Role of Repetitive Head Impact Exposure in the Onset of Concussion:
Evidence of a Possible Second Mechanism of Concussion for Contact Sports

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I. INTRODUCTION

A primary mechanism of concussion in sports and automobile crashes has long been understood to involve head impact leading to high rate head accelerations [1]. These accelerations produce strains within the brain tissues that can exceed mechanical or physiologic thresholds and result in brain tissue injury and dysfunction. Injury risk functions were developed based on this understanding, using data derived from experimental testing of animals and physical models [2-3] and field studies that incorporated head impact sensors in athletes during athletic participation [4]. However, field studies focused on quantifying concussive impacts often reported wide variation in biomechanical parameters. For example, reported on the biomechanics of 10 concussions sustained by high school and collegiate American football athletes instrumented with the Head Impact Telemetry (HIT) System [5]. Those concussions were associated with head impact linear accelerations between 54 and 111 g's, and rotational accelerations between 2,174 and 6,560 rad/s^2. Other studies of American football and ice hockey athletes have reported similar or wider ranges for the biomechanics associated with concussion onset [6-7]. These findings imply that the head impact immediately preceding concussion may not be uniquely responsible for injury onset, which has led investigators to hypothesise a second mechanism involving repetitive head impact exposure (RHIE). This is supported by field studies demonstrating the development of cognitive and MRI changes in non-concussed athletes that were correlated to the number of head impacts during an athletic season. This study quantified the biomechanics of head impacts associated with concussion and RHIE relative to matched controls in concussed American football athletes to outline the possible mechanisms of concussion in contact sports.

II. METHODS

The Institutional Review Board at the Medical College of Wisconsin approved data collection and research methods for these studies. Informed consent was obtained from all participants prior to the initiation of data collection. Head impact accelerations were monitored during all contact activities using the Head Impact Telemetry (HIT) System (Riddell SRS, Riddell, Rosemont, IL, USA) for 608 NCAA Division III college and high school American football athletes at eight Institutions during the 2015-2017 seasons. The HIT System is a helmet-based head impact monitoring system that continually monitors head linear accelerations using six uniaxial accelerometers. Data acquisition triggered when a single accelerometer exceeded a 9.6-g threshold. Only impacts with peak resultant linear acceleration greater than 10 g were included for analysis.

Athlete concussions were identified and diagnosed by team medical staff according to a standardised protocol. Concussed athletes were entered into the concussion protocol and local study team members recorded detailed data on date and time of injury, activity type (practice, scrimmage, game), type of play, and location and direction of head impact. Following notification of the injury, the study team secured the HIT System and video data for the date of injury and participated in a detailed analysis to identify the head impact associated with concussion onset. The analysis included at least two team members and accounted for information in the concussion report, as well as head impact and video data from the injury date. *High risk* head impacts were associated with linear accelerations equal to or greater than 75 g and rotational accelerations equal to or greater than 3,500 rad/s^2.

RHIE was quantified on the injury date and for the season up to and including the injury date (season) using a previously-defined Cumulative Metric (CM) [8]. In short, the CM was calculated as the sum of concussion risk [4]

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associated with every recorded head impact for the athlete over the period of interest. Accordingly, CM magnitude increased for a greater number of head impacts and higher severity (linear/rotational acceleration) head impacts. CM for the injured athlete was compared to a matched control group that consisted of athletes that played the same position group on the same team, as those factors were previously shown to influence RHIE. Control athletes that participated in 30% greater or 30% fewer total contact days than the injured athletes for the season up to and including the injury date were not included in the analysis.

III. INITIAL FINDINGS

Forty-three athletes sustained concussion while instrumented with a HIT System after participating in 1-60 days of contact activities (average: 21 ± 14 days). Six concussions occurred in August, 17 in September, 12 in October, and 8 in November. Thirty-nine athletes had an average of 6.4 ± 3.1 matched controls each on the injury date and 5.5 ± 3.0 matched controls each for the season. Four athletes had zero matched controls on the injury date and another four athletes had zero matched controls for the season. Two of those four athletes had zero matched controls for either time period. Average linear and rotational accelerations for the 43 concussive head impacts were 71.8 ± 32.5 g and 3,337 ± 1,628 rad/s^2. Nineteen concussed athletes had concussive head impacts that were classified as *high risk*, with the remaining 24 athletes sustaining concussion in the absence of a remarkable head impact. Sixteen of those 19 concussed athletes also had evidence of high RHIE either on the injury date or for the season. Of the remaining 24 concussed athletes, 14 had evidence of high RHIE prior to concussion onset, with one athlete having no matched controls. Six of those 14 had the highest CM for their matched control group for the season, another four had the second highest CM for their matched control group for the season, eight athletes had the second highest CM for their matched control groups on the injury date, and one athlete had the highest CM for his matched control group on the injury date. The remaining 9 athletes had neither a remarkable concussive head impact nor significant RHIE up to the date of concussion. Seven of those 9 participated in 17 or fewer days of contact activities prior to concussion.

IV. DISCUSSION

These data highlight two possible mechanisms for concussion onset in contact sports. Forty-four percent of the concussed athletes in this study had *high risk* head impacts on the date of concussion that can be directly associated with concussion onset. However, the remaining 56% of concussed athletes did not have a remarkable head impact on the date of concussion and, therefore, concussion onset should be attributable to other factors. From a mechanical standpoint, fatigue-related injury is common in other tissues of the body [8] and progressive damage to the brain associated with RHIE appears plausible. As with fatigue failure, injury onset would then be associated with an unremarkable head impact. This appeared to be the case in 14 (33%) concussed athletes in this study that had evidence of elevated RHIE during the season leading up to their concussion, compared to athletes in the same position group on the same team. The percentage of athletes with evidence of significant RHIE in this study is somewhat lower than our previous analysis in NCAA Division I athletes [9], which may indicate higher RHIE magnitudes of the highest level of amateur football in the USA.

V. REFERENCES

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