doi:10.4085/1062-6050-0629.24

Title: Acute Changes in Sleep Stages Following Concussion in Collegiate Athletes: A Pilot Study

Authors: Catherine C. Donahue, Ph.D., ATC, [1,2] Laura E. Barnes, Ph.D., [3] Jay N. Hertel, Ph.D., ATC, [4] Jacob E. Resch, Ph.D., ATC [4]

Author Affiliations:
[1] Department of Orthopedics, University of Colorado School of Medicine, Aurora, CO, USA
[2] Sports Medicine Center, Children's Hospital of Colorado, Aurora, CO, USA
[3] Department of Systems and Information Engineering, University of Virginia, Charlottesville, VA, USA
[4] Department of Kinesiology, University of Virginia, Charlottesville, VA, USA

***Corresponding Author:**
Catherine C. Donahue, PhD, ATC
Sports Medicine Center, Children's Hospital Colorado; Department of Orthopedics, University of Colorado School of Medicine
Address: 13123 E. 16th Avenue, B060 Aurora, CO 80045
Email: catherine.donahue@CUAnschutz.edu
X: @CatDonahue
ORCID: 0009-0008-0071-2055

Acknowledgements: This study was partially funded by the Virginia Athletic Trainers Association

ACUTE CHANGES IN SLEEP STAGES FOLLOWING CONCUSSION IN COLLEGIATE ATHLETES: A PILOT STUDY

Context: Sleep has been suggested to be a modifier of recovery following a concussion and is associated with greater symptomatology and number of days until symptom resolution. However, the physiological mechanism for why sleep disturbances occur remains poorly understood. Alterations in time spent in stages of a sleep cycle following a concussion may contribute to recovery.

Objective: The purpose of our study was to use a non-invasive, sensor-derived measure of sleep stages to determine differences between collegiate athletes with or without a concussion, acutely following injury (<72 hours).

Design: Case-control.

Setting: Division 1 collegiate athletics.

Participants: Division 1 Collegiate athletes diagnosed with a concussion were compared to healthy-matched controls based on health history, demographics and sport.

Interventions: Individuals in both groups were provided with and instructed to wear an OURA ring actigraphy device, nightly, within 72 hours of their concussion.

Main Outcome Measures: Differences in sensor-derived time spent in Light, Deep, rapid eye-movement (REM) sleep, time awake, and total sleep time between groups.

Results: A total of 18 athletes were included in our analyses (9 concussed, 9 controls) with an average age of 19.3$\pm$1.3 years. Individuals with a concussion spent less time in deep sleep (113.1±33.1 vs 134.4±51.1 minutes, $p=0.03$), and more time awake (90.22±30.0 vs 49.28±11.5

minutes, p=0.02) compared to individuals without a concussion. No significant differences were found for time spent in total sleep time, Light or REM sleep.

Conclusion: Acutely following concussion, individuals may demonstrate changes in sleep stages. Our results suggest that time spent in different stages of sleep may be a potential mechanism underlying recovery from concussion. Our results provide an important step in using wearable sensors to better understand sleep disturbances following concussion to help mitigate risk of a prolonged recovery.

Key Points:

1. Collegiate athletes with a concussion spend up to 54% more time awake throughout the night compared to healthy controls, suggesting significant sleep disruption in the acute phase of injury.
2. Deep sleep duration was reduced by an average of 21 minutes in concussed athletes, which may impair essential neurophysiological recovery processes, including glymphatic clearance and synaptic plasticity.
3. Our findings provide new evidence that alterations in sleep architecture occur within 72 hours of concussion, highlighting a potential target for monitoring and intervention to optimize recovery.

Word Count: 288/300

INTRODUCTION

Concussion-related sleep disturbances are a known modifier of recovery following a diagnosed concussion. [1–5] Through the use of self-reported symptoms and actigraphy, research has demonstrated that individuals who experience sleep-related symptoms take a greater number of days to report symptom free and ultimately return-to-play following a diagnosed concussion. [2–6] For example, in adolescents (11-18 years of age), a fourfold increase in recovery time was observed if sleep symptoms (e.g.- difficulty falling asleep or staying asleep) were reported in the first week following injury when compared to those that did not endorse the same symptoms. [5] Similarly, collegiate athletes that self-reported sleep symptoms in the acute phase of injury (<72 hours), took longer to experience symptom resolution compared to those that did not report sleep symptoms. [7] While these, and previous studies[1,3,4,6,8–10] have provided foundational work to support sleep as a modifier of recover, they have focused on self-reported sleep disturbances or sensor-derived (e.g., FitBit, Apple Watch) sleep duration or sleep efficiency, overlooking potential changes in sleep architecture (i.e., time spent in different stages of sleep), particularly in the acute phase of concussion. Given the role of sleep in recovery, understanding sleep stage alterations immediately after injury may provide new insights into how sleep influences recovery.

Continued sleep disturbances beyond the typical symptom resolution period (i.e-10-14 days[11]) following a concussion are common and present similarly to trends observed in the acute and sub-acute phases of the injury. More specifically, a decreased sleep efficiency (i.e., the proportion of time spent asleep relative to the total time in bed[12]) and increased time spent awake has been reported up to one year after injury. [6] Research examining sleep disturbances immediately following concussion (<72 hours) remains limited. In one study of collegiate athletes, it was found that those with a concussion demonstrated longer time to fall asleep (15.8 minutes), compared to healthy-matched controls (5.8 minutes).[9] Moreover, those with a concussion demonstrated variability in the number of minutes awake after having initially fallen asleep (i.e.- wake after sleep onset [WASO]) and greater total sleep time, when compared to healthy controls.[9] Though these data support the role of sleep in recovery following concussion, the physiological mechanism of why sleep disturbances occur following injury remain poorly understood but may be linked to the physiological processes that regulate the sleep-wake cycle.

A sleep cycle can be separated into two stages: nonrapid eye movement (NREM), and rapid eye movement (REM).[13] More specifically, a sleep cycle consists of three NREM stages and one REM stage.[13] Typically individuals will experience four to five sleep cycles each night, with each cycle lasting between 70-120 minutes.[13] However, inadequate sleep cycles (i.e., time spent in each stage) can diminish the restorative benefits of sleep. The current gold standard for assessing sleep is polysomnography (PSG). [14] PSG is used to monitor sleep stages and cycles to determine if or when sleep patterns are disrupted and why. Although PSG allows researchers to collect physiologic parameters of sleep, the time and cost of the technology prevent it from being widely used, especially after a concussion.

Due to the barriers associated with PSG, , alternative measures of sleep based on actigraphy, such as a FitBit^{TM} or Apple Watch^{TM}, have been developed and used to indirectly measure sleep.[15–17] However, these devices measure overall time spent asleep as opposed to the different stages of sleep.[15,16] Additionally, the accuracy of wearable devices used to measure sleep has been brought into question. Wearable devices used to measure sleep primarily use a single accelerometer thereby only measuring movement (or lack thereof) to determine when an individual is awake or asleep. Provided this limitation, actigraphy devices, such as FitBit or Apple Watch, have demonstrated to have limited reliability compared to PSG.[16,18] The OURA ring (Oulu, Finland) is a smart ring purported to quantify biometric data specific to sleep such as body temperature, heart rate variability, as well as time spent in the various sleep stages (REM, NREM) through use of three sensors. When compared to PSG, the OURA ring was demonstrated to have high sensitivity (96%) and moderate specificity (48%) for detecting sleep, making it a better option to assess sleep as compared to traditional actigraphy devices. [19]

As mentioned, inadequate sleep cycles (i.e., time spent in each stage) can diminish the restorative benefits of sleep, particularly stage three of NREM.[13,20] Often referred to as deep sleep, this stage is of great importance as it is the primary stage of sleep in which the glymphatic system- a series of perivascular channels throughout the brain that serve to clear away and expel waste in the central nervous system- is active.[21] Disruption in sleep stages, particularly deep sleep, may results in decreased glymphatic clearance of metabolic waste (i.e., cellular debris) from concussion.[21,22] Past research has demonstrated that subjective and objective measures of sleep disturbances such as night time awakenings, increased daytime drowsiness, and reports of insomnia are associated with prolonged recovery from concussion. [1–5,9,23] Despite the advancements in our understanding of sleep as a modifier of recovery, these studies have not identified how stages of sleep may be associated with recovery, particularly in the acute phase of injury (<72 hours). As sleep is a known modifier of recovery following a diagnosed concussion, examining differences in stages of sleep may provide previously unknown evidence that sleep-wake cycles are affected by concussion. Therefore, the purpose of our study is to use a non-invasive, sensor-derived measure of sleep (Oura ring), to observe differences in sleep stages within 72 hours following a diagnosed concussion in collegiate athletes.

METHODS

Participants

This was a case-control study. Data were collected on Division 1, National Collegiate Athletic Association (NCAA) athletes from a single institution who were active in their respective sport, between the 2021-22' and 2022-23' academic years. For the concussed group, athletes were diagnosed with a concussion by an athletic trainer (AT) or team physician. Concussion diagnosis was adopted from the most recent international consensus statement on concussion in sport at the time of diagnosis.[24] Following the diagnosis of a concussion, athletes were referred to study personnel for recruitment within 72 hours post-injury. Participants in both groups were excluded if they had any prior traumatic brain injury (TBI) requiring hospitalization, presence of brain tumor, a concussion within six months of study participation (excluding present concussion), neurologic or neurodevelopmental disorder (epilepsy, dementia, autism, migraine, but not attention-deficit disorder). Additionally, potential participants with a concussion were excluded if they had a Glascow Coma Scale (GCS) score <13 on initial evaluation, traumatic injury requiring intensive care unit (ICU) monitoring or operative repair, a structural abnormality on brain CT (if obtained), or any additional comorbidity (e.g.- ACL tear).

All participants signed an informed consent form prior to participation in this study. This study was approved by the university's institutional review board.

Measures

Demographic Measurements

Basic demographic information collected consisted of biological sex, age, height, weight, sport, and position played. This information was collected as part of a health history form and was the basis of how participants with a concussion were matched with their respective control subjects.

OURA Ring

The OURA ring is consumer based health tracking device that measures body signals (i.e.- heart rate, heart rate variability, temperature), as well as sleep-specific metrics (e.g.- time in light, deep, REM sleep, and total sleep time (amount of time an individual spends asleep during a planned sleep period). [19] Aforementioned body signals are measured via infared photoplethysmography (PPG), negative temperature coefficient (NTC), and a triaxial accelerometer. Accelerometer data is sampled at a frequency of 50Hz.[19] Sleep is measured in five minute epochs, with sleep onset being defined as the first five consecutive minutes of persistent sleep. Previous studies have demonstrated a sensitivity (ability to detect sleep) of 96%, and a specificity (ability to detect wake) of 48% in the OURA ring when compared to PSG. [19]

Revised Head Injury Scale

The Revised Head Injury Scale (HIS-r) consists of 22 symptoms related to concussion, and measures symptom quantity, duration, and severity over a 24-hour period. To complete the HIS-r, participants first indicated which of the 22 symptoms (yes/no) they had experienced during the previous 24 hours that was atypical for them. For each symptom endorsed, the participant then rated the symptom duration and severity over the past 24 hours. Duration is rated on a Likert scale from 1 “briefly” to 6 “always”. Severity is rated on a Likert scale that ranges from 0 “not severe” to 6 “as severe as possible”. In addition to the total number of symptoms endorsed (ranging from 0 to 22), total symptom duration and total symptom severity scores were calculated by summing the respective individual duration and severity scores for each endorsed symptom, resulting in total duration and severity scores each ranging from 0 to 132. The HIS-r has been demonstrated to have high sensitivity (77.5%) and specificity (100%) in recognizing collegiate athletes diagnosed with a concussion acutely after injury.[25]

Procedure

Upon diagnosis of a concussion by the athlete’s AT or team physician, participants in both groups were recruited within 72 hours of injury. Upon successful recruitment, a control participant was identified and matched to their injured counterpart by sport, position played, height, weight, age, and the absence of acute medical complications (i.e., illness or musculoskeletal injury) or an active injury. Participants were then sized for an OURA ring to be worn on their second, third, or fourth digit (on either hand) and instructed to wear it nightly. [19] Additionally, at this time participants were instructed to download the OURA ring application on their personal smartphone. The application allowed for continuous collection of data from the ring while being encrypted. OURA system preferences were set so that participants could not see any of their sleep data in the application. Sleep data were automatically processed through Oura’s proprietary algorithm, as such the assessment of all sleep variables was identical for all participants. The research team acquired the data by exporting it from Oura’s cloud-based data platform. Once exported, the data were securely stored in a HIPAA-compliant digital repository before being processed and analyzed. OURA ring sleep parameters examined in this study included time (in minutes) spent in Light sleep, Deep sleep, REM sleep, awake, and total sleep time, in the first 72 hours following their injury.

Statistical Analysis

Independent T-tests were performed to determine if duration of time in Light, Deep, or REM sleep, time awake throughout night, and total sleep time, as measured by the Oura ring in the first 72 hours of injury was statistically different between groups. Cohen's d effect size were calculated to determine between group differences in stages of sleep and interpreted as large (>0.8), medium (0.50-0-79), or small (0.20-0.49). [26] Data were analyzed using SPSS version 26.0 (Armonk, NY) with α=0.05.

RESULTS

A total of 18 collegiate athletes (8 female, 10 male) were included in the study (9 concussed, 9 healthy matched control),) with an average age of 19.3$\pm$1.3 years. Individuals with a concussion demonstrated an initial symptom severity of 30.1 (out of 132,measured via HIS-r) and took approximately 10.0 ±10.5 days to self-report asymptomatic. Day 1 (i.e., <24 hours) was not included in the final analysis due to a small recruitment sample, thus, analysis of sleep data was the mean of Days 2 and 3. Significant differences in time spent awake were found between groups, with individuals with a concussion spending an average of 90.22±30.0 minutes awake, compared to matched controls who spent an average of 49.28± 11.5 minutes awake (t=-3.65,p=0.021, Cohen's d= -1.8) Additionally, differences were observed in deep sleep duration, with individuals in the concussed group spending significantly less time (113.1± 33.1 minutes) than matched controls (134.4± 51.1 minutes[t=1.36, p=0.027, Cohen's d=.50]). No significant differences between groups were observed in duration of Light sleep (p=0.31), REM sleep (p=0.09), or total sleep time (p=0.20). (Table 1.)

DISCUSSION

Our study is the first of its kind to examine the duration of NREM and REM sleep stages in the acute phase of a concussion using a commercially available smart ring to monitor sleep. Our results suggest that collegiate athletes with a concussion spend significantly more time awake- approximately 54% more on average- compared to athletes without a concussion, with a large effect size observed for this difference. Additionally, athletes with a concussion demonstrated less time in deep sleep, which may have important implications for recovery given the role of deep sleep in cellular repair.[21] Importantly, our results suggest that time spent in different sleep stages may be a potential mechanism underlying recovery from concussion that had not previously been examined in the collegiate athlete population, however further research is needed in a larger, more diverse sample to confirm these findings. Clinicians should consider monitoring post-injury sleep disturbances, as increased wakefulness and reduced deep sleep may contribute to prolonged recovery and impact return-to-play decisions.

In our study, there were no significant differences between groups for total sleep time, with both groups demonstrating an average of 6.5-7 hours of sleep. This finding is in line with previous research in which total sleep time differences were not observed between individuals with and without a concussion.[3,6,8,9,23,27–29] This suggests that total sleep time may not fully capture sleep-related differences following a concussion, but rather the length of time spent in different stages may be more relevant to understanding how sleep is altered after concussion. An individual with a concussion may demonstrate the same hours of sleep as an individual without a concussion, and as such total sleep time does not fully explain the frequency or duration of sleep stages that comprised the total sleep time. Additionally, we observed that individuals with a concussion spent more time awake during the night than those without a concussion. Following a concussion, subjective reports of frequent nighttime wakenings and increased time spent awake via actigraphy are commonly seen throughout research.[3,5,6,23] However, many of these studies included a wide range of time from injury to enrollment, ranging from weeks to months, rather than acutely after injury. [1,3–6,23]

The decreased duration of deep sleep in the concussed group immediately following injury may be explained by factors related to sleep regulation and physiology not analyzed in this study. The regulation of sleep is driven by the interaction of two separate biological mechanisms: the homeostatic need for sleep (Process S), and the circadian timing of when sleep happens (Process C).[13,20,30–32] The regulation of sleep and wakefulness by Process S and Process C is referred to as the 2 Process Model, and is reflective of the inhibition or activation of various neurotransmitters.[13,30,31] The excitement of sleep-promoting neurons or inhibition of wake-promoting neurons may prompt sleep. Conversely, waking can be promoted through the excitement of wake-promoting neurons and or the inhibition of sleep-promoting neurons.[13,20,31,32] Specifically, during sleep, various neurotransmitters are released throughout each stage that promote and maintain sleep.[31] These include, histamine, acetylcholine, serotonin, norepinephrine, dopamine, hypocrite, and gamma-Aminobutyric acid (GABA).[31] Changes in the inhibition or activation of these neurotransmitters can lead to a either a decreased or an increased amount of time spent in a specific sleep stage, as well as frequency of that stage.[13,31] Disrupted sleep stages, particularly stage three of non-rapid eye movement (NREM) otherwise known as deep sleep, are reflective of decreased homeostatic drive for sleep (Process S).[33] This stage of deep sleep is of great importance as it is the primary stage of sleep in which the glymphatic system is active.[33] The glymphatic system is a series of perivascular channels throughout the brain that serve to clear away and expel waste in the central nervous system (CNS) (Figure 1.). [21,34]Disruptions in sleep stages and their duration, particularly deep sleep, may therefore result in decreased glymphatic clearance of metabolic waste (i.e.- cellular debris) following injury. However, further research is needed to confirm this in a concussed population.

Though the use of the Oura ring in the present study has provided novel insight into sleep architecture following concussion, it is not yet feasible to use a routine clinical tool for concussion management. Despite this, our findings still have meaningful implications for concussion management. Given our results that individuals with concussion spent increased time awake and decreased time in deep sleep, clinicians can integrate subjective sleep assessments into standard care. Incorporating sleep diaries[35], validated questionnaires such as the Pittsburgh Sleep Quality Index (PSQI)[36], or directly asking athletes about their sleep, may help identify those at risk for prolonged recovery. Moreover, incorporating these assessments may serve as an early indication that sleep disturbances may be contributing to persisting symptoms. Finally, as deep sleep plays a vital role in neural recovery, mood regulation, and cognition, clinicians can place an emphasis on strategies that aid in promoting good sleep quality such as maintaining a consistent sleep schedule or limiting screen time before bedtime.[37]

This study was not without limitations. Our sample size was small, however given the limited number of prospective studies assessing sleep in the acute phase of a concussion, this study does provide valuable information regarding duration of sleep stages in individuals with or without a concussion. Second, we were unable to capture participant's sleep patterns prior to injury and as such can only draw limited conclusions by comparing to their healthy control.

Additionally, we acknowledge that being a consumer wearable device, the OURA ring is limited in its accuracy of measurement.

CONCLUSION

This study extends the current literature by using a commercially available, wearable device to assess sleep acutely following concussion, during which there is limited data. Individuals with a concussion demonstrated decreased duration of deep sleep and increased time spent awake during the acute phase after concussion. These findings provide new insight into the underlying mechanism by which sleep influences recovery from concussion.

ACKNOWLEDGMENTS

This study was partially funded by the Virginia Athletic Trainers' Association.

REFERENCES

1. Tham SW, Aaron R, Palermo TM. The Role of Sleep Deficiency in the Trajectory of Postconcussive Symptoms in Adolescents. *Brain Inj*. 2019;33(11):1413-1419. doi:10.1080/02699052.2019.1643921

2. Kostyun RO, Milewski MD, Hafeez I. Sleep Disturbance and Neurocognitive Function During the Recovery From a Sport-Related Concussion in Adolescents. *Am J Sports Med*. 2015;43(3):633-640. doi:10.1177/0363546514560727

3. Chung JS, Zynda AJ, Didehbani N, et al. Association Between Sleep Quality and Recovery Following Sport-Related Concussion in Pediatrics. *J Child Neurol*. 2019;34(11):639-645. doi:10.1177/0883073819849741

4. Murdaugh DL, Ono KE, Reisner A, Burns TG. Assessment of Sleep Quantity and Sleep Disturbances During Recovery From Sports-Related Concussion in Youth Athletes. *Arch Phys Med Rehabil*. 2018;99(5):960-966. doi:10.1016/j.apmr.2018.01.005

5. Bramley H, Henson A, Lewis MM, Kong L, Stetter C, Silvis M. Sleep Disturbance Following Concussion Is a Risk Factor for a Prolonged Recovery. *Clin Pediatr (Phila)*. 2017;56(14):1280-1285. doi:10.1177/0009922816681603

6. Tham SW, Fales J, Palermo TM. Subjective and Objective Assessment of Sleep in Adolescents with Mild Traumatic Brain Injury. *J Neurotrauma*. 2015;32(11):847-852. doi:10.1089/neu.2014.3559

7. Donahue CC, Walton SR, Oldham JR, et al. Influence of sleep symptoms on recovery from concussion in collegiate athletes: a LIMBIC MATARS consortium investigation. *Brain Inj*. Published online April 28, 2024:1-7. doi:10.1080/02699052.2024.2347542

8. Raikes AC, Schaefer SY. Sleep Quantity and Quality during Acute Concussion: A Pilot Study. *Sleep*. 2016;39(12):2141-2147. doi:10.5665/sleep.6314

9. Hoffman NL, O'Connor PJ, Schmidt MD, Lynall RC, Schmidt JD. Differences in sleep between concussed and nonconcussed college students: a matched case–control study. *Sleep*. 2019;42(zsy222). doi:10.1093/sleep/zsy222

10. Hoffman NL, O'Connor PJ, Schmidt MD, Lynall RC, Schmidt JD. Relationships between Post-Concussion Sleep and Symptom Recovery: A Preliminary Study. *J Neurotrauma*. 2020;37(8):1029-1036. doi:10.1089/neu.2019.6761

11. Patricios JS, Schneider KJ, Dvorak J, et al. Consensus statement on concussion in sport: the 6th International Conference on Concussion in Sport–Amsterdam, October 2022. *Br J Sports Med*. 2023;57(11):695-711. doi:10.1136/bjsports-2023-106898

12. Reed DL, Sacco WP. Measuring Sleep Efficiency: What Should the Denominator Be? *J Clin Sleep Med JCSM Off Publ Am Acad Sleep Med*. 2016;12(2):263-266. doi:10.5664/jcsm.5498

13. Colten HR, Altevogt BM, Research I of M (US) C on SM and. *Sleep Physiology*. National Academies Press (US); 2006. Accessed January 31, 2021. https://www.ncbi.nlm.nih.gov/books/NBK19956/

14. Rundo JV, Downey R. Chapter 25 - Polysomnography. In: Levin KH, Chauvel P, eds. *Handbook of Clinical Neurology*. Vol 160. Clinical Neurophysiology: Basis and Technical Aspects. Elsevier; 2019:381-392. doi:10.1016/B978-0-444-64032-1.00025-4

15. Van de Water ATM, Holmes A, Hurley DA. Objective measurements of sleep for non-laboratory settings as alternatives to polysomnography--a systematic review. *J Sleep Res*. 2011;20(1 Pt 2):183-200. doi:10.1111/j.1365-2869.2009.00814.x

16. Halson SL. Sleep Monitoring in Athletes: Motivation, Methods, Miscalculations and Why it Matters. *Sports Med Auckl NZ*. 2019;49(10):1487-1497. doi:10.1007/s40279-019-01119-4

17. Towns SJ, Zeitzer J, Kamper J, et al. Implementation of Actigraphy in Acute Traumatic Brain Injury (TBI) Neurorehabilitation Admissions: A Veterans Administration TBI Model Systems Feasibility Study. *PM&R*. 2016;8(11):1046-1054. doi:10.1016/j.pmrj.2016.04.005

18. Mantua J, Gravel N, Spencer RMC. Reliability of Sleep Measures from Four Personal Health Monitoring Devices Compared to Research-Based Actigraphy and Polysomnography. *Sensors*. 2016;16(5). doi:10.3390/s16050646

19. de Zambotti M, Rosas L, Colrain IM, Baker FC. The Sleep of the Ring: Comparison of the ŌURASleep TrackerAgainst Polysomnography. *Behav Sleep Med*. 2019;17(2):124-136. doi:10.1080/15402002.2017.1300587

20. Achermann P. The two-process model of sleep regulation revisited. *Aviat Space Environ Med*. 2004;75(3 Suppl):A37-43.

21. Jessen NA, Munk ASF, Lundgaard I, Nedergaard M. The Glymphatic System – A Beginner's Guide. *Neurochem Res*. 2015;40(12):2583-2599. doi:10.1007/s11064-015-1581-6

22. Piantino J, Schwartz DL, Luther M, et al. Link between Mild Traumatic Brain Injury, Poor Sleep, and Magnetic Resonance Imaging: Visible Perivascular Spaces in Veterans. *J Neurotrauma*. Published online April 20, 2021:neu.2020.7447. doi:10.1089/neu.2020.7447

23. Gosselin N, Lassonde M, Petit D, et al. Sleep following sport-related concussions. *Sleep Med*. 2009;10(1):35-46. doi:10.1016/j.sleep.2007.11.023

24. McCrory P, Meeuwisse W, Dvorak 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

25. Resch JE, Brown CN, Schmidt J, et al. The sensitivity and specificity of clinical measures of sport concussion: three tests are better than one. *BMJ Open Sport Exerc Med*. 2016;2(1):e000012. doi:10.1136/bmjsem-2015-000012

26. 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

27. Ouellet MC, Morin CM. Subjective and objective measures of insomnia in the context of traumatic brain injury: a preliminary study. *Sleep Med*. 2006;7(6):486-497. doi:10.1016/j.sleep.2006.03.017

28. Rao V, Bergey A, Hill H, Efron D, McCann U. Sleep Disturbance After Mild Traumatic Brain Injury: Indicator of Injury? *J Neuropsychiatry Clin Neurosci*. 2011;23(2):201-205. doi:10.1176/jnp.23.2.jnp201

29. Milroy G, Dorris L, McMillan TM. Sleep disturbances following mild traumatic brain injury in childhood. *J Pediatr Psychol*. 2008;33(3):242-247. doi:10.1093/jpepsy/jsm099

30. Schwartz JRL, Roth T. Neurophysiology of Sleep and Wakefulness: Basic Science and Clinical Implications. *Curr Neuropharmacol*. 2008;6(4):367-378. doi:10.2174/157015908787386050

31. Siegel JM. The Neurotransmitters of Sleep. *J Clin Psychiatry*. 2004;65(Suppl 16):4-7.

32. Borbély AA, Achermann P. Sleep homeostasis and models of sleep regulation. *J Biol Rhythms*. 1999;14(6):557-568. doi:10.1177/074873099129000894

33. Yan T, Qiu Y, Yu X, Yang L. Glymphatic Dysfunction: A Bridge Between Sleep Disturbance and Mood Disorders. *Front Psychiatry*. 2021;12. doi:10.3389/fpsyt.2021.658340

34. Lundgaard I, Lu ML, Yang E, et al. Glymphatic clearance controls state-dependent changes in brain lactate concentration. *J Cereb Blood Flow Metab*. 2017;37(6):2112-2124. doi:10.1177/0271678X16661202

35. Borba D de A, Reis RS, Lima PHT de M, et al. How many days are needed for a reliable assessment by the Sleep Diary? *Sleep Sci*. 2020;13(1):49-53. doi:10.5935/1984-0063.20190131

36. Buysse DJ, Reynolds CF, Monk TH, Berman SR, Kupfer DJ. The Pittsburgh Sleep Quality Index: a new instrument for psychiatric practice and research. *Psychiatry Res*. 1989;28(2):193-213. doi:10.1016/0165-1781(89)90047-4

37. Sletten TL, Weaver MD, Foster RG, et al. The importance of sleep regularity: a consensus statement of the National Sleep Foundation sleep timing and variability panel. *Sleep Health J Natl Sleep Found*. 2023;9(6):801-820. doi:10.1016/j.sleh.2023.07.016

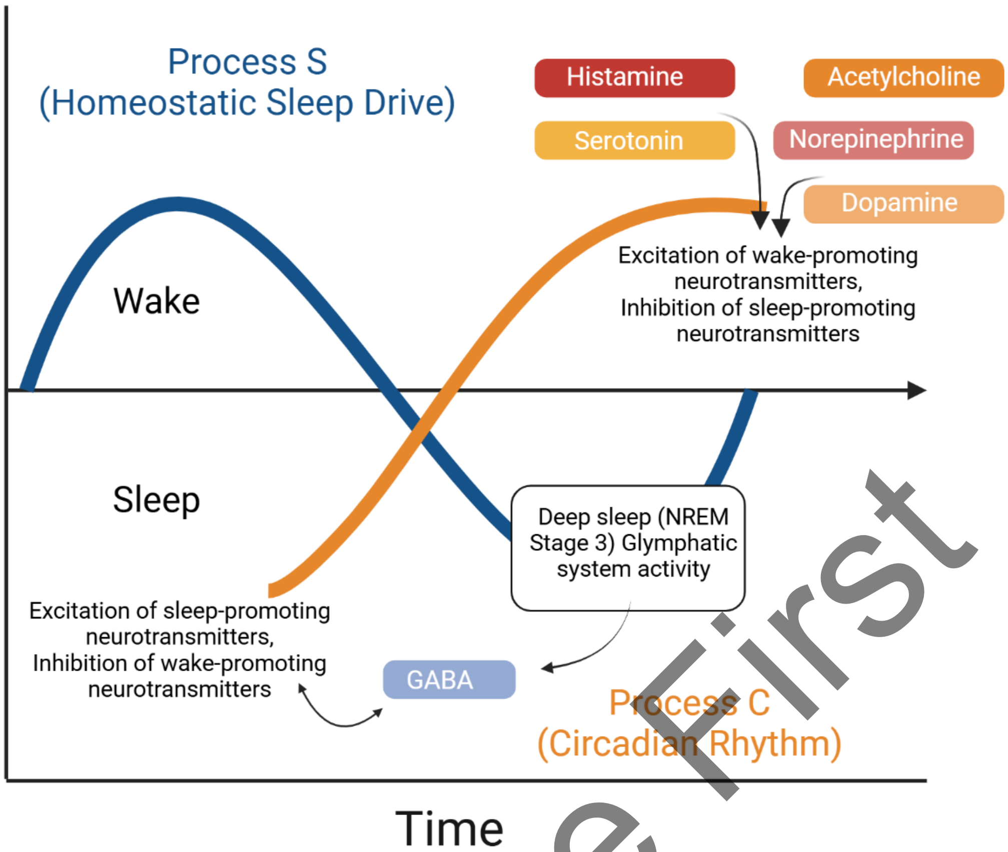
TABLES

Table 1. Average time spent (minutes) in Light, Deep, REM sleep, time awake, and total sleep time between groups <72 hours after injury.
** $p<0.05$

	Concussed	**Control**	***p***	**Cohen's d**	95% Confidence Interval	
					Lower	**Upper**
Time in minutes						
Light Sleep	247.61±71.4	208.28±72.6	0.307	-.533	-0.42	1.46
Deep Sleep	113.11±33.1	134.44±51.1	0.027*	-.500	-1.43	-0.45
REM	46.94±35.1	43.44±32.8	0.096	-.120	-0.81	1.04
Awake	90.22±30.0	49.28±11.5	0.021*	1.82	0.69	2.92
Total sleep time	407.66±82.2	386.16±112.1	0.201	-0.24	-1.145	.709

FIGURES

Figure 1. The Two-Process Model of Sleep, illustrating the interaction between the homeostatic sleep drive (Process S) and the circadian rhythm (Process C) in regulating sleep and wakefulness. Neurotransmitters involved in sleep-wake regulation are shown, with wake-promoting neurotransmitters (histamine, acetylcholine, serotonin, norepinephrine, dopamine, and hypocretin) facilitating alertness and sleep-promoting neurotransmitters (GABA) contributing to sleep onset and maintenance. Disruptions in these processes, such as those observed following concussion, may alter sleep architecture, particularly deep sleep, which plays a crucial role in neurophysiological recovery and glymphatic system function. *Created in BioRender. Donahue, C. (2025) https://BioRender.com/uvzpsx4*


Process S
(Homeostatic Sleep Drive)
Histamine
Acetylcholine
Serotonin
Norepinephrine
Dopamine
Excitation of wake-promoting neurotransmitters,
Inhibition of sleep-promoting neurotransmitters
Wake
Sleep
Deep sleep (NREM Stage 3) Glymphatic system activity
Excitation of sleep-promoting neurotransmitters,
Inhibition of wake-promoting neurotransmitters
GABA
Process C
(Circadian Rhythm)
Time