
Men's basketball	35 (6.8)	11 (10.8)	
Men's ice hockey	49 (9.6)	4 (3.9)	
Men's lacrosse	112 (21.9)	13 (12.7)	
Men's soccer	56 (10.9)	11 (10.8)	
Rugby	1 (0.2)	0 (0.0)	
Softball	1 (0.4)	1 (1.0)	
Volleyball	0 (0.0)	1 (1.0)	
Women's basketball	24 (4.7)	5 (4.9)	
Women's ice hockey	4 (0.8)	0 (0.0)	
Women's lacrosse	38 (7.4)	5 (4.9)	
Women's soccer	68 (13.3)	8 (7.8)	
Wrestling	20 (3.9)	2 (2.0)	
Other	2 (0.4)	1 (1.0)	

[a] Some participants did not answer all questions.

State University Cancellation, Digits Forward and Digits Backward, Stroop 1 and 2 Time, and SDMT Total. Of the 11 tests entered in the analysis, 10 of the variables loaded above 0.40 and were thus retained for the final APS Composite. Digits Backward (0.34) was eliminated. A comparable PCA was conducted with the following memory indices: ImPACT Verbal Memory Composite, ImPACT Visual Memory Composite, BVMT-R Total Immediate and Delayed Recall, and HVLT-R Total Immediate and Delayed Recall. All variables loaded above 0.40 and were retained for the final memory composite. Two neurocognitive composites of mean performance were created using the indices from each test from the battery described herein.

Cognitive variability indices were created for the 2 composites described in the previous paragraph. A measure of IIV that has been commonly used is the intraindividual

Table 2. Participant Demographics for Aim 2[a]

Variable	Sleep Group		*P* Value
	Sufficient (n = 492)	Insufficient (n = 100)	
	Mean ± SD		
Age, y	18.54 ± 1.11	18.53 ± 0.96	.94
Wechsler Test of Adult Reading Full Scale-IQ estimate	103.42 ± 6.08	103.22 ± 6.13	.78
	No. (%)		
Sex			.12
Male	357 (72.6)	80 (80.0)	
Female	135 (27.4)	20 (20.0)	
Previous concussions, No.			.15
0	273 (55.5)	46 (46.0)	
1	147 (29.9)	36 (36.0)	
2+	69 (14.0)	18 (18.0)	
History of learning disability			.79
Yes	16 (3.3)	3 (3.0)	
No	448 (91.1)	96 (96.0)	
Maybe	2 (0.4)	0 (0.0)	
History of attention-deficit/hyperactivity disorder			.52
Yes	26 (5.3)	6 (6.0)	
No	434 (88.2)	93 (93.0)	
Maybe	6 (1.2)	0 (0.0)	
History of headache treatment			.37
Yes	45 (9.1)	6 (6.0)	
No	415 (84.3)	83 (83.0)	
History of migraine treatment			.83
Yes	23 (4.7)	5 (5.0)	
No	430 (87.4)	84 (84.0)	
History of substance or alcohol use treatment			.43
Yes	2 (0.4)	1 (1.0)	
No	451 (91.7)	88 (88.0)	
Ethnicity			
African American	64 (13.0)	25 (25.0)	
Asian American	4 (0.8)	0 (0.0)	
Biracial or multiracial	10 (2.0)	4 (4.0)	
Caucasian	400 (81.3)	67 (67.0)	
Hispanic American	3 (0.6)	2 (2.0)	
Latin American	3 (0.6)	0 (0.0)	
Other	8 (1.6)	2 (2.0)	
Sport			
Baseball	1 (0.2)	0 (0.0)	
Crew	0 (0.0)	1 (1.0)	
Football	92 (18.7)	37 (37.0)	
Men's basketball	34 (6.9)	11 (11.0)	
Men's ice hockey	46 (9.3)	4 (4.0)	
Men's lacrosse	109 (22.2)	13 (13.0)	
Men's soccer	55 (11.2)	11 (11.0)	
Rugby	1 (0.2)	0 (0.0)	
Softball	2 (0.4)	1 (1.0)	
Volleyball	0 (0.0)	1 (1.0)	
Women's basketball	24 (4.9)	5 (5.0)	
Women's ice hockey	4 (0.8)	0 (0.0)	
Women's lacrosse	36 (7.3)	5 (5.0)	
Women's soccer	66 (13.4)	8 (8.0)	
Wrestling	20 (4.1)	2 (2.0)	
Other	2 (0.4)	1 (1.0)	

[a] Some participants did not answer all questions.

standard deviation score (ISD).[28] The ISD can be considered a measure of inconsistent performance and is created by taking the SD of the standard scores across the test battery. An ISD score was calculated for each composite, resulting in 1 IIV score each for the APS composite and memory composite.

Participants were dichotomized into 2 groups based on whether an athlete prospectively went on to sustain an SRC (n = 61) or not (n = 553).

Preliminary Data Analysis. Independent-samples *t* tests were conducted to compare demographic characteristics between sleep groups. The key demographics explored were age, Wechsler Test of Adult Reading Full-Scale IQ estimate, Previous Head Injury Questionnaire total number of concussions, self-reported history of learning disorder or attention-deficit/hyperactivity disorder, and history of treatment for headaches, migraines, or substance or alcohol

use. None of these demographic variables were different between the sleep groups, and therefore, none were retained as covariates. In addition, for our 2 SRC groups (insufficient sleepers who sustained an SRC and sufficient sleepers who sustained an SRC), we compared the time (in days) between baseline and SRC. No differences were present for the days between baseline and SRC for the insufficient (mean = 342.46 ± 246.72 days) or the sufficient (mean = 552.58 ± 509.18; $t_{47} = -1.42$; $P = .16$) group. For all analyses, α levels were set a priori at .05. Effect sizes were measured using η^2 with the following classifications: *small* (0.01), *medium* (0.06), or *large* (0.14) effects.[29]

Hypothesis Testing Analysis. ***Aim 1.*** A χ^2 test of independence was computed to compare the proportions of sufficient and insufficient sleepers who went on to sustain versus not sustain an SRC.

Aim 2. Multivariate analyses of variance (MANOVAs) were conducted to explore group differences in the outcome measures**.** Four MANOVAs allowed for comparisons between the 2 insufficient sleep groups and then the 2 groups that went on to sustain an SRC, according to self-reported and cognitive outcomes.

- **Insufficient sleepers with SRC versus insufficient sleepers without SRC.** To examine self-reported symptoms, we entered 5 dependent variables into this MANOVA: cognitive, affective, physical, and sleep symptom clusters from the PCSS and a headache rating. For cognitive outcomes, 4 dependent variables were entered into the MANOVA: mean memory composite, mean APS composite, memory IIV, and APS IIV.
- **Sufficient sleepers with SRC versus insufficient sleepers with SRC.** We also applied the same 2 MANOVAs to address differences between these groups for self-reported symptoms and cognitive outcomes.

RESULTS

Aim 1

A greater proportion of insufficient sleepers (15.69%) than sufficient sleepers (8.79%) went on to sustain an SRC (χ^2_1 (N = 614) = 4.52, $P = .03$, $\varphi = -0.09$). To rule out the possibility that either group had a greater opportunity to sustain an SRC (ie, more time being followed prospectively during their athletic careers at our university), we conducted follow-up independent-samples *t* tests to evaluate the time between baseline testing and SRC (in days). The 2 groups did not differ ($t_{47} = -1.42$, $P = .16$, $d = 0.18$).

Aim 2

With regard to baseline symptom reporting, insufficient sleepers with SRC and insufficient sleepers without SRC did not differ ($F_{4,94} = 1.44$, $P = .22$, $\eta^2 = 0.07$). Overall, the insufficient sleep group that sustained an SRC performed worse on cognitive outcomes at baseline than the insufficient sleep group that did not sustain an SRC ($F_{4,95} = 3.27$, $P = .02$, $\eta^2 = 0.12$). However, 1 cognitive domain was different between groups: insufficient sleepers who went on to sustain an SRC performed worse on the mean APS composite ($F_{1,98} = 9.31$, $P = .003$, $\eta^2 = 0.09$). The 2 insufficient sleep groups did not differ regarding the mean memory composite ($F_{1,98} = 0.21$, $P = .65$, $\eta^2 = 0.002$) or IIV in either the memory or APS domain ($F_{1,98} = 0.14$, $P = .71$, $\eta^2 = 0.001$ and $F_{1,98} = 0.55$, $P = .46$, $\eta^2 = 0.006$, respectively). Results of the symptom report and cognitive functioning comparisons are provided in Tables 3 and 4, respectively.

Table 3. Multivariate Analysis of Variance Results for Self-Reported Symptoms[a]

Comparison	*F* Value	*P* Value	Partial η^2
Insufficient sleepers with SRC vs insufficient sleepers without SRC			
Overall	1.44	.22	0.07
Cognitive	6.60	.01[b]	0.06
Affective	3.53	.06	0.04
Physical	4.15	.04[b]	0.04
Sleep	3.62	.06	0.04
Headache	2.96	.09	0.03
Sufficient vs insufficient sleepers with SRC			
Overall	4.89	.001[b]	0.31
Cognitive	12.55	.001[b]	0.18
Affective	5.29	.03[b]	0.08
Physical	12.01	.001[b]	0.17
Sleep	21.02	<.001[b]	0.26
Headache	13.08	.001[b]	0.18

Abbreviation: SRC, sport-related concussion.

[a] Partial η^2 effect sizes: *small* (0.01–0.05); *medium* (0.06–0.14); *large* (>0.14).

[b] Significant at the .05 level.

In terms of symptom reporting, the 2 groups that went on to sustain an SRC differed in that the insufficient sleep group described more overall symptoms ($F_{5,55} = 4.89$, $P = .001$, $\eta^2 = 0.31$). The insufficient sleep group that went on to sustain an SRC recounted more symptoms than the sufficient sleep group that went on to sustain an SRC in each of the 4 symptoms clusters: cognitive ($F_{1,59} = 12.55$, $P = .001$, $\eta^2 = 0.18$), affective ($F_{1,59} = 5.29$, $P = .03$, $\eta^2 = 0.08$), physical ($F_{1,59} = 12.01$, $P = .001$, $\eta^2 = 0.17$), and sleep ($F_{1,59} = 21.02$, $P < .001$, $\eta^2 = 0.26$) and in headache ($F_{1,59} = 13.08$, $P = .001$, $\eta^2 = 0.18$). Overall, the effect size of the difference in cognitive functioning between the groups was large, though it did not meet the threshold for statistical significance ($F_{4,54} = 2.31$, $P = .07$, $\eta^2 = 0.15$). Regarding

Table 4. Multivariate Analysis of Variance Results for Cognitive Outcomes[a]

Comparison	*F* Value	*P* Value	Partial η^2
Insufficient sleepers with SRC vs without SRC			
Overall	3.27	.02[b]	0.12
Mean memory composite	0.21	.65	0.002
Mean APS composite	9.31	.003[b]	0.09
Memory IIV	0.14	.71	0.001
APS IIV	0.55	.46	0.006
Sufficient vs insufficient sleepers with SRC			
Overall	2.31	.07	0.15
Mean memory composite	0.06	.81	0.001
Mean APS composite	7.72	.007[b]	0.12
Memory IIV	0.008	.93	0.00
APS IIV	0.66	.42	0.01

Abbreviations: APS, attention and processing speed; IIV, intraindividual standard deviation; SRC, sport-related concussion.

[a] Partial η^2 effect sizes: *small* (0.01–0.05); *medium* (0.06–0.14); *large* (>0.14).

[b] Significant at the .05 level.

specific cognitive outcomes, the group differences were driven by mean performance on the APS composite ($F_{1,57} = 7.72$, $P = .007$, $\eta^2 = 0.12$). No differences were seen between the groups for mean memory performance ($F_{1,57} = 0.06$, $P = .81$, $\eta^2 = 0.001$) or either IIV composite (APS: $F_{1,57} = 0.66$, $P = .42$, $\eta^2 = 0.01$; memory: $F_{1,57} = 0.008$, $P = .93$, $\eta^2 = 0.00$). Results of the symptom report and cognitive functioning comparisons are located in Tables 3 and 4, respectively.

DISCUSSION

The broader literature has identified reduced quality of life, increased stress responsivity, somatic pain, cognitive and memory deficits, and increased risks for hypertension, cardiovascular disease, and morbidity and mortality as some of the long-term consequences of poor sleep.[30] Researchers who specifically investigated the effect of insufficient sleep in college students have mostly explored how insufficient sleep affected baseline symptom reporting and cognitive functioning. Previous authors[8,9] have suggested that in healthy, nonconcussed athletes at baseline, insufficient sleep leads to increased symptom reporting but not objective cognitive deficits. Yet the long-term consequences of insufficient sleep on collegiate student-athletes as a specific population have been less well studied. Our aims were to examine the relationships among insufficient sleep at baseline, the risk for prospectively sustaining an SRC, and the symptom reporting and cognitive performance associated with both factors.

Our hypothesis for aim 1 was supported. When compared at baseline, a greater proportion of athletes reporting insufficient sleep went on to sustain an SRC during their playing careers at our university than did those reporting sufficient sleep. In fact, the insufficient sleepers sustained SRCs at nearly twice the rate of the sufficient sleepers in our sample. It may be that insufficient sleep at baseline reflects broader sleep patterns in athletes and that the cumulative effect of these sleep difficulties increases the risk for SRC. This may occur via more dangerous playing behavior, reduced attention or reaction time while playing sport, or a reduced threshold for brain injury.

Our second aim was to explore baseline factors that might be associated with insufficient sleep at baseline and the likelihood of sustaining an SRC in the future. We conducted 2 sets of group comparisons: 1 among all insufficient sleepers (those who sustained an SRC and those who did not) and 1 among all athletes who went on to sustain an SRC (insufficient sleepers and sufficient sleepers at baseline). Our hypotheses for our second aim were partially supported. The insufficient sleep group that went on to sustain an SRC performed worse on the mean APS composite at baseline than the insufficient sleepers who did not sustain an SRC. This was a medium to large effect size accounting for 9% of the variance in group differences with the insufficient sleepers who did not sustain an SRC. The insufficient sleepers who did and those who did not sustain an SRC were not different in terms of mean memory performance or IIV. We also evaluated symptom reporting and, regardless of whether insufficient sleepers went on to sustain an SRC, at baseline, all described similar levels of symptoms. Given that prior researchers[9,31] demonstrated that insufficient sleep at baseline was associated with reports of the heterogeneous and nonspecific symptoms of an SRC, we expected that our insufficient sleep group would recount a high level of symptoms. However, our results expand our understanding of baseline cognitive functioning in this insufficient sleep group. Prior investigators[8,10] did not identify an association between cognitive deficits and insufficient sleep at baseline. When we differentiated between insufficient sleepers who went on to incur an SRC and those who did not, we found that the former group performed worse on APS measures at baseline. It may be that for a subset of insufficient sleepers, processing speed and attention are impaired and this combination leads to an increased risk of injury.

Consistent with earlier results,[9] among all athletes who went on to sustain an SRC, the insufficient sleep group reported more baseline symptoms in all 4 clusters and headache than the sufficient sleep group. Regarding cognitive outcomes, the 2 groups that went on to sustain an SRC differed only in mean APS performance at baseline: the insufficient sleep group performed worse on tests of APS at baseline than the sufficient sleepers, and the effect size was large. This outcome provides further evidence that reduced attention and slowed processing speed, combined with insufficient sleep, may play roles in the risk for future SRC. Taheri and Arabameri[32] found that, in collegiate male athletes, total sleep deprivation was associated with significantly slower reaction time. Our work demonstrated that total sleep deprivation may not be necessary, as insufficient sleep at baseline may have a cumulative effect leading to reduced processing speed. Overall, when compared with either insufficient sleepers who did not go on to experience an SRC or sufficient sleepers who did, the latter sleep group performed worse at baseline on APS measures.

Several potential explanations exist for why insufficient sleep at baseline may increase the likelihood of sustaining an SRC. Previous authors[12] observed that chronic lack of sleep predicted other types of injuries, including orthopaedic injuries. Taken together, the combination of insufficient sleep, reduced attention, and slowed processing speed, as described in aim 2, may create conditions under which an athlete is more susceptible to an SRC, as we noted for aim 1. This may be the result of lapses in attention potentially increasing the risk for injury.[33,34] Alternatively, as has been suggested for orthopaedic injuries, SRC may be more common due to degraded sport performance, reduced body control, increased difficulty responding on the field, and poor visual tracking and reaction time during in-sport activities, which may be secondary to insufficient sleep and deficits in APS.[12]

These findings have important implications for SRC management, particularly at baseline. Athletes reporting insufficient sleep at baseline may be referred for more comprehensive assessment of the type and nature of their sleep difficulties. These can then be targeted with interventions. It is important to acknowledge the many nonpharmacologic, evidence-based practices to target sleep difficulties, such as cognitive-behavioral therapy for insomnia and sleep hygiene practices.[35]

The current study had several limitations. First, sleep was measured using a single-item, self-reported measure. A single night's sleep may be influenced by idiosyncratic factors the night before baseline testing. However, in our

sample, 197 athletes at baseline completed the Cognitive Health Questionnaire, which asked them to report the "average amount of sleep in a 24-hour period." This average rating of sleep and the rating of the single night's sleep from the ImPACT were moderately positively correlated ($r_{195} = 0.29$, $P < .001$). Although this method is cost-effective and time-effective and has been used in prior research involving student-athletes, polysomnography is the criterion standard objective measure of sleep quantity, and future researchers should focus on objective sleep measurement. Objective measures can provide detailed information regarding both the quantity and quality of sleep, including metrics such as sleep efficiency and sleep architecture. Understanding more about sleep quality will be an important area of future study because this factor is often the first target in evidence-based practices to improve sleep, such as cognitive-behavioral therapy for insomnia. Our method was additionally limited given that a single night's sleep was used to infer athletes' behavior longitudinally. Another limitation was that important elements were cross-sectional, specifically with sleep and cognitive functioning being measured on the same day. Therefore, this limits our ability to draw assumptions about the direction of these relationships, and future investigators may measure sleep over longer periods of time to determine the directionality between poor sleep and deficits in APS. Furthermore, we conducted this study on a sample of majority male, White, Division I collegiate athletes. Thus, these findings may not be generalizable to a more heterogeneous population, and assessment of more diverse populations is warranted.

Overall, our results contribute to the literature and show that insufficient sleep at baseline may have prospective implications for athletes by increasing their risk of sustaining an SRC. In addition, insufficient sleepers with deficits in APS are also at increased risk for sustaining an SRC. Whereas prior authors have focused on baseline differences in cognitive functioning, we were able to prospectively examine this variable among insufficient sleepers who went on to sustain an SRC. The combination of insufficient sleep and worse APS performance was associated with sustaining an SRC and highlights the possibility that sleep may be an important target for primary intervention at baseline to reduce the risk of an SRC.

REFERENCES

1. Irick E. Student-athletes participation 1981–82–2018–19 NCAA sports sponsorship and participation rates report. Accessed February 5, 2023. https://ncaaorg.s3.amazonaws.com/research/sportpart/2018-19RES_SportsSponsorshipParticipationRatesReport.pdf
2. McCrory P, Meeuwisse W, Dvořák J, et al. Consensus statement on concussion in sport—the 5th International Conference on Concussion in Sport held in Berlin, October 2016. *Br J Sports Med*. 2017;51(11):838–847. doi:10.1136/bjsports-2017-097699
3. Scopaz KA, Hatzenbuehler JR. Risk modifiers for concussion and prolonged recovery. *Sports Health*. 2013;5(6):537–541. doi:10.1177/1941738112473059
4. Iverson GL. A biopsychosocial conceptualization of poor outcomes from mild traumatic brain injury. In: Vasterling JJ, Bryant RA, Keane TM, eds. *PTSD and Mild Traumatic Brain Injury*. Guilford Press; 2012:37–60.
5. Kroshus E, Wagner J, Wyrick D, et al. Wake up call for collegiate athlete sleep: narrative review and consensus recommendations from the NCAA Interassociation Task Force on Sleep and Wellness. *Br J Sports Med*. 2019;53(12):731–736. doi:10.1136/bjsports-2019-100590
6. Mah CD, Kezirian EJ, Marcello BM, Dement WC. Poor sleep quality and insufficient sleep of a collegiate student-athlete population. *Sleep Health*. 2018;4(3):251–257. doi:10.1016/j.sleh.2018.02.005
7. Brauer AA, Athey AB, Ross MJ, Grandner MA. Sleep and health among collegiate student athletes. *Chest*. 2019;156(6):1234–1245. doi:10.1016/j.chest.2019.08.1921
8. Mihalik JP, Lengas E, Register-Mihalik JK, Oyama S, Begalle RL, Guskiewicz KM. The effects of sleep quality and sleep quantity on concussion baseline assessment. *Clin J Sport Med*. 2013;23(5):343–348. doi:10.1097/JSM.0b013e318295a834
9. Riegler KE, Guty ET, Thomas GA, Arnett PA. Sleep deprived or concussed? The acute impact of self-reported insufficient sleep in college athletes. *J Int Neuropsychol Soc*. 2021;27(1):35–46. doi:10.1017/S135561772000065X
10. Silverberg ND, Berkner PD, Atkins JE, Zafonte R, Iverson GL. Relationship between short sleep duration and preseason concussion testing. *Clin J Sport Med*. 2016;26(3):226–231. doi:10.1097/JSM.0000000000000241
11. Scullin MK, Bliwise DL. Sleep, cognition, and normal aging: integrating a half century of multidisciplinary research. *Perspect Psychol Sci*. 2015;10(1):97–137. doi:10.1177/1745691614556680
12. Milewski MD, Skaggs DL, Bishop GA, et al. Chronic lack of sleep is associated with increased sports injuries in adolescent athletes. *J Pediatr Orthop*. 2014;34(2):129–133. doi:10.1097/BPO.0000000000000151
13. Sufrinko A, Pearce K, Elbin RJ, et al. The effect of preinjury sleep difficulties on neurocognitive impairment and symptoms after sport-related concussion. *Am J Sports Med*. 2015;43(4):830–838. doi:10.1177/0363546514566193
14. Raikes AC, Athey A, Alfonso-Miller P, Killgore WDS, Grandner MA. Insomnia and daytime sleepiness: risk factors for sports-related concussion. *Sleep Med*. 2019;58:66–74. doi:10.1016/j.sleep.2019.03.008
15. Covassin T, Elbin RJ, Burkhart S, Crutcher B, Kontos A. The relationship between coping, neurocognitive performance, and concussion symptoms in high school and collegiate athletes. *Sport Psychol*. 2013;27(4):372–379. doi:10.1123/tsp.27.4.372
16. LeMonda BC, Tam D, Barr WB, Rabin LA. Assessment trends among neuropsychologists conducting sport-related concussion evaluations. *Dev Neuropsychol*. 2017;42(2):113–126. doi:10.1080/87565641.2016
17. Thomas GA, Guty ET, Riegler KE, Arnett PA. Comorbid affective symptomatology and neurocognitive performance in college athletes. *J Int Neuropsychol Soc*. 2022;28(2):177–187. doi:10.1017/S1355617721000412
18. Lovell MR. Administration and interpretation manual. ImPACT Applications. Published 2016. Accessed February 5, 2023. https://platform-static-files.s3.amazonaws.com/ecb/document/2017/02/07/a05aa3da-93d7-4fff-b9cb-2fb3a89c960f/IMPACT-interpretation-manual.pdf
19. Lauderdale DS, Knutson KL, Yan LL, Liu K, Rathouz PJ. Self-reported and measured sleep duration: how similar are they? *Epidemiology*. 2008;19(6):838–845. doi:10.1097/EDE.0b013e318187a7b0
20. Riegler KE, Guty ET, Arnett PA. Neuropsychological test performance in depressed and nondepressed collegiate athletes following concussion. *Neuropsychology*. 2020;34(1):63–76. doi:10.1037/neu0000582
21. Merritt VC, Meyer JE, Cadden MH, et al. Normative data for a comprehensive neuropsychological test battery used in the assessment of sports-related concussion. *Arch Clin Neuropsychol*. 2017;32(2):168–183. doi:10.1093/arclin/acw090
22. Mild Traumatic Brain Injury Committee, ACRM. Definition of mild traumatic brain injury. *J Head Trauma Rehabil*. 1993;8(3):86–87. doi:10.1097/00001199-199309000-00010

23. Merritt VC, Arnett PA. Premorbid predictors of postconcussion symptoms in collegiate athletes. *J Clin Exp Neuropsychol*. 2014;36(10):1098–1111. doi:10.1080/13803395.2014.983463
24. Echemendia RJ, Thelen J, Meeuwisse W, Comper P, Hutchison MG, Bruce JM. Testing the hybrid battery approach to evaluating sports-related concussion in the National Hockey League: a factor analytic study. *Clin Neuropsychol*. 2020;34(5):899–918. doi:10.1080/13854046.2019.1690051
25. Iverson GL, Schatz P. Advanced topics in neuropsychological assessment following sport-related concussion. *Brain Inj*. 2015;29(2):263–275. doi:10.3109/02699052.2014.965214
26. Kerr ZY, Snook EM, Lynall RC, et al. Concussion-related protocols and preparticipation assessments used for incoming student-athletes in National Collegiate Athletic Association member institutions. *J Athl Train*. 2015;50(11):1174–1181. doi:10.4085/1062-6050-50.11.11
27. Costello AB, Osborne J. Best practices in exploratory factor analysis: four recommendations for getting the most from your analysis. *Pract Assess Res Eval*. 2005;10:1–9. doi:10.7275/JYJ1-4868
28. Burton CL, Strauss E, Hultsch DF, Moll A, Hunter MA. Intraindividual variability as a marker of neurological dysfunction: a comparison of Alzheimer's disease and Parkinson's disease. *J Clin Exp Neuropsychol*. 2006;28(1):67–83. doi:10.1080/13803390490918318
29. Lakens D. Calculating and reporting effect sizes to facilitate cumulative science: a practical primer for t-tests and ANOVAs. *Front Psychol*. 2013;4:863. doi:10.3389/fpsyg.2013.00863
30. Medic G, Wille M, Hemels ME. Short- and long-term health consequences of sleep disruption. *Nat Sci Sleep*. 2017;9:151–161. doi:10.2147/NSS.S134864
31. Iverson GL, Silverberg ND, Mannix R, et al. Factors associated with concussion-like symptom reporting in high school athletes. *JAMA Pediatr*. 2015;169(12):1132–1140. doi:10.1001/jamapediatrics.2015.2374
32. Taheri M, Arabameri E. The effect of sleep deprivation on choice reaction time and anaerobic power of college student athletes. *Asian J Sports Med*. 2012;3(1):15–20. doi:10.5812/asjsm.34719
33. Lim J, Dinges DF. A meta-analysis of the impact of short-term sleep deprivation and cognitive variables. *Psychol Bull*. 2010;136(3):375–389. doi:10.1037/a0018883
34. Waters F, Bucks RS. Neuropsychological effects of sleep loss: implication for neuropsychologists. *J Int Neuropsychol Soc*. 2001;17(4):571–586. doi:10.1017/S1355617711000610
35. Cheung JMY, Jarrin DC, Ballot O, Bharwani A, Morin CM. A systematic review of cognitive behavioral therapy for insomnia implemented in primary care and community settings. *Sleep Med Rev*. 2019;44:23–36. doi:10.1016/j.smrv.2018.11.001

Address correspondence to Kaitlin Elizabeth Riegler, MS, Pennsylvania State University, 372 Moore Building, State College, PA 16802. Address email to kriegler25@gmail.com.