

# Association between Preseason/Regular Season Head Impact Exposure and Concussion Incidence in NCAA Football

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## ABSTRACT

STEMPEL, B. D., J. HAREZLAK, A. S. SHAH, S. ROWSON, J. P. MIHALIK, L. RIGGEN, S. DUMA, P. PASQUINA, S. P. BROGLIO, T. W. MCALLISTER, M. A. MCCREA, and CARE CONSORTIUM INVESTIGATORS. Association between Preseason/Regular Season Head Impact Exposure and Concussion Incidence in NCAA Football. *Med. Sci. Sports Exerc.*, Vol. 54, No. 6, pp. 912–922, 2022. **Purpose:** Contact sport athletes are exposed to a unique environment where they sustain repeated head impacts throughout the season and can sustain hundreds of head impacts over a few months. Accordingly, recent studies outlined the role that head impact exposure (HIE) has in concussion biomechanics and in the development of cognitive and brain-based changes. Those studies focused on time-bound effects by quantifying exposure leading up to the concussion, or cognitive changes after a season in which athletes had high HIE. However, HIE may have a more prolonged effect. This study identified associations between HIE and concussion incidence during different periods of the college football fall season. **Methods:** This study included 1120 athlete seasons from six National Collegiate Athletic Association Division I football programs across 5 yr. Athletes were instrumented with the Head Impact Telemetry System to record daily HIE. The analysis quantified associations of preseason/regular season/total season concussion incidence with HIE during those periods. **Results:** Strong associations were identified between HIE and concussion incidence during different periods of the season. Preseason HIE was associated with preseason and total season concussion incidence, and total season HIE was associated with total season concussion incidence. **Conclusions:** These findings demonstrate a prolonged effect of HIE on concussion risk, wherein elevated preseason HIE was associated with higher concussion risk both during the preseason and throughout the entire fall season. This investigation is the first to provide evidence supporting the hypothesis of a relationship between elevated HIE during the college football preseason and a sustained decreased tolerance for concussion throughout that season. **Key Words:** HEAD INJURIES/CONCUSSION, HEAD IMPACT EXPOSURE, BIOMECHANICS, FOOTBALL (AMERICAN)

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The scientific understanding of biomechanical mechanisms of sport-related concussion (SRC) has continued to improve with large-scale studies incorporating head impact measurement (HIM) tools in contact sport athletes. Using video analysis and head kinematics data collected from helmet and head-mounted impact sensors, researchers quantified accelerations and velocities of head impacts associated with concussion in athletes. Biomechanical risk functions based on the magnitudes of linear and rotational acceleration for concussive impacts were developed for youth (1,2) and adult (3,4) football athletes. However, head impact biomechanics and concussion mechanisms in contact sports differ from the traditional single-impact understanding of concussion in that participants are often exposed to repeated head impacts during contact

practices and games over the course of a season without diagnosed concussion. For example, a recent publication reported that National Collegiate Athletic Association (NCAA) Division I football athletes (459 athlete seasons) sustained a median of 426 impacts (75th percentile: 754 impacts) over the course of an entire fall football season (5). A primary outcome from that study was that total head impact exposure (HIE) during the preseason occurred at twice the proportion of the regular season (6), implicating the preseason as a time of elevated head impact burden. Broglio and colleagues (7) earlier reported an average of 652 head impacts per season across all high school football athletes enrolled in their study (156 athlete seasons). Whereas the majority of football head impacts result in lower magnitude head accelerations, athletes still routinely sustain head impacts greater than 50g that do not lead to diagnosed concussion (8). Accordingly, research has begun to focus on the possible role that subconcussive head impacts may play in concussion risk and mechanism. A recent publication from the NCAA–Department of Defense Concussion Assessment, Research and Education (CARE) Consortium identified that head impacts occurring at the time of incident concussion (i.e., concussive impacts) were often among the highest magnitude head impacts that injured athletes sustained that season, although, in many cases, concussive impact magnitudes were not remarkable when compared with the distribution of head impacts sustained across the entire sample of athletes without diagnosed concussion (9). This finding makes the case for individualized concussion tolerance levels that may be influenced by, among other things, HIE, which may reduce concussion tolerance for athletes.

A more direct association between HIE and concussive injury (i.e., incident concussion) was revealed in a number of studies, although the precise biological mechanism has not yet been characterized. Beckwith and colleagues (10) reported differences in the magnitude of concussive impacts and HIE between athletes with immediate or delayed concussion diagnosis. They reported that athletes with immediate diagnosis had significantly higher peak head kinematic measures associated with the concussive impact, whereas athletes with delayed diagnosis sustained a significantly higher number of head impacts leading up to injury. Broglio and colleagues identified that concussed athletes had significantly higher head impact density (sum of impact severities divided by the time between impacts) than uninjured controls who were matched by the severity of concussive impact for the concussed group or the final impact for the control group, number of impacts in the 24 h leading up to the final impact, and athlete position and team (11). These types of findings, as well as other findings of cumulative magnetic resonance imaging and cognitive changes in nonconcussed athletes with high HIE (12–14), led Talavage and colleagues (15) and others (8) to propose two separate mechanisms for concussion consisting of either a singularly traumatic event or a mechanism by which athletes accumulate symptom-inducing injury more gradually through HIE.

Researchers have begun to focus on quantifying HIE over the course of a season in an attempt to characterize injury risk

functions associated with the accumulation of HIE. A consistent method for quantifying individual athlete HIE has not yet been defined, and studies used a variety of different metrics. A common method for quantifying HIE involves simply counting the number of recorded head impacts over a period of interest (day, week, season). However, the concussive mechanism remains a biomechanical event. As such, higher magnitude impacts should be associated with greater injury risk. Incorporation of head impact magnitudes in the calculation of HIE may be an important addition to improve the predictive ability of HIE metrics. For example, Urban and colleagues (16) developed cumulative injury risk functions to account for the number and magnitude of head impacts sustained over a given time period. Those risk-weighted exposure (RWE) functions sum individual concussion risks (4), based on the magnitudes of each impact over the time period of interest. Using both RWE and the cumulative number of head impacts from the start of the season until injury, significantly higher HIE was recently identified in a cohort of 50 concussed NCAA Division I football athletes compared with uninjured controls matched by team and position (8). This finding revealed that concussed athletes had higher HIE than uninjured controls who were exposed to essentially the same game and practice activities. This may highlight differences in playing style that may predispose an athlete to higher HIE and a correspondingly higher rate of accumulating impact burden that may lead to eventual injury. Playing style differences and resulting differences in HIE are likely associated with individual characteristics such as playing position, aggressiveness, and other psychosocial attributes that will be consistent throughout the season and playing career.

However, the process of predicting accumulating injury is likely more complex for concussion than simply adding up the number of head impacts or calculating RWE, because of secondary injury processes and healing at the level of the cerebral tissue that results in a time-varying risk function. Using an animal model, Prins and colleagues (17) identified that the duration of increased vulnerability to second injury after a single traumatic brain injury was related to the duration of metabolic depression. Similarly, researchers identified prolonged neuroinflammation that can persist for months to years after moderate and severe traumatic brain injury (18,19). Those mechanisms may also be present, albeit to a lesser degree, for head impacts that do not result in diagnosed concussion, with accumulating brain tissue load associated with the number, frequency, and severity of cumulative HIE. Therefore, elevated HIE may have a more prolonged effect on concussion risk than simply an accumulating brain tissue loading process that eventually exceeds a cumulative threshold and results in concussion report for the athlete. For example, within the bounds of an athletic season, elevated HIE early in the season may contribute to elevated season-long risk of concussion. However, the time course of changing tolerance has not been defined, and it remains unknown whether this is a short-term (i.e., resolving within days to weeks) or long-term (i.e., season-long) phenomenon. Therefore, this study was conducted to determine if an association exists between levels of preseason and regular

season HIE with concussion incidence in NCAA Division I football athletes, with the hypothesis that higher levels of pre-season HIE may be related to elevated concussion risk beyond the preseason and throughout the entire fall season. A secondary hypothesis was that the level of HIE was based on athlete-related characteristics, and as such, individual preseason HIE would be associated with season-long HIE.

## METHODS

This analysis quantified the association between HIE and concussion incidence at different times during the college football season. Real-time HIE data were collected according to the HIM protocol in a six-team subset of the NCAA–Department of Defense Grand Alliance CARE Consortium, called the Advanced Research Core (ARC). Concussion incidence was monitored in the same subset of athletes. The protocols for HIM and concussion incidence tracking were approved by the institutional review board at the Medical College of Wisconsin and the US Army Human Research Protection Office, with the local sites using a reliance agreement with the Medical College of Wisconsin institutional review board. All subjects provided written and informed consent before participation in the research study.

**Concussion incidence.** This study consented and enrolled varsity college football athletes from six NCAA Division I programs across the United States during the 2015 through 2019 fall football seasons. Detailed methods for the CARE Consortium were previously reported (20). Concussions were identified and diagnosed by team medical staff, according to a standardized protocol outlined by the CARE Consortium, that defined concussion using the consensus definition from the US Department of Defense evidence-based guidelines initiative, which closely parallels the American Academy of Neurology definition (21). Concussed athletes were entered into the CARE research protocol where local study team members recorded detailed information on the injury date and time, type of activity (practice/game), and type of play. Personal identifying information was removed from the data by assigning a study specific number to each athlete.

Incidence and timing of concussion were categorized as preseason (before the first game) or regular season (up to the conference championship week). Total season concussions accounted for all concussions that occurred in preseason and the regular season. Concussions that occurred during spring practices or spring competition were not included in this analysis. Concussion incidence was the number of concussions during a specific time period (preseason, regular season, total season) divided by the total number of consented and enrolled athletes during that period.

**HIM in the ARC.** HIE was monitored for the same six NCAA Division I teams that were used to quantify concussion incidence. The HIM protocol consisted of recording all head impacts greater than 10 g in instrumented athletes during all practices, scrimmages, and games of the 2015 through 2019 fall seasons using the Head Impact Telemetry (HIT) System (Riddell SRS; Riddell, Rosemont, IL). The HIT System

measures head linear accelerations using six accelerometers inside the football helmet and computes peak component and resultant linear and rotational accelerations. HIT System encoders were included in Riddell Speed and SpeedFlex helmets. Data acquisition triggered any time a single accelerometer exceeded a 9.6-g threshold, and only head impacts with greater than 10-g resultant linear acceleration were included in this analysis. Previous research indicated that impacts with peak resultant linear acceleration less than 10g can be associated with nonimpact dynamic movements of the athlete (22). Head impact data were wirelessly transmitted to a laptop computer on the sidelines and later uploaded to the Riddell cloud for storage. The study team was provided access to the HIT System data in the cloud or by direct transfer from the site via a secure ftp server. Personal identifying information was removed from the data by assigning a study-specific number to each athlete that was used for all study-related data.

Data collection was managed by HIT System operators at each institution. The operators placed battery-charged sensors in each helmet before every practice, scrimmage, and game. Operators charged the sensor batteries once per week and offloaded any head impacts that had not yet been downloaded. The fall football season initiated with August preseason practices as outlined in the NCAA Division I Manual and continued through the end of the regular season for each team. Video verification of head impacts was not possible because of the volume of impacts recorded across all enrolled athletes. However, quality control procedures consisted of confirming that HIT System data were consistent with practice and game dates/times outlined on activity logs maintained by local study coordinators, checking data outputs for missing values, and confirming consent and study participation for each athlete.

**Head impact exposure.** Cumulative HIE was computed as the total number of recorded head impacts and cumulative RWE (RWE-CP; equation 1) (16) for each enrolled athlete during the preseason, regular season, and total season. Total season exposure was the sum of preseason and regular season exposure. Median athlete exposure was defined as the 50th percentile in number of head impacts and RWE for the preseason, regular season, and total season among participating athletes for each team during each of the five seasons included in this study. High exposure was the 90th percentile of head impacts and RWE for each time period among participating athletes for each team during each of the five seasons. Median and 90th percentile values for linear and rotational acceleration were also computed for the preseason, regular season, and total season for each of the five seasons included in this study.

$$RWE = \sum \frac{1}{1 + e^{-(\beta_0 + \beta_1 A + \beta_2 \alpha + \beta_3 A\alpha)}} \quad [1]$$

**Association of HIE and concussion incidence.** The association between HIE and concussion incidence was analyzed across the 5 yr of the study. Exposure sustained by all enrolled football athletes at each Institution during each season was treated as a separate observation in our analyses. For this

analysis, concussion incidence was only analyzed for athletes in the HIM protocol. Four of the six teams collected HIM data for the 2016 through 2019 seasons, and the remaining two teams collected HIM data for the 2015 through 2019 seasons. Accordingly, each association between HIE and concussion incidence was based on 26 team-season observations. Investigated associations included the following: preseason HIE and preseason/regular season/total season concussion incidence, regular season HIE and regular season concussion incidence, and total season HIE and total season concussion incidence. Concussion incidence, defined as the number of sustained concussions by HIT System–equipped athletes divided by the number of participating athletes, was modeled using logistic regression (equations 2–3) where the probability of a concussion during the respective time period (preseason/season/total) was modeled as a function of the extracted features of the HIT System (R Statistical Software, R Foundation for Statistical Computing). Those factors included the number of recorded head impacts (median and 90th percentile) and RWE (median and 90th percentile) per team per season. A total of 20 associations (4 HIE metrics  $\times$  5 season period-based associations) were analyzed. Associations between the three season periods (preseason, regular season, total season) were temporally limited under the assumption that concussion incidence (outcome) is causally related to HIE (input). Therefore, HIE was only associated with concussion incidence in season periods during or subsequent to the periods in which HIE was measured. For example, preseason concussion incidence would not likely be influenced by regular season HIE, and therefore, that association was not included in this analysis. Accordingly, a total of 20 associations (4 HIE metrics  $\times$  5 season period-based associations) were analyzed, where five season period-based analyses are as follows: three analyses where preseason exposure is associated with preseason, season, and total concussion incidence; one analysis where regular season exposure is associated with regular season concussion incidence; and one analysis where total exposure is associated with total concussion incidence. We report the odds ratios (OR) derived from the logistic regression analysis together with their 95% confidence intervals and *P* values.

$$\text{logit}(p) = \beta_1 + \beta_2 * (\text{Variable of Interest}) \quad [2]$$

where,

$$\text{logit}(p) = \frac{p}{1-p} \text{ and } p = \text{Probability of Concussion} \quad [3]$$

A secondary analysis was conducted to determine whether preseason HIE was correlated to regular season HIE. This analysis quantified the strength of correlations using Pearson's coefficient between preseason and regular season HIE for individual athletes enrolled in the HIM protocol on the six ARC teams. Each season was treated as a separate observation for athletes who participated in multiple seasons. The analysis included a total of 786 athlete seasons for which athletes participated in both preseason and regular season contact activities. An athlete season was counted any time a unique athlete participated in at

least one contact activity and sustained one or more head impacts. Two separate analyses were conducted to characterize the correlation between the number of preseason/regular season impacts and the total preseason/regular season RWE.

## RESULTS

HIM data were collected for an average of  $10.9 \pm 3.0$  (mean  $\pm$  SD) regular season games per fall season for each of the six participating teams across the 5 yr of the study. The six teams had an average of  $21.7 \pm 3.6$  preseason contact practices per season, with two-a-day practices counting as two separate practices. Participating teams also had an average of  $36.4 \pm 10.9$  regular season contact practices.

Total season enrollment of athletes instrumented with the HIT System in the HIM protocol varied between 82 athletes (2015) and 290 athletes (2017) for a total of 1120 athlete-seasons. However, only two teams participated in 2015. Therefore, the minimum number of enrolled athletes in a six-team year was 210 athletes in 2019. HIM enrollment accounted for between 22% (2019) and 37% (2016) of all enrolled football athletes for those six programs. Athletes in the HIM protocol sustained a total of 70 concussions during the fall season over the 5 yr of the study. Total concussion rate across all years of the study was 6.3%. The highest annual season concussion rate occurred in 2015 (8.5%), followed by the lowest total season concussion rate in 2016 (5.8%), and a relatively stable annual rate from 2017 to 2019 (6.1%–6.2%). Concussion rate was higher during the regular season (avg. 3.5%) than the preseason (avg. 3.1%) across the 5 yr of the study. However, more preseason than regular season concussions occurred in 2019, although this may be explained by the decreased number of regular season concussions in 2019 ( $n = 5$ ) compared with other years.

**Head impact exposure.** HIE was quantified using both the cumulative number of head impacts and RWE for the preseason, regular season, and total season. Median and 90th percentile values across all participating athletes in the HIM protocol were analyzed to characterize typical athlete exposures as well as HIE in high-exposure athletes. Median HIE ranged from 336 (2017) to 434 (2018) head impacts over the course of the entire season across the 5 yr of this study (Table 1). High-exposure athletes (90th percentile) sustained between 958 (2015) and 1190 (2017) head impacts over the course of the entire season across the 5 yr of the study. Athletes sustained an average of  $39.6\% \pm 23.8\%$  (mean  $\pm$  SD) of their season-long head impacts during the preseason across all athletes and years of the study.

**Association of HIE with concussion incidence.** This analysis identified some remarkable associations between HIE and concussion incidence (number and/or rate). Total season HIE was associated with total season concussion incidence. For example, three metrics for HIE across the entire fall season (preseason + regular season) had strong positive associations with concussion incidence (Fig. 1). Those associations indicated that fall seasons with greater total HIE had greater concussion incidence. The strongest of these associations was for 90th percentile total season RWE and number of total season concussions

TABLE 1. Annual enrollment and number of concussions across the six NCAA Division I teams participating in the HIM Core of the NCAA–DoD Grand Alliance and the CARE Consortium.

|                                         | 2015      | 2016        | 2017        | 2018        | 2019        |
|-----------------------------------------|-----------|-------------|-------------|-------------|-------------|
| Enrolled athletes: PS/RS/TS             | 82/71/82  | 253/255/274 | 277/265/290 | 264/250/264 | 209/188/210 |
| No. teams                               | 2         | 6           | 6           | 6           | 6           |
| Concussions: no. (incidence)            |           |             |             |             |             |
| Total                                   | 7 (8.5%)  | 16 (5.8%)   | 18 (6.2%)   | 16 (6.1%)   | 13 (6.2%)   |
| Preseason                               | 6 (7.3%)  | 7 (2.8%)    | 8 (2.9%)    | 5 (1.9%)    | 8 (3.8%)    |
| Regular season                          | 1 (1.4%)  | 9 (3.5%)    | 10 (3.8%)   | 11 (4.4%)   | 5 (2.7%)    |
| HIE, median:90th percentile             |           |             |             |             |             |
| Season impacts                          | 422:958   | 414:1117    | 336:1190    | 434:1102    | 351:973     |
| Season RWE                              | 1.01:2.49 | 0.91:3.27   | 0.79:4.25   | 0.95:3.67   | 0.50:2.72   |
| PS impacts                              | 144:355   | 122:327     | 149:441     | 158:446     | 137:327     |
| PS lin. accel. ( <i>g</i> )             | 21:50     | 20:47       | 21:47       | 20:45       | 20:44       |
| PS rotat. accel. (rad·s <sup>-2</sup> ) | 950:2025  | 957:1993    | 975:2026    | 971:2007    | 960:1967    |
| PS RWE                                  | 0.31:1.32 | 0.17:1.00   | 0.21:1.64   | 0.22:1.57   | 0.11:1.12   |
| RS impacts                              | 319:685   | 309:793     | 224:819     | 281:733     | 238:691     |
| RS lin. accel. ( <i>g</i> )             | 20:47     | 21:48       | 21:48       | 21:47       | 20:45       |
| RS rotat. accel. (rad·s <sup>-2</sup> ) | 922:1986  | 968:2075    | 974:2110    | 970:2097    | 954:2026    |
| RS RWE                                  | 0.63:1.92 | 0.71:2.52   | 0.47:3.21   | 0.57:2.36   | 0.33:1.79   |

Concussion incidence is reported as the percent of all enrolled athletes during that season. Head impact data are represented as the 50th and 90th percentiles (50th percentile: 90th percentile). DoD, Department of Defense; lin. accel., linear acceleration; PS, preseason; rotat. accel., rotational acceleration; RS, regular season; TS, total season.

(OR, 1.288;  $P = 0.005$ ). However, median total season impacts (OR, 1.002;  $P = 0.031$ ) and median total season RWE (OR, 1.793;  $P = 0.028$ ) were also associated with the number of total season concussions.

Similarly, preseason HIE was associated with preseason concussion incidence. Once again, higher preseason HIE was associated with higher preseason concussion incidence. The strongest of these associations was for median preseason RWE

and number of preseason concussions (OR, 19.55;  $P = 0.008$ ; Fig. 2). However, median preseason impacts (OR, 1.006;  $P = 0.076$ ) and 90th percentile preseason RWE (OR, 1.631;  $P = 0.051$ ) were also marginally associated with the number of preseason concussions.

Perhaps most interestingly, preseason HIE had strong positive associations with total season concussion incidence. Team seasons with higher preseason HIE also had higher total

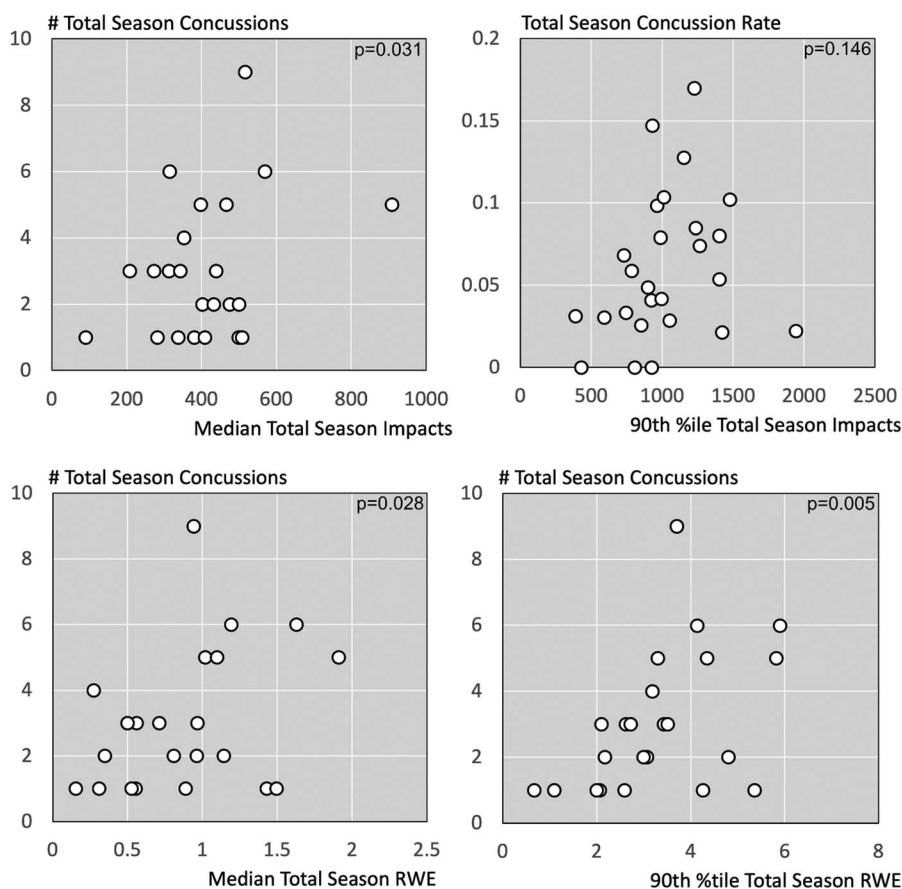


FIGURE 1—Association between different measures of total season HIE and total season concussion incidence.

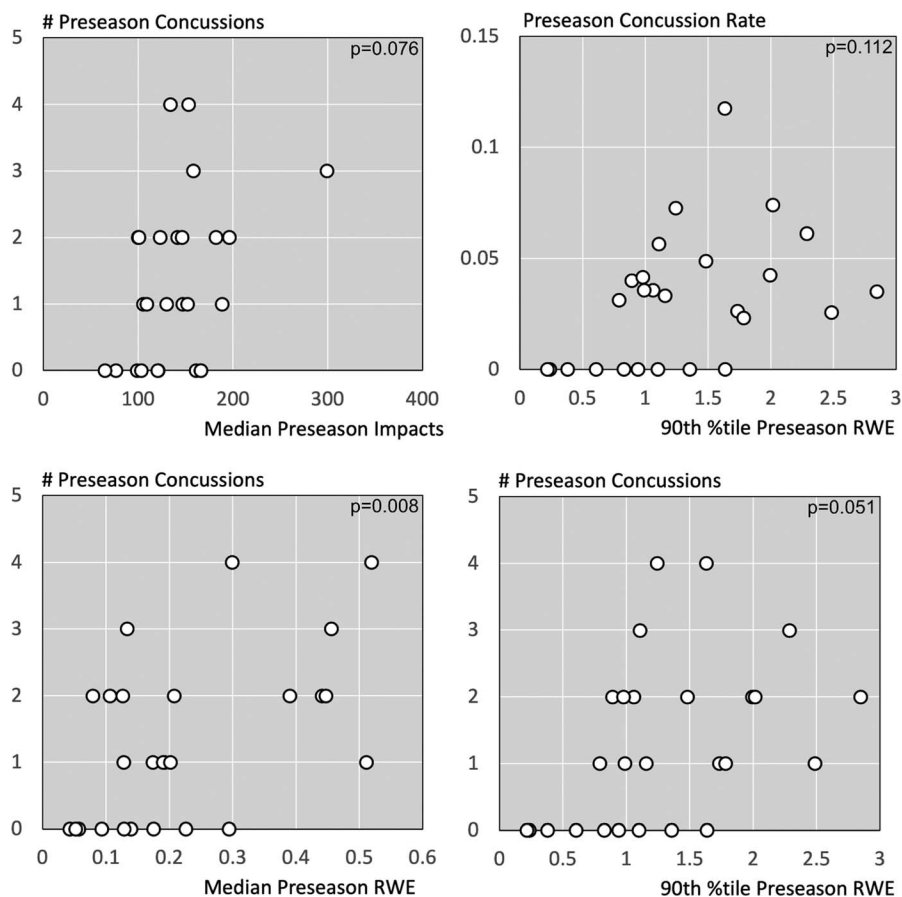


FIGURE 2—Association between different measures of preseason HIE and preseason concussion incidence.

season concussion incidence. This may imply that elevated HIE during the college football preseason has a prolonged effect of reducing concussion tolerance beyond the preseason and throughout the entire fall football season. The strongest of these associations was for median preseason RWE and season-long concussion rate (OR, 7.124;  $P = 0.015$ ; Fig. 3). However, 90th percentile preseason RWE (OR, 1.533;  $P = 0.016$ ), as well as median (OR, 1.005;  $P = 0.051$ ) and 90th percentile (OR, 1.002;  $P = 0.035$ ) number of preseason impacts had associations with total season concussion incidence.

However, not all HIE metrics were associated with concussion incidence (Table 2). For example, preseason HIE metrics had very limited associations with regular season concussion incidence ( $P > 0.20$ ). Likewise, regular season HIE metrics had limited associations with regular season concussion incidence ( $P > 0.12$ ).

#### Correlation of preseason and regular season HIE.

Pearson's correlation coefficient was used to characterize the strength of correlations between preseason and regular season HIE for individual athletes. Preseason HIE was significantly correlated to regular season HIE when analyzed on an athlete-by-athlete basis ( $P < 0.0001$ ; Fig. 4), although the strength of the correlations according to the Pearson's correlation coefficient was somewhat limited, particularly for RWE ( $R = 0.48$ ).

Correlation of preseason to regular season HIE in individual athletes was stronger when analyzed separately for each year, and the ratio of preseason to regular season HIE changed

between years. This indicates that preseason and/or regular season HIE varied across the entire sample from year to year. Average Pearson's correlation coefficient for the relationship between the number of recorded preseason/regular season head impacts was  $0.86 \pm 0.02$  (mean  $\pm$  SD). The slope of the correlation varied from a minimum of 1.57 in 2018 to a maximum of 2.03 in 2016. This indicated that athletes recorded a maximum of 2.03 times the number of head impacts during the regular season than they did during the preseason in 2016. The correlation between preseason and regular season HIE was weaker for RWE, wherein the average Pearson's coefficient was  $0.64 \pm 0.09$ . Interestingly, the slope of that correlation was closer to 1.0, with a minimum of 0.95 in 2019 and a maximum of 1.66 in 2016. A slope of 0.95 in 2019 indicates that athletes included in this study actually sustained greater total RWE during the preseason than the regular season, and slope values of 1.19 and 1.16 in 2017 and 2018 indicate that athletes had less than 20% greater HIE during the regular season.

## DISCUSSION

Elevated HIE in the absence of a single high magnitude impact has been linked to concussive injuries (8,11,15,23). Those studies implied cumulative brain tissue load with repeated head impacts sustained during contact sport participation. Cumulative brain tissue load may reduce the tolerance for concussion

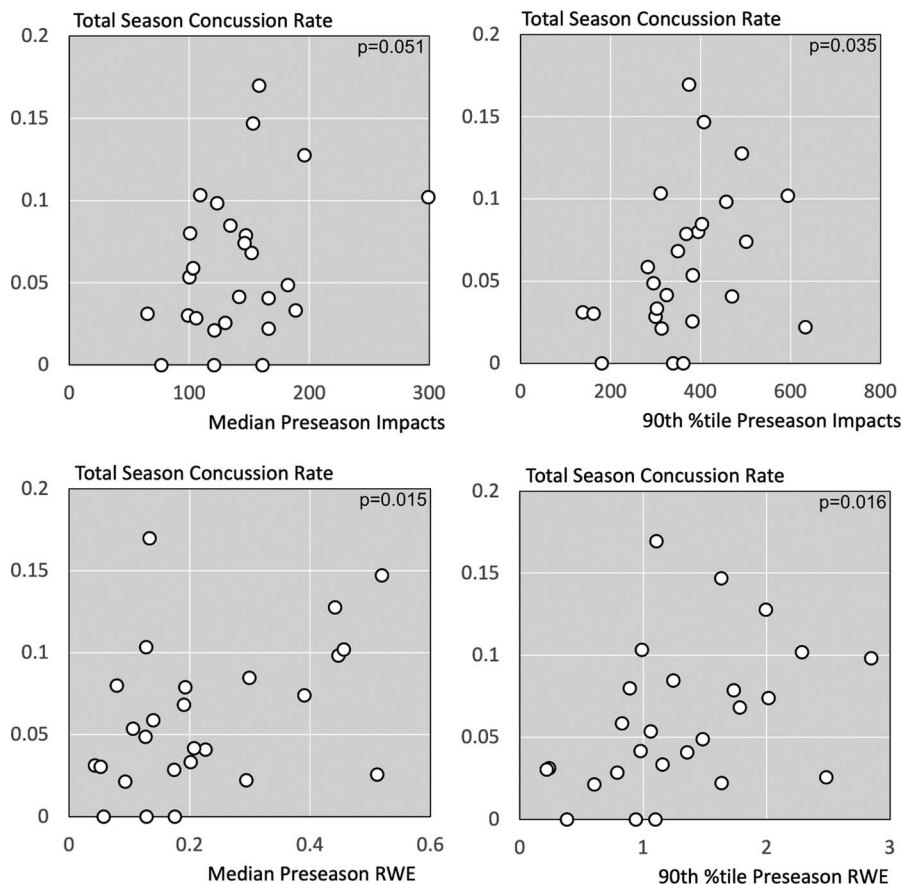


FIGURE 3—Association between different measures of preseason HIE and total season concussion incidence.

associated with any single head impact and contribute to differing injury tolerance between individual athletes (9). However, the time course of elevated concussion risk associated with HIE was not well defined. For example, it remains unknown whether high levels of preseason HIE increase concussion risk only during the preseason or have a sustained effect of elevated concussion risk throughout the entire season. This study identified strong associations between HIE and concussion incidence both within the period of interest (i.e., preseason) and, more importantly, beyond that period. For example,

levels of HIE during the college football preseason were associated with season-long concussion incidence. This investigation is the first to provide evidence supporting the hypothesis of a relationship between elevated HIE during the college football preseason and a prolonged decreased tolerance for concussion throughout that season, even though the rate of HIE (impacts per week) considerably decreases during the regular season (5). It also would tend to defuse the argument that higher exposure athletes simply have more head impact opportunities for concussion without any cumulative changes associated with

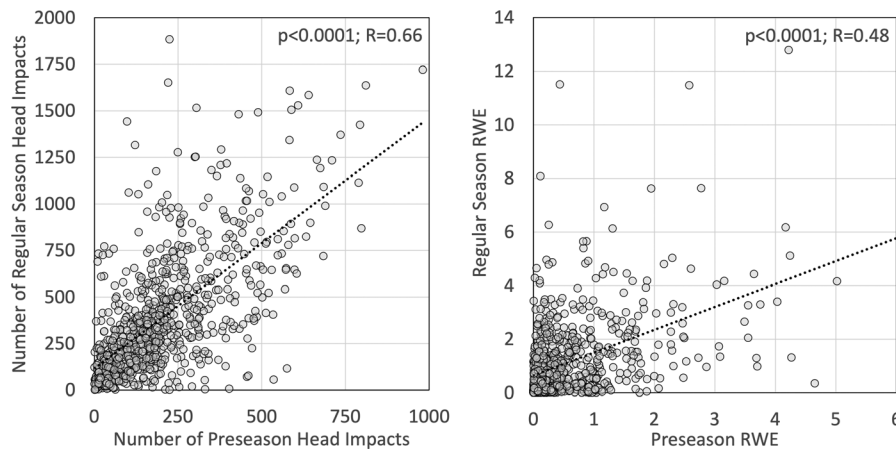
TABLE 2. OR with their respective 95% confidence intervals for each unit change in the predictor variables and *P* values of associations between HIE and concussion incidence (presented as OR (95% CI) and *P* value).

|              | Preseason Rate                      | Regular Season Rate          | Total Season Rate                   |
|--------------|-------------------------------------|------------------------------|-------------------------------------|
| Preseason    |                                     |                              |                                     |
| 50th Imp.    | 1.006 (0.999 to 1.011) 0.076        | 1.003 (0.995 to 1.009) 0.449 | 1.005 (1.000 to 1.009) 0.051        |
| 90th Imp.    | 1.002 (0.999 to 1.005) 0.112        | 1.002 (0.999 to 1.005) 0.236 | <b>1.002 (1.000 to 1.004) 0.035</b> |
| 50th RWE     | <b>19.55 (2.137 to 177.4) 0.008</b> | 2.21 (0.213 to 20.08) 0.491  | <b>7.124 (1.441 to 34.17) 0.015</b> |
| 90th RWE     | 1.631 (0.988 to 2.655) 0.051        | 1.367 (0.837 to 2.189) 0.201 | <b>1.533 (1.078 to 2.165) 0.016</b> |
| Reg season   |                                     |                              |                                     |
| 50th Imp.    |                                     | 1.002 (0.999 to 1.005) 0.231 |                                     |
| 90th Imp.    |                                     | 1.001 (0.999 to 1.002) 0.357 |                                     |
| 50th RWE     |                                     | 1.807 (0.705 to 4.368) 0.202 |                                     |
| 90th RWE     |                                     | 1.321 (0.930 to 1.866) 0.115 |                                     |
| Total season |                                     |                              |                                     |
| 50th Imp.    |                                     |                              | <b>1.002 (1.000 to 1.003) 0.031</b> |
| 90th Imp.    |                                     |                              | 1.001 (1.000 to 1.001) 0.146        |
| 50th RWE     |                                     |                              | <b>1.793 (1.059 to 3.012) 0.028</b> |
| 90th RWE     |                                     |                              | <b>1.288 (1.077 to 1.540) 0.005</b> |

Statistically significant associations ( $P < 0.05$ ) are in bold format.

*P*-values less than 0.1 and greater than 0.05 are italicized.

CI, confidence interval; Imp., impact; Reg., regular.



**FIGURE 4—Correlation of preseason and regular season HIE.**

subconcussive impacts. These findings may have significant implication in understanding the confluence between repetitive subconcussive impacts and concussion, although the time course of elevated concussion risk associated with HIE has yet to be characterized.

An explanation for prolonged elevation of injury risk associated with HIE lies in the cascade of secondary injury after the primary insult (i.e., head impact). Studies focused on traumatic brain injuries identified that a number of secondary injury processes initiate after the initial injury that can include neuroinflammation, cerebral blood flow dysfunction, and breakdown of the blood–brain barrier (24). In concussed individuals, those changes can last for days to weeks and outlast the symptomatic period (25). That period has become known as the window of cerebral vulnerability after SRC (26). Identification of risks associated with the window of cerebral vulnerability led to more conservative clinical management of SRC (27). However, for repeated subconcussive impacts, the degree to which these secondary processes contribute to accumulating brain changes, the duration of this effect, and the influence of number, frequency, and severity of subconcussive head impacts remains unknown. Preclinical studies identified evidence of neuroinflammation in rodents after “mild” injuries without significant behavioral deficits (28). Similar findings were identified in humans acutely after SRC (29). These findings may imply a neuroinflammatory response in the brain after “subconcussive” head impacts that do not produce outward evidence of concussion such as somatic symptoms, anxiety/depressive behaviors, spatial cognitive deficits, and sensorimotor deficits. This type of finding was also identified in human studies of contact sport athletes, wherein functional impairment and changes in diffusion tensor imaging metrics of white matter diffusion were identified in athletes who had participated in a season of contact sports but were not diagnosed with concussion (12–14,30). In some cases, white matter changes identified using diffusion tensor imaging persisted for up to 6 months after the season (12). Those findings may indicate an extended period of vulnerability associated with higher levels of repetitive HIE that may last beyond the acute phase (days/weeks) and persist to some degree throughout the entire football season. This extended period of vulnerability would

validate present results that indicated an association between preseason HIE and total season concussion incidence, wherein elevated HIE during the preseason resulted in higher concussion incidence throughout the entire season.

The present analysis focused on within-season effects of HIE by quantifying HIE and concussion risk during only the fall football season and excluding spring football practices and scrimmages. Deleterious effects of high levels of HIE during a single fall season have been highlighted in studies focused on cognitive and magnetic resonance imaging changes in football athletes without diagnosed concussion. The decision not to expand the current analysis to include spring activities was based on the months-long period between the end of the fall season and spring football activities in which athletes do not experience regular football contact activities. It was our assertion that this noncontact time would provide a period of recovery during which decreased injury tolerance associated with daily head impact activities would eventually return toward baseline. However, a recent study hypothesized a correlation between lifelong HIE and chronic cognitive and emotional changes (31). Although that study relied on estimates of HIE, the association between HIE and more chronic injury risk should be a focus of continued analyses. For example, a more in-depth characterization of carryover effects from one season of elevated HIE into spring practices and the following season may be warranted, although to date only repetitive concussion has been linked to chronic neuropsychological abnormalities (32).

This study also identified a significant correlation between preseason and regular season HIE for individual athletes (Fig. 4). The strength of this correlation was likely attributable to factors that may influence the frequency of HIE including status as a starter or reserve player, primary playing position, and individual playing style that will generally be consistent from the preseason through the regular season. The analysis also demonstrated that the preseason/regular season correlation changed from year-to-year, which was likely influenced by changes to the NCAA Division I preseason schedule that affected preseason HIE (5,33). These novel and important findings indicate that higher levels of HIE during the preseason may contribute to prolonged elevation in concussion risk that

results in higher levels of concussion incidence throughout the fall season and not just during the preseason.

As with prior studies, this analysis demonstrated a high degree of individual variability in HIE (Fig. 4). Variability in HIE for football athletes has been attributed to a number of factors including primary playing position and team (33,34). However, Campolettano and colleagues reported that 48% of variability in practice HIE was attributable to individual differences, even after accounting for other factors including primary playing position, team, and athlete ability (35). Given the proposed correlation between HIE and concussion risk, highlighted in the results of this analysis and others, significant variability in HIE may contribute to high-exposure athletes experiencing a more pronounced effect on individual concussion risk and tolerance. This may contribute to the significant variability in concussive impact biomechanics that has been highlighted in previous studies (8,23,36). Rowson and colleagues (9) hypothesized that differences in individual tolerance may explain the variability in concussive impact magnitudes by showing that a majority of concussive impacts were the highest or near the highest magnitude impacts sustained by the concussed athlete during that season. This may indicate differing tolerance between athletes, as the magnitude of the concussive impacts was often not remarkable when compared with the entire distribution of head impacts sustained across all athletes enrolled in the study. Present findings add weight to the role of HIE in altered concussion tolerance between individuals as high levels of HIE during the season may result in an athlete reporting concussion from a head impact that may otherwise not result in clinically relevant outcomes or concussion report.

Identification of injury tolerance is one of the pillars of injury biomechanics research and significant effort has been devoted to characterizing tolerance for different body regions. Injury tolerance refers to the body's capacity to resist specific loads before sustaining injury and is often characterized using injury risk curves that relate increasing biomechanical loads to greater injury risk. For example, studies have proposed injury tolerance relationships for concussion (4), lumbar spine fracture (37), cervical spine fracture (38), blunt thoracic injury (39), and a variety of other body components. However, characterization of concussion tolerance is more complicated than many other injuries for various reasons that include significant variability in the severity and type of concussive symptoms (40) and varying athlete attitudes toward concussion reporting (41–43) that may contribute to underreporting or overreporting of concussions. However, in addition to variability in diagnosis, concussion tolerance is also complicated from a mechanistic standpoint. As discussed previously, research has now begun to recognize two mechanisms for concussion consisting of single high magnitude impacts and repetitive subconcussive impacts. Therefore, the biomechanical relationship might be expressed as the magnitude/direction of the concussive impact or the number, frequency, and magnitude of subconcussive impacts leading up to incident concussion. Our research suggests that repetitive subconcussive impacts reduce the tolerance for single high magnitude impacts, resulting in incident

concussion from an otherwise noninjurious head impact. All of these factors deserve continued attention at the clinical and preclinical levels. Results of this study can add clarity with regard to the sustained effects of repetitive subconcussive head impacts on injury risk throughout the season.

A limitation of the current analysis was associated with possible inaccuracies of the HIT System. Several laboratory-based studies focused on quantifying the level of accuracy for the HIT System when compared with laboratory instrumentation. In general, those studies highlighted the ability of the system to detect helmet/head impacts. For example, Siegmund and colleagues (44) reported that the HIT System identified 96.1% of 896 laboratory head impacts to different areas of the helmet. This provides important support for the current study because the number of recorded head impacts during different periods of the season was a primary outcome for this analysis. However, some laboratory studies reported a lower level of accuracy with regard to peak linear and rotational acceleration. Earlier studies reported that the HIT System indicated resultant linear accelerations that were within 4% to 8% of the reference system in the anthropomorphic test device headform (45). However, more recent studies reported somewhat increased variability between the HIT System and the reference system. Across an extensive test series with laboratory impacts to 12 different helmet locations, the HIT System was shown to generally overestimate linear and rotational accelerations with impact location-based correlation slope values between 0.58 (underestimate) and 1.005 (overestimate) for linear acceleration and between 0.237 and 1.127 for rotational accelerations (44). Lowest accuracy for rotational acceleration was for impacts to the rear of the helmet and the facemask. Given the frequency of head impacts to the front and back of the helmet during contact practices and games (46), these inaccuracies in peak linear and rotation acceleration may have affected calculations for RWE incorporated into this analysis. However, assuming the inaccuracy to be consistent across the sample, this may have had a limited effect on the current dataset because it focused changes across the entire enrolled population and not the magnitude of specific head impacts.

Interpretation of these associations between HIE and concussion incidence requires consideration of the OR, statistical significance, and confidence interval. This analysis identified statistically significant associations ( $P < 0.05$ ) of preseason HIE with preseason and total season concussion incidence, and of total season HIE with total season concussion incidence. Those statistically significant associations were supported by strong OR. However, the precision of these findings is somewhat reduced by large confidence intervals for some of the significant associations shown in Table 2. Regardless, because the confidence intervals do not overlap the null value (i.e., lower bound  $> 1.0$ ) and the associations attained statistical significance ( $P < 0.05$ ), these findings still indicate strong positive associations between HIE and concussion incidence. In other words, some metrics of HIE were significantly associated with concussion incidence, despite reduced precision with regard to the strength of those associations. For example, the significant

associations of 50th percentile preseason RWE with 1) preseason concussion incidence and 2) total season concussion incidence both had very large confidence intervals. In both cases, the lower bound for the confidence interval was well above 1.0, which indicates a strong association. However, the large upper bound may indicate a possible confounding factor for increased strength of association in some athlete groups or may be representative of large variation in RWE values between individual athletes. Therefore, these findings may require additional data to more precisely characterize the strength of these associations, although the present findings provide strong and novel evidence for an association between levels of preseason HIE and season-long concussion incidence.

This study demonstrated strong associations between HIE and concussion incidence for NCAA Division I football athletes. Perhaps most importantly, levels of preseason HIE had strong associations with total season concussion incidence. This may indicate a prolonged effect of HIE on concussion risk that can last beyond the period of higher HIE and persist throughout the entire season. Athletes that have high exposure during the college football preseason may be more susceptible to concussion throughout the entire fall season. In addition, as demonstrated in the results of this study, preseason and regular season HIE considerably varied between individual athletes, which may influence individual tolerance for concussion. Although the mechanism for prolonged elevated concussion risk requires further investigation, this analysis highlights the deleterious role of high levels of preseason HIE and the need to explore coaching or regulatory adjustments to improve athlete safety.

Results of this study are presented clearly, honestly, and without fabrication, falsification, or inappropriate data manipulation. Results of the present study do not constitute endorsement by the American College of Sports Medicine.

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## REFERENCES

1. Broglio SP, Schnebel B, Sosnoff JJ, et al. Biomechanical properties of concussions in high school football. *Med Sci Sports Exerc.* 2010; 42(11):2064–71.
2. Campoletano ET, Gellner RA, Smith EP, et al. Development of a concussion risk function for a youth population using head linear and rotational acceleration. *Ann Biomed Eng.* 2020;48(1):92–103.
3. Elliott MR, Margulies SS, Maltese MR, Arbogast KB. Accounting for sampling variability, injury under-reporting, and sensor error in concussion injury risk curves. *J Biomech.* 2015;48(12):3059–65.
4. Rowson S, Duma SM. Brain injury prediction: assessing the combined probability of concussion using linear and rotational head acceleration. *Ann Biomed Eng.* 2013;41(5):873–82.
5. Stemper BD, Shah AS, Harezlak J, et al. Repetitive head impact exposure in college football following an NCAA rule change to eliminate two-a-day preseason practices: a study from the NCAA–DoD CARE Consortium. *Ann Biomed Eng.* 2019;47(10):2073–85.
6. McCrea MA, Shah A, Duma S, et al. Opportunities for prevention of concussion and repetitive head impact exposure in college football players: a Concussion Assessment, Research, and Education (CARE) Consortium Study. *JAMA Neurol.* 2021;78(3):346–50.
7. Broglio SP, Eckner JT, Martini D, Sosnoff JJ, Kutcher JS, Randolph C. Cumulative head impact burden in high school football. *J Neurotrauma.* 2011;28(10):2069–78.
8. Stemper BD, Shah AS, Harezlak J, et al. Comparison of head impact exposure between concussed football athletes and matched controls: evidence for a possible second mechanism of sport-related concussion. *Ann Biomed Eng.* 2019;47(10):2057–72.
9. Rowson S, Duma SM, Stemper BD, et al. Correlation of concussion symptom profile with head impact biomechanics: a case for individual-specific injury tolerance. *J Neurotrauma.* 2018;35(4): 681–90.
10. Beckwith JG, Greenwald RM, Chu JJ, et al. Timing of concussion diagnosis is related to head impact exposure prior to injury. *Med Sci Sports Exerc.* 2013;45(4):747–54.
11. Broglio SP, Lapointe A, O'Connor KL, McCrea M. Head impact density: a model to explain the elusive concussion threshold. *J Neurotrauma.* 2017;34(19):2675–83.
12. Bazarian JJ, Zhu T, Zhong J, et al. Persistent, long-term cerebral white matter changes after sports-related repetitive head impacts. *PLoS One.* 2014;9(4):e94734.

13. McAllister TW, Ford JC, Flashman LA, et al. Effect of head impacts on diffusivity measures in a cohort of collegiate contact sport athletes. *Neurology*. 2014;82(1):63–9.
14. Talavage TM, Nauman EA, Breedlove EL, et al. Functionally-detected cognitive impairment in high school football players without clinically-diagnosed concussion. *J Neurotrauma*. 2014;31(4):327–38.
15. Talavage TM, Nauman EA, Leverenz LJ. The role of medical imaging in the recharacterization of mild traumatic brain injury using youth sports as a laboratory. *Front Neurol*. 2016;6:273.
16. Urban JE, Davenport EM, Golman AJ, et al. Head impact exposure in youth football: high school ages 14 to 18 years and cumulative impact analysis. *Ann Biomed Eng*. 2013;41(12):2474–87.
17. Prins ML, Alexander D, Giza CC, Hovda DA. Repeated mild traumatic brain injury: mechanisms of cerebral vulnerability. *J Neurotrauma*. 2013;30(1):30–8.
18. Johnson VE, Stewart JE, Begbie FD, Trojanowski JQ, Smith DH, Stewart W. Inflammation and white matter degeneration persist for years after a single traumatic brain injury. *Brain*. 2013;136(1):28–42.
19. Ramlackhansingh AF, Brooks DJ, Greenwood RJ, et al. Inflammation after trauma: microglial activation and traumatic brain injury. *Ann Neurol*. 2011;70(3):374–83.
20. Broglio SP, McCrea M, McAllister T, et al. A national study on the effects of concussion in collegiate athletes and US Military Service Academy members: the NCAA–DoD Concussion Assessment, Research and Education (CARE) Consortium structure and methods. *Sports Med*. 2017;47(7):1437–51.
21. Carney N, Ghajar J, Jagoda A, et al. Concussion guidelines step 1: systematic review of prevalent indicators. *Neurosurgery*. 2014;75(1 Suppl):S3–15.
22. Crisco JJ, Chu JJ, Greenwald RM. An algorithm for estimating acceleration magnitude and impact location using multiple nonorthogonal single-axis accelerometers. *J Biomech Eng*. 2004;126(6):849–54.
23. Beckwith JG, Greenwald RM, Chu JJ, et al. Head impact exposure sustained by football players on days of diagnosed concussion. *Med Sci Sports Exerc*. 2013;45(4):737–46.
24. Dashnaw ML, Petraglia AL, Bailes JE. An overview of the basic science of concussion and subconcussion: where we are and where we are going. *Neurosurg Focus*. 2012;33(6):E5: 1–9.
25. Giza CC, Hovda DA. The new neurometabolic cascade of concussion. *Neurosurgery*. 2014;75 Suppl 4(0 4):S24–33.
26. Guskiewicz KM, McCrea M, Marshall SW, et al. Cumulative effects associated with recurrent concussion in collegiate football players: the NCAA concussion study. *JAMA*. 2003;290(19):2549–55.
27. McCrea M, Broglio S, McAllister T, et al. Return to play and risk of repeat concussion in collegiate football players: comparative analysis from the NCAA Concussion Study (1999–2001) and CARE Consortium (2014–2017). *Br J Sports Med*. 2020;54(2):102–9.
28. Shultz SR, MacFabe DF, Foley KA, Taylor R, Cain DP. Subconcussive brain injury in the Long-Evans rat induces acute neuroinflammation in the absence of behavioral impairments. *Behav Brain Res*. 2012;229(1):145–52.
29. Meier TB, Nelson LD, Huber DL, Bazarian JJ, Hayes RL, McCrea MA. Prospective assessment of acute blood markers of brain injury in sport-related concussion. *J Neurotrauma*. 2017;34(22):3134–42.
30. Bahrami N, Sharma D, Rosenthal S, et al. Subconcussive head impact exposure and white matter tract changes over a single season of youth football. *Radiology*. 2016;281(3):919–26.
31. Montenegro PH, Alosco ML, Martin BM, et al. Cumulative head impact exposure predicts later-life depression, apathy, executive dysfunction, and cognitive impairment in former high school and college football players. *J Neurotrauma*. 2017;34(2):328–40.
32. McAllister T, McCrea M. Long-term cognitive and neuropsychiatric consequences of repetitive concussion and head-impact exposure. *J Athl Train*. 2017;52(3):309–17.
33. Stemper BD, Shah AS, Mihalik JP, et al. Head impact exposure in college football after a reduction in preseason practices. *Med Sci Sports Exerc*. 2020;52(7):1629–38.
34. Broglio SP, Martini D, Kasper L, Eckner JT, Kutcher JS. Estimation of head impact exposure in high school football: implications for regulating contact practices. *Am J Sports Med*. 2013;41(12):2877–84.
35. Campoletano ET, Rowson S, Duma SM, et al. Factors affecting head impact exposure in college football practices: a multi-institutional study. *Ann Biomed Eng*. 2019;47(10):2086–93.
36. Duhaime AC, Beckwith JG, Maerlender AC, et al. Spectrum of acute clinical characteristics of diagnosed concussions in college athletes wearing instrumented helmets: clinical article. *J Neurosurg*. 2012;117(6):1092–9.
37. Stemper BD, Chirvi S, Doan N, et al. Biomechanical tolerance of whole lumbar spines in straightened posture subjected to axial acceleration. *J Orthop Res*. 2018;36(6):1747–56.
38. Nightingale RW, McElhaney JH, Camacho DL, Kleinberger M, Winkelstein BA, Myers BS. The dynamic responses of the cervical spine: buckling, end conditions, and tolerance in compressive impacts. *SAE Transact*. 1997;106:3968–88.
39. Kroell CK, Schneider DC, Nahum AM. Impact tolerance and response of the human thorax II. *SAE Transact*. 1974;83(4):3724–62.
40. McCrory P, Meeuwisse W, Dvořák J, et al. Consensus statement on concussion in sport—the 5(th) international conference on concussion in sport held in Berlin, October 2016. *Br J Sports Med*. 2017;51(11):838–47.
41. Asken BM, McCrea MA, Clugston JR, Snyder AR, Houck ZM, Bauer RM. “Playing through it”: delayed reporting and removal from athletic activity after concussion predicts prolonged recovery. *J Athl Train*. 2016;51(4):329–35.
42. Meehan WP 3rd, Mannix RC, O’Brien MJ, Collins MW. The prevalence of undiagnosed concussions in athletes. *Clin J Sport Med*. 2013;23(5):339–42.
43. Davies SC, Bird BM. Motivations for underreporting suspected concussion in college athletics. *J Clin Sport Psychol*. 2015;9(2):101–15.
44. Siegmund GP, Guskiewicz KM, Marshall SW, DeMarco AL, Bonin SJ. Laboratory validation of two wearable sensor systems for measuring head impact severity in football players. *Ann Biomed Eng*. 2016;44(4):1257–74.
45. Duma SM, Manoogian SJ, Bussone WR, et al. Analysis of real-time head accelerations in collegiate football players. *Clin J Sport Med*. 2005;15(1):3–8.
46. Crisco JJ, Fiore R, Beckwith JG, et al. Frequency and location of head impact exposures in individual collegiate football players. *J Athl Train*. 2010;45(6):549–59.