
Research Submissions

Comprehensive Headache Experience in Collegiate Student-Athletes: An Initial Report From the NCAA Headache Task Force

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Background.—The prevalence of primary headache disorders in the general population provides a unique challenge in the evaluation of headache occurring in the context of sport. Despite a wealth of studies exploring the epidemiology of headache in the layperson, little is known about the prevalence and nature of headaches in collegiate student-athletes. These scenarios are challenging in the return to play context, as it is often unclear whether an athlete has an exacerbation of a primary headache disorder, new onset headache unrelated to trauma, or has suffered a concussive injury.

Purpose.—To establish the prevalence and nature of headaches in collegiate student-athletes.

Study Design.—Retrospective cross-sectional survey.

Methods.—This cross-sectional survey evaluated the characteristics and prevalence of headache in 834 student-athletes from four NCAA Division-I institutions. Because headache occurrence may vary by sport (collision, contact, non-contact), by sex, and medical history, our sample included male and female athletes in a variety of sports, with differing degrees of contact exposure. The 20 question survey collected data on personal and family history of headache, as well as concussion history.

Results.—A total of 23.7% (n = 198) of participants reported having a personal history of migraine, 25.2% (n = 210) history of sinus headache, and 12.3% (n = 103) history of tension type headache. Among athletes with a prior history of concussion, 46.3% (n = 25) of females reported a history of migraine, while only 32.2% of males reported history of migraine ($\chi^2 = 3.421$, $P = .064$).

Conclusions.—The etiology of increased prevalence of migraine in our study is unclear. Whether this is due to increased awareness of headache disorders, a consequence of contact exposure, or a predisposition for migraine development in this age group remains unclear. Further studies are indicated.

Key words: headache, college, NCAA, concussion, migraine

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What is known about this subject: Despite a wealth of studies exploring the epidemiology of headache in the layperson, little is known about the prevalence and nature of headaches in collegiate student-athletes.

What this study adds to existing knowledge: This study provides a much better (and current) understanding of the prevalence and nature of headaches in collegiate student-athletes. To our knowledge, it is the largest study of its type in this unique patient population.

INTRODUCTION

Little is known about the spectrum of headache disorders in collegiate student-athletes. Headache is a common neurological complaint in both traumatic and nontraumatic settings. Sport and exercise-related headaches have received increased attention over the last decade, largely through increased awareness of concussive injury in sport and the military. Despite this renewed interest in posttraumatic headache (PTH), other nontraumatic headaches occurring in sport are often misdiagnosed or overlooked altogether.¹ These scenarios can complicate return to play (RTP) decisions, as it is often unclear whether an athlete has an exacerbation of a primary headache disorder, new onset headache unrelated to trauma, or is in the recovery phase following concussion.² Despite headache occurring frequently in competitive athletes, very few epidemiological studies of sports-related headache have been undertaken in the collegiate athlete population and their prevalence and nature of primary, headaches has remained largely uncertain.³

Per accepted headache classification, primary headaches (ie, “benign” headaches) are those headaches that generally are chronic processes

characterized by repeated acute exacerbations.⁴ Specific neuromodulating networks mediate such headaches via underlying neurobiological mechanisms and dural neurogenic inflammation patterns.⁵ Secondary headaches are due to any number of conditions (including trauma) that subsequently activate the pain-sensitive structures of the head.

There are several primary headache diagnoses that may be overlooked in collegiate student-athletes. According to the Centers for Disease Control (CDC), 14.2% of young adults reported at least one severe headache across a 3-month span.⁶ Tension type headache (TTH) and migraine are the most commonly diagnosed primary headache disorders and affect up to 80% of the general population.⁷ Both of these primary headache disorders are more common in females and peak onset is adolescence or early adulthood.^{8,9} TTH is characterized by mild to moderate headache intensity and described as a dull, tightening sensation.⁴ In contrast, migraine is typically characterized by unilateral, moderate-severe throbbing headache accompanied by typical migrainous features, such as nausea, vomiting, or exacerbation with routine physical activity, and sensory sensitivities, such as photophobia and phonophobia.⁴ One study that found 2.9% of male and female Division-I NCAA basketball players met diagnostic criteria for migraine.¹⁰ However, the study was limited by sampling only one sport, and other headaches were not evaluated. Up to 90% of headache sufferers who complain of “sinus headaches” actually meet criteria for migraine.^{11,12} The prevalence of primary exercise headache, which arises from sustained physical activity, is as high as 12% in the general population.¹³

Other headache disorders that may be relevant to athletes include medication overuse, cervicogenic, and cluster headaches. PTH is a secondary headache that develops following trauma to the head and neck, and can be acute or persistent, if lasting beyond 3 months.⁴ Although PTH characteristics may vary, they most commonly demonstrate a migraine phenotype.¹⁴ Common behavioral or lifestyle factors among college students that trigger or exacerbate primary and secondary headaches

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include stress or negative effect, certain foods,¹⁵ and sleep restriction.¹⁶

Headache is one of the top 10 disabling conditions in the US and can greatly interfere with overall quality of life.^{6,17,18} Several aspects of student-athlete life are vulnerable to disruption, including sport performance, academics, and social life. One study found physical exercise was a headache trigger for 22% of migraine patients¹⁹ and 2% of the population have discontinued sports participation altogether due to primary exertional headache.¹⁵ College students diagnosed with migraine and TTH are more likely to miss class,^{20,21} and a negative relationship between pain intensity and academic performance was apparent in students that met criteria for migraine.²⁰ College students with migraines also have higher rates of comorbid psychiatric diagnoses and report lower quality of life across domains of role functioning, social functioning, mental health, pain, and health perception.²¹

Although headaches can interfere with several aspects of collegiate student athlete's life, headaches are also ubiquitous and athletes may perceive them as a "normal experience" or too trivial to report.²² Primary headache disorders may not be regularly assessed in pre-participation physicals.²³ Similarly, athletic trainers may not document headache unless it is associated with another injury or medical condition, such as PTH following concussion, or if the athlete is seeking over the counter pain medications. Traditionally, only injuries or illnesses that caused restricted participation are reported in the literature or in the NCAA-Injury Surveillance System (the forerunner of the current system), another reason why detailed headache assessment is omitted.²⁴ Therefore, the purpose of the current study was to learn more about headache in collegiate student-athletes by administering a targeted headache questionnaire to a cross-sectional sample of NCAA Division-I student athletes.

METHODS

Sample Selection.—Participating teams were drawn from current NCAA Injury Surveillance Program (ISP) participants. The head athletic trainer

or team physician from participating teams informed the team of the study, explaining the ultimate goal of establishing the prevalence of specific headache types in the collegiate athlete, including both injury/sport-related and those presumed not sport-related (ie, migraine/tension type/sinus).

The number of athletes required was based on known headache rates^{6,17} and the desired goal of the project: to establish the current prevalence of various headache types in NCAA Division-I student athletes. We pursued a smaller sample size in this study to determine the feasibility of future prospective investigations. The current study was completed from July 1, 2013 to June 30, 2014 via survey of contact/noncontact-risk sports in both male and female athletes at four randomly selected institutions: the University of Pittsburgh, University of Kentucky, West Virginia University, and Western Kentucky University. The University of Kentucky Institutional Review Board approved the study protocol and all volunteer participants provided written informed consent.

Study Design.—The subset of the NCAA ISP data allowed for a cross-sectional survey and the (1) calculation of the total headache rate and (2) comparison of headache sufferers' demographics, as well as (3) headache history based on gender, injury mechanics, and concussion(s).

Instrumentation.—A modified questionnaire devised through references obtained at the NINDS Common Data Elements website for Headache Research served as the primary data collection instrument²⁵ (Supporting Information Appendix A1).

Data Analysis.—The variables were summarized using percentages. Chi-square tests were used to determine differences between the groups of interest. Significance was defined as $P < .05$. All analyses were performed using SAS (SAS 9.4, Cary, NC, USA).

RESULTS

Nine hundred and twenty student athletes were targeted to participate in this study. All varsity members of each respective team were eligible for participation. A total of 834 student athletes (597 males and 237 females) from four Division-I

Table 1.—Percentage Reporting Each Headache Type

	Frequency	Percent
Migraine	198	23.7%
Tension	103	12.3%
Sinus	210	25.2%
Other	51	6.1%
Never diagnosed	85	10.2%
No headache hx	187	22.4%

institutions completed the survey, representing a response rate of 90.7%. These included participants from the following sports: football, softball, volleyball, baseball, men's and women's basketball, men's and women's diving, men's and women's golf, men's gymnastics, men's and women's soccer, men's and women's swimming, men's and women's tennis, and men's and women's track and field.

A total of 23.7% ($\pm 2.9\%$ 95% CI, $n = 198$) of participants reported having a history of migraine (either self-diagnosed or diagnosed by a medical professional), including 27.8% ($n = 118$) of contact sports participants and 22.9% ($n = 80$) of noncontact sports participants ($X^2 = 2.490$, $P = .115$). The authors considered only those athletes as having migraine if that specific option was checked under headache diagnosis on the provided survey. A total of 25.2% ($\pm 2.9\%$ 95% CI, $n = 210$) of participants reported a history of sinus headache. Other headache types reported by participants included tension type headache (12.3% $\pm 2.9\%$ 95% CI, $n = 103$) and "other – not specified" (6.1% $\pm 2.9\%$ 95% CI, $n = 51$). Of note, some participants self-reported experiencing multiple subtypes of headache (Table 1).

Among participants that indicated a headache history, 7.3% ($\pm 2.9\%$ 95% CI, $n = 61$) reported

averaging a headache frequency of 9 or greater days/month over the previous 3 months. A total of 28.3% ($\pm 2.9\%$ 95% CI, $n = 236$) of participants reported their headaches to be of moderate/severe intensity. Nearly 60% ($\pm 2.9\%$ 95% CI, $n = 484$) of participants described the pain from their headache as throbbing/pounding/pulsating. A total of 33.5% ($\pm 2.9\%$ 95% CI, $n = 280$) of participants reported functioning at or below 75% of maximum during sport with concurrent headache. This number increased to 46.3% ($\pm 2.9\%$ 95% CI, $n = 386$) functioning at or below 75% of maximum during class when headache was present.

Among participants indicating a prior history of concussion, 35.9% ($\pm 2.9\%$ 95% CI, $n = 74$) also reported a history of migraine compared to 21.8% ($n = 137$) with no concussion history ($X^2 = 15.768$, $P < .001$). Likewise, 32.0% ($n = 66$) of those with a history of prior concussion indicated having sinus headache, compared to 25.5% ($n = 160$) of those with no prior concussions ($X^2 = 3.419$, $P = .065$). 14.6% ($n = 30$) reported headache frequency of 9 or greater days/month. 34.0% ($\pm 2.9\%$ 95% CI, $n = 70$) reported their headaches to be of moderate/severe intensity. 85.9% ($\pm 2.9\%$ 95% CI, $n = 177$) described the pain as throbbing/pounding/pulsating. Of those reporting a prior history of multiple concussions, 40.4% ($n = 38$) reported a history of migraine ($X^2 = 12.3841$, $P < 0.001$) and 33.0% ($n = 31$) ($X^2 = 1.850$, $P = .174$) indicated a history of sinus headache.

Some notable findings with regard to sex were also observed, particularly among those student-athletes reporting a prior history of concussion. Among females with a prior history of concussion, 46.3% ($n = 25$) reported a history of migraine. This percentage of males having experienced prior

Table 2.—Participants With Concussion History Reporting Migraines Gender Differences

Variables	Total	No Migraines	Migraines	P Value
Gender ($n = 206$)				0.064
Female	54 (26.2%)	29 (53.7%)	25 (46.3%)	
Male	152 (73.8%)	103 (67.8%)	49 (32.2%)	

Table 3.—Participants With Concussion History Reporting Sinus Headaches Gender Differences

Variables	Total	No Sinus Headaches	Sinus Headaches	P Value
Gender (n = 206)				0.564
Female	54 (26.2%)	35 (64.8%)	19 (35.2%)	
Male	152 (73.8%)	105 (69.1%)	47 (30.9%)	

concussion and a concurrent history of migraine ($n = 49$) decreased to 32.2% ($X^2 = 3.421$, $P = .064$). These sex-specific observations were similarly noted in those subjects with a prior concussion history and the concurrent complaint of sinus headache, with 35.2% ($n = 19$) and 30.9% ($n = 47$) reported in females and males, respectively ($X^2 = 0.333$, $P = .564$) (Tables 2 and 3).

DISCUSSION

Background.—Benign headaches may be caused by physical activity incidental to the context of head trauma exposure; however, headache is also the most common symptom following head trauma and occurs in up to 93% of individuals after concussion.² The influence of preexisting headache in athletes suffering concussion, whether episodic or chronic, is still poorly understood. The complaint of baseline headache may be associated with an increased incidence and severity of other symptoms and few studies have closely examined the effects of headache (PTH or otherwise) on the quality of life in the athletic population.²⁶ Triggers for migraine include sleep deprivation, physical exertion, changes in environmental conditions, emotional stress, and dehydration. With the increased intensity of activity and greater frequency of head trauma, athletes are certainly exposed to significant headache triggers. The nonspecific nature of headaches also complicates RTP decision-making, as it is often unclear whether an athlete has an exacerbation of a primary headache disorder, new onset headache unrelated to trauma, or is still recovering from a concussion.²⁷ While returning an athlete to play in contact sports with an ongoing post-traumatic headache may be contraindicated based on risk of more severe injury and longer recovery

duration, there is no known risk to competing in athletics with a primary headache disorder.²⁸

Migraine.—Migraine is more common than many other chronic, incapacitating conditions,¹⁷ thus it should not be unexpected for sports medicine clinicians and other providers to encounter a significant number of athletes suffering from this primary headache disorder.¹² A primary finding in our study was that 23.7% of our sample reported history of migraine. A previous study in university students observed similar migraine prevalence.²⁰ Stewart et al also indicated the prevalence of migraine in older adolescents to be up to 23%,²⁹ although the lifetime prevalence in the general population is estimated to be 16%.¹²

Furthermore, the reported sinus headache prevalence of 25.2% also raises the question of an even higher prevalence of migraine or probable migraine in our sample. A 2007 landmark study by Eross and Dodick investigating subjects with a self-diagnosis of sinus headache found that 75% fulfilled International Headache Society criteria for migraine or probable migraine.¹¹ The most common reasons for an initial misdiagnosis included perceived headache triggers, pain location, and associated features commonly attributed to “sinus headache.”³⁰ If this indeed translates to our sample, the prevalence of those reporting migraine would certainly exceed 23.7%, even when accounting for those student athletes reporting multiple headache subtypes.

A number of possibilities could account for this finding, including better recognition of this headache type among patients and more accurate diagnoses being provided by physicians. Collegiate student-athletes are exposed to a variety of different triggers that are not experienced by other

college students, in turn predisposing them to migraine. These include the stress of balancing of academics and athletics, media attention, dehydration, physical exhaustion, sleep deprivation, travel, and others. Another plausible explanation could be due to the age group within which these student athletes fall. Migraines are known to affect a younger patient population relative to other disease states; however, for both sexes, migraine prevalence is not thought to peak until the ages of 25-55.⁹ Another possibility is the potential role of contact and/or collision sport exposure. Contact and/or collision sport athletes in our sample indeed were found to have a higher prevalence of migraine (27.8%) vs noncontact sports participants (22.9%), although this prevalence did not reach statistical significance in our study; however, further bolstering this argument is the potential role of concussion in propagating later headache development. Of those indicating a prior history of concussion, 35.9% reported a history of migraine and 32.0% self-reported sinus headache. In those reporting a history of multiple concussions, these numbers further increased to 40.4 and 33.0%, respectively; however, as this study was retrospective in nature, it is unclear if a predisposition to headaches existed prior to documented head injury.

Our study contained a number of other limitations. The self-report of headache type could potentially introduce inaccuracy due to misdiagnosis or either under- or over-reporting. Regarding the possibility that migraine may have been over-reported in our study, most literature on this subject indicates the under-reporting of migraine in similar study designs. The differentiation between primary and secondary headache types was not possible in our study due to an inability to verify self-reported diagnosis via independent medical evaluation. A further limitation included the inability to determine the number of lifetime migraine attacks. An individual who had one or two attacks would not officially meet ICHD-3 (beta) criteria for migraine, as five attacks are required to fulfill diagnostic criteria. Finally, despite the coexistence of certain headaches and prior history of concussion, the retrospective

nature of our study rendered it impossible to establish a temporal relationship between headache onset and time of head trauma. As mentioned above, it is unclear if headaches pre-dated concussion, were a direct result of concussion, or neither. Future prospective studies will help to further delineate such relationships.

The Influence of Migraine in Concussion and Post-Traumatic Headache.—In the context of concussion, studies have interestingly suggested that a preexisting history of migraine may be an independent risk factor for concussive injury. Gordon et al reported an association between migraine and concussion in a large population study, with an odds ratio of 2.36.³¹ In 2013, Schneider et al reported that male youth ice hockey players reporting headache at preseason baseline were at an increased risk of concussion during the season, after adjusting for age and concussion history.³² In a prospective cohort, Kuczynski et al reported a personal or family history of migraine in greater than 80% of those subjects experiencing symptomatic mild traumatic brain injury (mTBI).¹⁴ The reasons for this possible association remain unclear. Endogenous opioids such as beta-endorphins are known to increase following mTBI.³³ In migraineurs, beta-endorphins are also thought to be reduced at baseline.³⁴ It is conceivable, therefore, that these baseline biochemical changes predispose migraineurs to the syndrome of concussion following head trauma. Hypometabolism has also been noted in the orbitofrontal cortex of migraineurs vs controls. This, coupled with PET scan anomalies in a similar study of migraineurs, suggests that enhanced cortical excitability at baseline may predispose to an increased risk of concussion in those with migraine.^{35,36} Pain “remapping” has also been observed in migraineurs following injury to the trigeminal system. This response is seemingly specific to migraine relative to other primary headache disorders, suggesting the pathophysiology of migraine is impressionable and responds uniquely to external insults such as head trauma.³⁷

The underlying pathophysiology of PTH is largely unknown; however, the diagnosis occurs frequently in individuals with an inherent susceptibility to migraine.³⁸ Previous research, in fact,

supports a common molecular pathophysiological cause of migraine and mTBI.³⁹⁻⁴¹ Individuals with persistent post-traumatic headache have exhibited nociceptive dysfunction consistent with a generalized impairment of the spinothalamic system. Persistent PTH seems to result from a combination of injury to both central and peripheral structures, possible leading to central pain, and chronic sensitization of intra- and extracranial nociceptors.^{42,43} This sensory profile is compatible with the central sensitization seen in migraine sufferers. Furthermore, a similar association between chronic pain and dysfunctional pain modulation has been reported for various types of primary headache disorders, including migraine.⁴⁴ This suggests that the refractory clinical course noted in some persistent PTH is due to an inherent susceptibility for dysfunctional pain modulation, rather than the TBI itself. Specifically, impaired pain modulation may not necessarily be a prerequisite for the development of persistent PTH, but rather a risk factor; therefore, persistent PTH develops in some patients in whom the TBI-induced damage to nociceptive structures existing within an already sensitized nervous system.

Mild head trauma may also stimulate the trigeminal sensory afferents through cervicogenic mechanisms, similar to that seen in migraine.⁴⁵ This, in turn, results in the sequential activation of second and third order neurons in the nociceptive pathways of the brain stem, hypothalamus, and thalamus, leading to cortical spreading depression. The upper cervical sensory nerve roots that converge on the trigeminal nucleus caudalis contribute to the activation process, as inciting trauma results in forced flexion and extension of the cervical spine.⁴⁵ If cervical damage also occurred during the TBI, then nociceptive input from cervical segments arriving to the trigeminal nucleus may serve as a source of referred pain that contributes to mechanical hyperalgesia.⁴⁶

Other Etiologies.—Headache, like most postconcussive symptoms, can also occur due to various non-traumatic reasons. Dehydration in athletes can clinically resemble sports related mTBI, with symptoms mimicking those of a true concussive

injury.⁴⁷ Prescription medication use/abuse should also be considered in the differential of athletes with concussion-like symptoms in the absence of reported injury. The overuse of analgesics, triptans, or other acute headache compounds may contribute to frequent headache in some individuals independent of concussion, with population-based prevalence estimated to be 0.7-1.7%.⁴⁸ Excessive use of analgesics may also contribute to persistent PTH after concussion, with one study reporting up to 70.1% meeting criteria for probable medication overuse headache⁴⁹ rather than isolated PTH.

Summary.—Despite the significant occurrence of PTH following concussion, the isolated complaint of headache may often be discounted after head trauma. Even when investigated appropriately, the opportunity for human error remains, leading to the potential misdiagnosis of PTH for migraine or another primary headache disorder. Conversely, some athletes competing in contact sports are likely misdiagnosed with concussion when, in actuality, they are simply having a primary headache (e.g., migraine) exacerbation. Lastly, the isolated complaint of headache following trauma does not necessarily indicate a concussive injury has occurred. A recent large cohort analysis of children with minor head trauma found that headache was a common complaint despite having no other signs or symptoms suggestive of TBI.⁵⁰ In many instances, there is likely some underlying non-traumatic reason for the headache, but these reasons remain poorly described in the collegiate student-athlete population. In the setting of sport participation, clinical skill and experience of the evaluating clinician is paramount.

CONCLUSION

Chronic headaches can affect quality of life in a variety of ways. Much like the general population, the complaint of headache in sport is not unusual. Understanding the prevalence and conditions under which student-athletes suffer headaches is the first step in prevention and management. Despite their frequent occurrence in this specific population, and the presence of many coexisting triggers such as

travel and stress, there is little epidemiological data concerning sports-related headache. The increased prevalence of migraine seen in our sample is intriguing, as it remains unclear if this represents a genuine trend in a young adult sample routinely exposed to typical migraine triggers. Another more ominous possibility is a falsely elevated prevalence due to cumulative head trauma exposure in contact/collision sport athletes. Future prospective studies that do not rely solely on self-reported diagnosis will help clarify this provocative question. In the interim, helping to educate medical staff and athletes alike on the clinical phenomenon of migraine will help limit associated dysfunction and time away from sport participation. A comprehensive preseason evaluation that reviews personal headache history, family history of headache, and documentation of previous head trauma is recommended to aid medical staff in the diagnosis and management of future headache complaints.

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REFERENCES

1. Seifert T. Headache in sports. *Curr Pain Headache Rep.* 2014;18:448-454.
2. Meehan WP, d'Hemecourt P, Comstock RD. High school concussion in the 2008-2009 academic year: Mechanism, symptoms, and management. *Am J Sports Med.* 2010;38:2405-2409.
3. Williams SJ, Nukada H. Sport and exercise headache: Part 2. Diagnosis and classification. *Br J Sports Med.* 1994;28:96-100.
4. Headache Classification Subcommittee of the International Headache Society. The International Classification of Headache Disorders, 3rd ed. (beta version). *Cephalalgia.* 2013;33:629-808.
5. Pietrobon D, Striessnig J. Neurobiology of migraine. *Nat Rev Neurosci.* 2003;4:386-398.
6. Burch RC, Loder S, Loder E, Smitherman TA. The prevalence and burden of migraine and severe headache in the United States: Updated statistics from government health surveillance studies. *Headache J Head Face Pain.* 2015;55:21-34.
7. Stovner L, Hagen K, Jensen R, et al. The global burden of headache: A documentation of headache prevalence and disability worldwide. *Cephalalgia.* 2007;27:193-210.
8. MacGregor EA, Rosenberg JD, Kurth T. Sex-related differences in epidemiological and clinic-based headache studies. *Headache.* 2011;51:843-859.
9. Victor TW, Hu X, Campbell JC, Buse DC, Lipton RB. Migraine prevalence by age and sex in the United States: A life-span study. *Cephalalgia.* 2010;30:1065-1072.
10. Kinart CM, Cuppett MM, Berg K. Prevalence of migraines in NCAA division I male and female

- basketball players. National Collegiate Athletic Association. *Headache*. 2002;42:620-629.
11. Eross E, Dodick D, Eross M. The sinus, allergy, and migraine study (SAMS). *Headache*. 2007;47:213-224.
 12. Lipton RB, Stewart WF, Diamond S, et al. Prevalence and burden of migraine in the United States: Data from the American Migraine Study II. *Headache*. 2001;41:646-657.
 13. Sjaastad O, Bakketeig LS. Exertional headache. I. Vaga study of headache epidemiology. *Cephalalgia*. 2002;22:784-790.
 14. Kucziynski A, Crawford S, Bodell L, et al. Characteristics of PTMs in children following mild traumatic brain injury and their response to treatment: A prospective cohort. *Dev Med Child Neurol*. 2013;55:636-641.
 15. Haque B, Rahman KM, Hoque A, et al. Precipitating and relieving factors of migraine versus tension type headache. *BMC Neurol*. 2012;12:82.
 16. Lund N, Westergaard ML, Barloese M, Giamer C, Jensen RHJ. Epidemiology of concurrent headache and sleep problems in Denmark. *Cephalalgia*. 2014;0333102414543332.
 17. American Headache Society. Epidemiology and impact of headache and migraine. 2014. Available at: http://www.americanheadachesociety.org/assets/1/7/NAP_for_Web_-_Epidemiology___Impact_of_Headache___Migraine.pdf.
 18. Lipton RB, Bigal ME, Kolodner K, Stewart WF, Liberman JN, Steiner TJ. The family impact of migraine: Population-based studies in the USA and UK. *Cephalalgia*. 2003;23:429-440.
 19. Kelman L. The triggers or precipitants of the acute migraine attack. *Cephalalgia*. 2007;27:394-402.
 20. Bigal ME, Bigal JM, Betti M, Bordini CA, Speciali JG. Evaluation of the impact of migraine and episodic tension-type headache on the quality of life and performance of a university student population. *Headache J Head Face Pain*. 2001;41:710-719.
 21. Smitherman TA, McDermott MJ, Buchanan EM. Negative impact of episodic migraine on a university population: Quality of life, functional impairment, and comorbid psychiatric symptoms. *Headache J Head Face Pain*. 2011;51:581-589.
 22. Sallis RE, Jones K. Prevalence of headaches in football players. *Med Sci Sports Exerc*. 2000;32:1820-1824.
 23. Matheson GO, Anderson S, Robell K. Injuries and illnesses in the preparticipation evaluation data of 1693 college student-athletes. *Am J Sports Med*. 2015;43:1518-1525.
 24. Dick R, Agel J, Marshall SW. National Collegiate Athletic Association injury surveillance system commentaries: Introduction and methods. *J Athl Train*. 2007;42:173-182.
 25. National Institute of Neurological Disorders and Stroke. Common Data Elements. 2016. Available at: <https://commondataelements.ninds.nih.gov/Headache.aspx#tab=References>.
 26. Register-Mihalik MA, Guskiewicz KM, Douglas Mann J, et al. The effects of headache on clinical measures of neurocognitive function. *Clin J Sport Med*. 2007;17:282-288.
 27. Meehan WP. Medical therapies for concussion. *Clin Sports Med*. 2011;30:115-124.
 28. Asken BM, McCrea MA, Clugston JR, et al. "Playing through it": Delayed reporting and removal from athletic activity after concussion predicts prolonged recovery. *J Ath Train*. 2016;51:329-335.
 29. Stewart WF, Lipton RB, Celentano DD, et al. Prevalence of migraine headache in the United States. Relation to age, income, race, and other socioeconomic factors. *JAMA*. 1992;267:64-69.
 30. Levine H, Setzen M, Holy C. Why the confusion about sinus headache? *Otolaryngol Clin N Am*. 2014;47:169-74.
 31. Gordon KE, Dooley JM, Wood EP. Is migraine a risk factor for the development of concussion? *Br J Sports Med*. 2006;40:184-185.
 32. Schneider KJ, Meeuwisse WH, Kang J, et al. Pre-season reports of neck pain, dizziness, and headache as risk factors for concussion in male youth ice hockey players. *Clin J Sports Med*. 2013;23:267-272.
 33. McIntosh TK, Hayes RL, DeWitt DS, et al. Endogenous opioids may mediate secondary damage after experimental brain injury. *Am J Physiol*. 1987;253:E565-E574.
 34. Packard RC, Ham LP. Pathogenesis of posttraumatic headache and migraine: A common headache pathway? *Headache*. 1997;37:142-152.
 35. Fumal A, Laureys S, Di Clemente L, et al. Orbitofrontal cortex involvement in chronic analgesic-overuse headache evolving from episodic migraine. *Brain*. 2006;129:543-550.
 36. Aurora SK, Barrodale RN, Tipton RL, et al. Brainstem dysfunction in chronic migraine as evidenced

- by neurophysiological and positron emission tomography studies. *Headache*. 2007;47:996-1003.
37. Hussain A, Stiles MA, Oshinsky ML. Pain remapping in migraine: A novel characteristic following trigeminal nerve injury. *Headache*. 2010;50:669-671.
 38. Weiss HD, Stern BJ, Goldberg J. Posttraumatic migraine: Chronic migraine precipitated by minor head or neck trauma. *Headache*. 1991;31:451-456.
 39. Gilkey SJ, Ramadan NM, Aurora TK, et al. Cerebral blood flow in chronic posttraumatic headache. *Headache*. 1997;37:583-587.
 40. Lauritzen M. Pathophysiology of the migraine aura: The spreading depression theory. *Brain*. 1994;117:119-210.
 41. Lucas S. Headache management in concussion and mild traumatic brain injury. *PMR*. 2011;2:S406-S412.
 42. de Thommaso M, Federici A, Franco G, et al. Suggestion and pain in migraine: A study by laser evoked potentials. *CNS Neurol Disord Drug Targets*. 2012;11:110-126.
 43. Nahman-Averbuch H, Granovsky Y, Coghill RC, et al. Waning of "conditioned pain modulation": A novel expression of subtle pronociception in migraine. *Headache*. 2013;53:1104-1115.
 44. Defrin R, Riabinin M, Feingold Y, et al. Deficient pain modulatory systems in patients with mild traumatic brain and chronic PTM: Implications for its mechanism. *J Neurotrauma*. 2015;32:28-37.
 45. Piovesan EJ, Kowacs PA, Oshinsky ML. Convergence of cervical and trigeminal sensory afferents. *Curr Pain Headache Rep*. 2003;7:377-383.
 46. Defrin R. Chronic PTM: Clinical findings and possible mechanisms. *J Man Manip Ther*. 2014;22:36-44.
 47. Sabin MJ, Van Boxtel BA, Nohren MW, et al. Presence of headache does not influence sideline neurostatus or balance in high school football athletes. *Clin J Sport Med*. 2011;21:411-415.
 48. Evers S, Marziniak M. Clinical features, pathophysiology, and treatment of medication-overuse headache. *Lancet Neurol*. 2010;9:391-401.
 49. Heyer GL, Idris SA. Does analgesic overuse contribute to chronic PTMs in adolescent concussion patients? *Ped Neurol*. 2014;50:464-468.
 50. Dayan PS, Holmes JF, Hoyle J, et al. Headache in traumatic brain injuries from blunt head trauma. *Pediatrics*. 2015;135:504-512.

SUPPORTING INFORMATION

Additional Supporting Information may be found in the online version of this article.

Appendix A1: NCAA Headache Questionnaire