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No Clinical Predictors of Postconcussion Musculoskeletal Injury in College Athletes

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Abstract

Purpose: To identify clinical predictors of post-concussion subsequent musculoskeletal (MSK) injuries.

Methods: We recruited 66 NCAA intercollegiate student-athletes with a diagnosed concussion as well as 36 NCAA student-athletes without a concussion. All participants completed a multifaceted concussion baseline consisting of 1) 22-item 0–6 self-reported symptom checklist with outcomes including both the number of symptoms endorsed (0 – 22) and 2) total symptom score (0 – 132), 3) Standard Assessment of Concussion (SAC), 4) Balance Error Scoring System (BESS), 5) Immediate Post-Concussion Assessment and Cognitive Testing (ImPACT), 6) clinical reaction time (CRT), and 7) the King-Devick (KD) as well as demographic and injury characteristics. The concussion participants completed the same exam acutely post-concussion and binary logistic regression was used to identify predictors of subsequent MSK from the change scores (Acute minus Baseline). From the 66 concussed student-athletes, a subset 36, matched with the healthy athletes, compared the risk of subsequent MSK in the year prior to and year following their concussion.

Results: The concussion participants were 1.78x (95% CI: 1.12 – 2.84, p=0.015) more likely to suffer a LE MSK in the year following their concussion then the control participants. The participant demographics and injury characteristics (p=0.318) and concussion clinical outcomes (p=0.461) did not predict subsequent MSK.

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CONFLICT OF INTERESTS.

There are no conflicts of interest related to this study and no companies or manufacturers will benefit from the results. The host institution did receive free use of the electronic King-Devick test as part of their enrollment in the NCAA-DoD CARE study, but no one from the company had any role in the development, design, analysis, or interpretation of this experiment. The results of this study do not constitute endorsement by ACSM. The results of the study are presented clearly, honestly, and without fabrication, falsification, or inappropriate data manipulation.

Conclusion: The concussion participants were 1.78x more likely to sustain a subsequent MSK; however, no demographic, injury characteristic, or concussion assessments predicted the MSK. Thus, clinicians are not able to utilize common neurological measures or participant demographics to identify those at risk for subsequent LE MSK. Injury prevention strategies should be considered for collegiate student-athletes upon RTP following a concussion to reduce the subsequent MSK.

Keywords

Mild Traumatic Brain Injury; Injury Prediction; Balance; Neurocognitive

INTRODUCTION

Sports related concussions impair numerous components of the central nervous system resulting in cognitive, postural control, cardiovascular function, and oculomotor deficits,(1–7) thus the current recommendation from the 5th Concussion in Sport (5th CIS) consensus statement incorporates a multifaceted assessment battery.(1, 8) Typical clinical practice involves a baseline test prior to participation which is then re-administered following a suspected concussion in support of a clinical diagnosis.(9–12) While the sensitivity of this battery can exceed 90% acutely post-concussion,(13) it lacks sensitivity to recovery likely due to a practice effect secondary to repeat administration and poor test-retest reliability.(14–16) Indeed, numerous approaches (e.g., neuroimaging, laboratory tests, and instrumented gait) have identified persistent deficits beyond clinical recovery suggesting athletes may be returning to participation prior to complete neurological recovery.(16, 17)

Historically, the primary concern related to premature return to participation (RTP) was an elevated risk of subsequent concussion, particularly within the first two weeks. Modern advances in management protocols have seen substantial reductions in recurrent concussion risk.(18) Recently, the elevated risk of musculoskeletal injury (MSK), particularly to the lower extremity (LE), in the year following concussion has emerged as an additional concern.(19–30) A diverse range of study methodologies and populations including high school, college, and professional (both current and retired) athletes have consistently identified an elevated rate (1.3 – 3.4) of post-concussion LE MSK.(19–30) While the rate of subsequent LE MSK is highly consistent across studies, limited evidence exists identifying the underlying mechanism.(19) Persistent neurological deficits in the cognitive or postural control have been postulated as a potential mechanism.(19, 31) Howell identified lingering dual task postural control deficits could predispose athletes to LE MSK.(20) Houston reported that both female athletes and athletes with a history of multiple concussions had elevated rates (1.9 – 4.3x) of subsequent LE MSK; however, the study design precluded identification of underlying mechanisms.(23) Thus, clinicians have limited ability to identify which athletes are at risk of post-concussion LE MSK.

Lower extremity musculoskeletal injuries, such as lateral ankle sprains are highly commonplace with an estimated 9 million incidents annually in the U.S. and an aggravate annual cost of $2 – 4B USD.(32, 33) Anterior cruciate ligament (ACL) injuries, while less common, require surgical repair, lost academic and athletic time, with annual costs of approximately $4B USD.(34) In a retrospective study, Gilbert reported that 70.8% (17/24) of

collegiate athletes who had an ACL injury also had experienced a concussion, although no mechanism was identified.(26) Both lateral ankle sprains and ACL injuries are associated with elevated risk of osteoarthritis and Lynall identified a dose-response relationship in retired NFL players whereby the increasing number of concussions was associated with a significantly greater prevalence of diagnosed osteoarthritis.(35) Thus, identifying at-risk individuals and implementing injury prevention programs can potentially reduce the substantial personal and economic costs of post-concussion subsequent LE MSK.

Early preliminary evidence suggests impaired postural control, as measured by instrumented assessments, may be only currently identified modifiable predictor of subsequent LE MSK; (20) however, this approach is not clinically feasible for health care providers. However, if standard clinical examination outcomes, injury presentation and recovery, as well as patient demographics can identify individuals at elevated risks, clinically feasible injury prevention strategies can be implemented. Therefore, the primary purpose of this study was to identify clinical predictors of post-concussion MSK injuries in collegiate student-athletes. We hypothesized that increased post-concussion time loss, female sex, and poor performance on the clinical concussion assessment battery would predict elevated rates of subsequent MSK injury Secondarily, we aimed to confirm the presence of an elevated subsequent LE MSK injury rate. We hypothesized, consistent with prior studies, there would be an elevated post-concussion subsequent injury rate as compared to matched control athletes.

METHODS

Participants

We recruited 66 intercollegiate student-athletes with a diagnosed concussion identified by an athletic trainer and diagnosed by the team physician using guidelines consistent with the 4th or 5th Concussion in Sports Consensus statement based on the current guideline in effect at the time of the evaluation (2015 – 2018).(1, 36) (Table 1) All individuals participated in intercollegiate athletics for at least one year prior to and one year subsequent to the concussion at the host institution. The concussion participants all had performed a baseline concussion assessment prior to participation and, after returning to baseline values (equal to or better performance at post-injury than at baseline on all assessments), completed a progressive RTP protocol consistent with the current CIS guidelines in place at the time of injury.(1, 36) The exclusion criteria were a prior concussion at the university, a concurrent injury with their concussion which restricted return to participation status (e.g., cervical injuries, substantial orthopedic injury), invalid concussion baseline test, delayed concussion reporting beyond 48 hours which precluded an acute assessment time point, or other incomplete or missing assessments. Concussions which occurred prior to the collegiate level were not an exclusion criteria given the noted low validity and reliability of concussion reporting.

To address the second aim, confirming the presence of an elevated post-concussion LE MSK risk, a subset of the concussion participants (N=36) and 36 matched control student-athletes were recruited. (Table 1) The concussion participants in this subset were tightly matched to control participants who were teammates that played the same sport, the same or similar positions, had comparable anthropometric characteristics, and who had no documented

history of concussion. The controls were retrospectively matched to the date of concussion RTP for their matched participant and also had one year of intercollegiate athletic participation before and after their matched concussion RTP date (i.e., if the concussion participant RTP on October 1st, then both participants were followed for one year before and one year after the October 1st date thus ensuring the identical two-year window was used for both participants). This matching criteria limited the concussion group to only 36 participants as the remaining 30 concussions could not be matched with these criteria. While this approach restricted the number of control participants available, it limited differences due to changes in coaching staffs, season specific differences (e.g., playoffs or no playoffs), and ensured both groups had similar exposures (e.g., practices, games, and workouts). The exclusion criteria for the matched participants included a lifetime history of concussion which was assessed through a reliable questionnaire(26) as well as interview format to identify probable concussions (e.g., memory loss following a head impact), the student-athlete leaving the team, or incomplete medical records. All participants provided written and oral informed consent as approved by the university's Institutional Review Board.

Procedures

All participants completed a baseline concussion assessment prior to the start of their intercollegiate athletic careers.(11) The assessment battery was consistent with the NCAA/DoD CARE protocol and included 1) a 22-item 0–6 self-reported symptom checklist with outcomes including both the number of symptoms endorsed (0 – 22) and 2) total symptom score (0 – 132), 3) Standard Assessment of Concussion (SAC) mental screening, 4) Balance Error Scoring System (BESS) for static postural control, 5) Immediate Post-Concussion Assessment and Cognitive Testing (ImPACT) for neurocognition, 6) clinical reaction time (CRT), and 7) the King-Devick (KD) for horizontal saccade performance.(8, 11) (Table 2) These tests have been extensively described in the literature and are commonly utilized in concussion management.(1, 8–11, 14)

Following a suspected concussion, the student-athlete was assessed by a certified athletic trainer who identified the concussion which was confirmed by a licensed physician consistent with the current CIS guidelines.(1) The student-athlete was assessed acutely post-concussion (≤48 hours) on the concussion assessment battery which helped confirm the clinical concussion diagnosis and was the "Acute" time point. Consistent with current recommendations, student-athletes completed sub-symptomatic activities of daily living until symptom free and had serial follow-up assessments with both the athletic trainers and team physicians.(1) Once the student-athlete was symptom free, achieved baseline values on the multifaceted concussion assessment, and received clearance from the team physician, the student-athlete completed an approximately six-day progressive exercise program which concluded with unrestricted RTP.(1) If the student-athlete experienced symptom provocation during the progressive exercise program, they were stopped for the day and regressed one step in the protocol the following day.

Following RTP, all participants were tracked for one calendar year, both pre and post-injury, for LE MSK. As per standard clinical injury management, the athletic trainers recorded all injuries in an electronic medical record (EMR) and review of these records was included in

the participant's informed consent. The LE MSK was defined as acute injuries (e.g., fracture, sprain, strains) which occurred to the foot, ankle, lower leg, knee, thigh, or hip and required at least one day of limited activity along with receiving treatment.(37) Chronic overuse injuries, general medical illnesses, and non-MSK injuries (e.g., contusions, abrasions) were not considered LE MSK injuries.(24)

Statistical Analysis

The outcome measures for each dependent variable are provided in Table 2. The change score from Baseline to Acute (≤48 hours) for each assessment served as the dependent variable for each concussion assessment outcome. All ImPACT tests were reviewed by the research team and no invalid baseline tests were identified. The total number of days missed for the concussion was the time from the injury occurred to the first day of unrestricted participation. The presence of loss of consciousness (LOC), post-traumatic amnesia (PTA), concussion history, sport type (contact/collision or non-contact) and sex were coded as binary variables.

Subsequent MSK Injury Risk Analysis—A Cox proportional hazard model was used to assess the risk of subsequent LE MSK between groups the concussion and control groups (N=36 per group). Within the model, the study end point was 365 days from the day of RTP or the occurrence of a new injury, whichever came first.(27) The presence of a LE MSK in the prior year was included as a binomial covariate (yes/no) in the model. Time to further injury was also assessed using a Mann-Whitney U test for the number of days to a LE MSK only for those participants who experienced a LE MSK.(24)

Predictors of Subsequent MSK Analysis—To assess the capability of the clinical outcome measures, concussion characteristics, and demographics to predict a subsequent LE MSK, two sequential binary logistic regressions with Enter method were performed for all 66 concussion participants. The clinical outcomes measure binary logistic regression predictors included the change scores (Acute minus Baseline) for; symptoms endorsed, total symptom score, SAC, BESS, ImPACT (each of the four composite scores), CRT, and KD. The second binary logistic regression incorporated demographic and injury characteristics; presence of PTA, presence of LOC, sport type, prior MSK injury, prior concussion, days missed, and sex.

Concussion Assessment Battery Outcomes—A 2 (group: LEMSK and no-LEMSK) x 2 (Time: Baseline and Acute) repeated measures ANOVA was performed to compare performance on each of the concussion assessment battery outcome measures for the Concussion participants. As no interaction was hypothesized, exploratory post-hoc was intentionally performed to assess Concussion group (LEMSK and No-LEMSK) differences at each time point (Baseline, Acute) and to assess Time (Baseline to Acute). Partial eta squared (η^2) effect sizes, classified as small (0.01), medium (0.06), and large (>0.14), were calculated for significant interactions.

RESULTS

Subsequent LE MSK Risk

The concussion participants were more likely to suffer a LE MSK in the year following their concussion then the control participants (Hazard ratio (HR) = 1.78x (95% CI: 1.12 – 2.84, p=0.015). (Figure 1) Among the participants who experienced a subsequent MSK, the mean time to injury was 160.1 ± 101.7 days in the Concussion group and 244.2 ± 97.3 days for the Control group (U=354, p<0.001) . There was no difference between groups for the year prior to the concussion (HR: 0.858, 95% CI: 0.52 – 2.09, p=0.910).

Predictors of Subsequent MSK

The participant demographic and injury characteristics model did not predict subsequent LE MSK (p=0.318, Exp(B)=1.020, Nagelkerke R^2 = 0.223). Post-hoc testing failed to identify any individual predictors. (Table 3)

The concussion clinical outcomes model did not predict subsequent LE MSK (p=0.461, Exp(B)=1.200, Nagelkerke R^2 = 0.183). Post-hoc testing failed to identify any individual predictors. (Table 4)

Concussion Assessment Battery Outcomes

There were no significant interactions for any of the concussion outcome measures: Symptom Severity (F=0.530, p=0.469, power=1.000), number of Symptoms (F=0.953, p=0.333, power=1.000), SAC (F=0.350, p=0.556, power=0.759), BESS (F=0.858, p=0.358, power=0.174), Verbal Memory (F=1.528, p=0.221, power=0.552), Visual Memory (F=0.000, p=0.996, power=0.696), Motor Speed (F=3.968, p=0.051, power=0.692), Reaction Time (F=1.127, p=0.292, power=0.932), Clinical Reaction Time (F=1.957, p=0.167, power=0.946), and King Devick (F=0.316, p=0.576, power=0.974).

There were no significant differences between concussion groups (LEMSK and No-LEMSK) on concussion test performance at baseline (p>0.05) or at the Acute (p>0.05) time points. (Table 3) There were significant differences between Baseline and Acute for Symptom Severity (baseline: 3.8 ± 7.6, Acute: 28.3 ± 19.6, F=110.964, p<0.001, η^2 = 0.638), number of Symptoms (baseline: 2.2 ± 3.7, Acute: 12.4 ± 7.6, F=94.355, p<0.001, η^2=0.630), SAC (baseline: 27.1 ± 1.8, Acute: 26.2 ± 2.0, F=5.973, p=0.016, η^2=0.106), Visual Memory (Baseline: 77.5 ± 13.5, Acute: 72.5 ± 13.9, F=4.396, p=0.038, η^2=0.089), Motor Speed (Baseline: 40.6 ± 6.0, Acute: 38.7 ± 7.2, F=7.604, p=0.008 η^2=01.08), Reaction Time (Baseline: 0.58 ± 0.06, Acute: 0.63 ± 0.11, F=7.880, p=0.006, η^2=0.171), Clinical Reaction Time (Baseline: 205.6 ± 24.2, Acute: 229.7 ± 56.1, F=10.108, p=0.002, η^2=0.185), and King-Devick (Baseline: 40.3 ± 5.7, Acute: 48.2 ± 17.7, F=11.730, p=0.001, η^2=0.201). There were no differences between Baseline and Acute for BESS (Baseline: 15.0 ± 7.0, Acute: 15.8 ± 5.8 errors, F=0.609, p=0.437, η^2=0.013) and Verbal Memory (Baseline: 86.4 ± 11.2, Acute: 86.6 ± 13.8, F=0.008, p=0.928, η^2=0.001).

DISCUSSION

An elevated risk (1.3 – 3.4x) of LE MSK injury in the year following concussion has recently been routinely identified;(19–30) however clinical predictors of this risk are limited.(20) Herein, the concussion participants were nearly 1.8x more likely to suffer a LE MSK in the year following concussion as compared to a tightly matched control group and there was no difference between groups in the year prior to concussion. However, within the concussion participants only, there were no predictors from the concussion clinical assessment battery, concussion presentation, or demographics which predicted the subsequent LE MSK. This finding reinforced the elevated LE MSK risk following concussion, but failed to identify any subsequent LE MSK predictors from clinically feasible data. Thus, clinicians are not able to utilize common neurological measures to identify those at risk for subsequent LE MSK for targeted injury prevention interventions.

There are no established predictors of post-concussion LE MSK and there is mixed evidence in the literature for the initial concussion presentation predicting either prolonged recovery or subsequent concussion.(38) Of these, elevated symptom score, presence of PTA, female sex, and prior concussion tended to have the strongest predictive capabilities and were plausible subsequent LE MSK predictors;(38) however, none of these were significant predictors here. The last decade has seen considerable changes in clinical concussion management (e.g., more conservative treatment, longer durations before RTP) and it is unclear how these changes influence subsequent injury risk.(12, 39) Recently, McCrea identified a substantial reduction in same season subsequent concussion in football players in CARE compared to two decades earlier suggesting current management techniques have reduced overall risks.(18) As all the participants in this study were enrolled in CARE Grand Alliance, this more conservative approach (mean time loss: 18.4 ± 17.2) is well beyond the commonly reported 7 – 10 days.(1) While not statistically significant, it is interesting to note the subsequent LE MSK group RTP 6.4 days earlier than the non-LE MSK group and future studies should continue to investigate this in larger cohorts including quadratic analysis to investigate if either “too-short” or “too-long” of recovery are risk factors. .

There are currently two hypothesized predictors of subsequent LE MSK in the literature;(20, 23) female athletes with a prior concussion history and athletes with worsening dual task postural control at RTP. Herein, sex (p=0.799 Exp(B): 0.836), concussion history (p=0.563, Exp(B): 2.046), and number of prior concussions (p=0.210, Exp(B):0.321) were not significant predictors. Houston utilized a retrospective survey, in a larger population (N=468), but was unable to identify an injury timeline suggesting that the LE MSK could have preceded the concussion.(23) Nordstrom previously identified an elevated injury rate in both the year prior to and the year after a concussion and suggested that these individuals could simply be “injury prone” or at elevated risk due to risky/aggressive on-field behavior.(27) Herein there was no difference in the LE MSK injury rate in the year prior to concussion (p=0.795, Exp(B): 0.858) between the LE MSK (36.1%) and the no-LE MSK groups (32.1%) which reduces the likelihood of injury prone being the explanation. The participants herein were closely matched by sport and position making risk exposure unlikely to explain the elevated risk. Howell previously identified a worsening of dual task gait at RTP in adolescent athletes; however, our clinical concussion assessment was limited

to single task assessments and neither the BESS or SAC acute changes predicted subsequent LE MSK.(20) Future studies should investigate instrumented gait and posture measures, including pre-injury data, as potential predictors of subsequent LE MSK as the potential for persistent neurophysiological deficits beyond clinical recovery is a plausible potential mechanism.

Within the Concussion participants, no group by time interaction was hypothesized for any of the outcome measures and no significant interactions were identified. Furthermore, there were no differences between groups (LEMSK and No-LEMSK) at either Baseline or Acute assessment time points suggesting there were no underlying differences between groups which influenced the outcomes. As expected, and consistent with most concussion literature, there were significant differences between Baseline and Acute post-concussion, with moderate to large effect sizes, for concussion measures except BESS and ImPACT Verbal Memory composite score.(1, 15) The lack of differences in the BESS may result from the known test psychometric limitations (e.g., practice effects, high minimal detectable change scores) and low sensitivity.(3, 15) The ImPACT Verbal Memory composite score was not identified as a key assessment to improve concussion battery optimization in a recent CARE investigation.(15) Despite these two specific outcomes not being significantly worse at the 48 hour test time point, the remainder of the multifaceted assessment battery demonstrated changes consistent with previous findings and suggest these were “typical” concussions.(6, 13, 15)

The participants were NCAA student-athletes and the results should not be extrapolated to other populations. Furthermore, as enrolled participants in the CARE Grand Alliance the athletes followed highly prescribed timelines and testing by research staff independent of the athletic staff which may not reflect standard care at other collegiate athletic programs. Concussions which occurred prior to college were not an exclusion criteria as concussion reporting reliability is notoriously poor.(40) The clinical assessment battery has poor to moderate reliability likely due to testing and scoring inconsistencies and a practice effect from repeat test administration;(14, 17) however these tests are recommended components and/or commonly utilized by clinicians thus increasing ecological validity.(8–11) Future work should investigate more comprehensive neurophysiological assessments (e.g., neuroimaging, blood based biomarkers, instrumented postural control, neuropsychologist administered cognitive assessments) in an effort to identify predictors of subsequent LE MSK. While this study was adequately powered to identify the elevated risk over time, it was underpowered to identify individual predictors. Finally, larger cohorts in future studies could use more sophisticated analysis techniques (e.g., Forest models) which would allow for the development of cut-points in outcomes measures as predictors.

The participants in this study were ~1.8x more likely to suffer an LE MSK in the year following a concussion than closely matched control participants. However, no clinical predictors were identified which restricts the ability of sports medicine professionals to identify individuals at elevated risk of subsequent LE MSK. Moving forward, injury prevention strategies and protocols should be considered for collegiate student-athletes upon RTP following a concussion in an effort to reduce the subsequent LE MSK.

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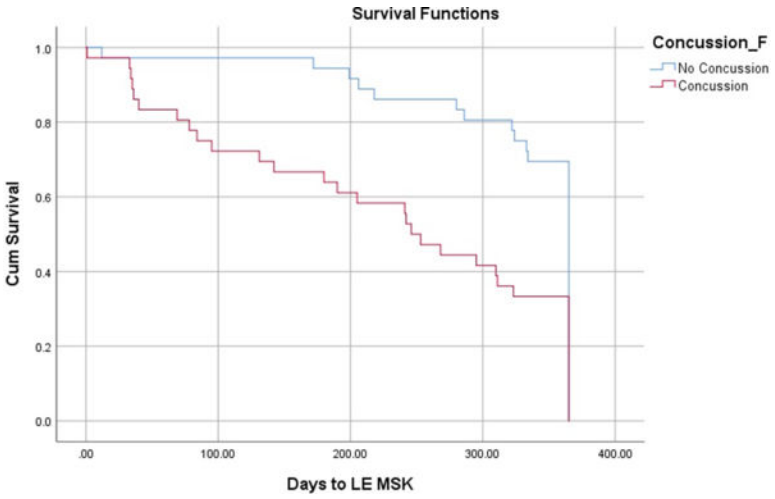

Figure 1.

There was a significant difference between groups for subsequent LEMSK (Wald: 5.925, p=0.015). The Concussion group was 1.78 (1.12 – 2.84) times more likely to experience a LEMSK than the No-Concussion group.

Table 1.

Demographics and Anthropometrics.

There were no significant differences between groups for any of the demographic characteristics. The Concussion Group Demographics section refers to the 66 concussion participants alone for which clinical, demographic, and injury characteristics prediction models were calculated. The Subsequent Injury Subgroup Demographics refers to the matched participants (Concussion, Non-Concussions groups) to evaluate the risk of subsequent injury.

	Concussion Group Demographics				**Subsequent Injury Subgroups Demographics**	
	Subsequent LEMSK (N=36)	**No Subsequent LEMSK (N=30)**	**Concussion Group Overall (N=66)**		**Concussion Group (N=36)**	**Non-Concussion Control Group (N=36)**
Sex	Male: 52.8% 19/36	Female: 60% 18/30	Female: 53.0% 37/66		Female: 52.8% 19/36	Female: 52.8% 19/36
Age (years)	19.9 ± 1.0 (Range: 18 – 22)	20.2 ± 1.2 (Range: 18 – 23)	20.0 ± 1.1 (Range: 18 – 23)		19.9 ± 1.3 (Range:18 – 22)	19.8 ± 1.1 (Range: 18 – 22)
Height (cm)	177.5 ± 10.0 (Range: 161 – 205)	171.7 ± 11.6 (Range: 142 – 198)	174.9 ± 11.0 (Range: 142 – 205)		177.2 ± 12.3 (Range: 152 – 206)	175.3 ± 10.8 (Range: 151 – 196)
Weight (kg)	82.3 ± 23.4 (Range: 53 – 139)	73.8 ± 16.7 (Range: 50 – 125)	78.7 ± 20.9 (Range: 50 – 139)		78.2 ± 20.7 (Range: 45 – 139)	75.0 ± 19.9 (Range: 54 – 135)
Sports	Football: 16.7% M Soccer: 13.9% Volleyball: 11.1% M Lax: 11.1% W Lax: 8.3% W Soccer: 8.3% W Basketball: 8.3% M Basketball: 5.6% W T&F: 2.8% Crew: 2.8% Field Hockey: 2.8% Baseball: 2.8% M Tennis: 2.8% W Swim: 2.8%	Football: 23.3% Softball: 13.3% Volleyball: 10% Cheer: 10% M Soccer: 6.7% M Lax: 6.7% W Lax: 6.7% Crew: 6.7% M Basketball: 3.3% Field Hockey: 3.3% W Soccer: 3.3%	Football: 19.7% Volleyball: 10.6% M Soccer: 10.6% M Lax: 9.1% W Lax: 7.6% Softball: 6.1% W Soccer: 6.1% Cheerleading: 4.5% W T&F: 4.5% Crew: 4.5% M Basketball: 4.5% W Basketball: 4.5% Field Hockey: 3.0% Baseball: 1.5% M Tennis: 1.5% W Swim: 1.5%		Football: 19.4% M Soccer: 11.1% Volleyball: 11.1% M Lax: 8.3% W Lax: 8.3% W Soccer: 8.3% W Basketball: 5.6% Softball: 8.3% M Basketball: 5.6% Cheerleading: 2.8% Crew: 2.8% Field Hockey: 2.8% Baseball: 2.8% W T & F: 2.8%	Football: 19.4% M Soccer: 11.1% Volleyball: 11.1% M Lax: 8.3% W Lax: 8.3% W Soccer: 8.3% W Basketball: 5.6% Softball: 8.3% M Basketball: 5.6% Cheerleading: 2.8% Crew: 2.8% Field Hockey: 2.8% Baseball: 2.8% W T & F: 2.8%

Table 2.

Clinical Outcome Measures.

Dependent Variable	Outcome Measure
Symptoms	The total number of symptoms reported (0 – 22)
Graded Symptom Checklist	The weighted total (0 – 6) of the 22 symptoms with higher scores reflecting greater symptom burden (0 – 132)
Standard Assessment of Concussion (SAC)	The total score (0 – 30) with a higher score reflecting better performance
Balance Error Scoring System (BESS)	The total number of errors across the six stances (0 – 60) with a lower score reflecting better performance
Clinical Reaction Time (CRT)	The mean of 8 trials with a lower score reflecting better performance
King Devick (KD)	At baseline, the faster of two trials which was performed error free. Following concussion, only trial is performed. A faster time reflects better performance.
Immediate Post-Concussion Assessment and Cognitive Testing (ImPACT)	Verbal Memory: A higher score reflects better performance Visual Memory: A higher score reflects better performance Motor Speed: A higher score reflects better performance Reaction Time: A lower score reflects better performance

Table 3.

Concussion Clinical Outcomes Regression Outcomes.

	No LEMSK Group		LEMSK Group		Regression Outcome	
	Baseline	**Acute Concussion**	**Baseline**	**Acute Concussion**	**p-Value**	**Exp(B)(95% CI Exp B)**
Total GSC Score (0 – 132) *	5.4 ± 9.7	31.9 ± 23.6	2.4 ± 5.1	25.5 ± 15.3	0.738	0.993 (0.950 – 1.037)
Symptoms Endorsed (0 – 22) *	2.9 ± 4.3	14.2 ± 9.8	1.6 ± 3.1	10.9 ± 5.0	0.619	0.979 (0.899 – 1.066)
SAC (0 – 30) *	27.0 ± 1.9	26.0 ± 1.9	27.1 ± 1.8	26.4 ± 2.2	0.983	0.997 (0.784 – 1.268)
BESS (0 – 60)	15.6 ± 6.3	15.2 ± 5.9	14.5 ± 8.1	13.9 ± 6.1	0.474	1.032 (0.947 – 1.125
Verbal Memory	85.0 ± 10.5	87.6 ± 13.4	87.6 ± 11.7	85.9 ± 14.2	0.064	0.957 (0.913 – 1.003)
Visual Memory *	76.9 ± 13.1	71.8 ± 13.9	78.1 ± 14.0	73.0 ± 14.1	0.724	1.006 (0.971 – 1.043)
Motor Speed *	41.5 ± 5.2	37.9 ± 7.3	39.8 ± 6.6	39.2 ± 7.1	0.297	1.068 (0.944 – 1.208)
Reaction Time *	0.58 ± 0.06	0.64 ± 0.14	0.58 ± 0.06	0.62 ± 0.10	0.642	0.994 (0.000 – 235.1)
Clinical Reaction Time *	208.9 ± 23.7	243.2 ± 71.8	203.0 ± 24.6	218.8 ± 36.8	0.446	0.994 (0.980 – 1.009)
King Devick *	40.8 ± 6.2	50.0 ± 21.3	40.0 ± 5.3	46.9 ± 14.3	0.792	1.007 (0.958 – 1.058)

* Main effect for time with the Acute Concussion time point worse ($p<0.05$) than baseline. There were no differences between groups (LEMSK and No-LEMSK) at either Baseline or Acute Concussion time points. None of the concussion clinical test outcomes were significant predictors of subsequent MSK.

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Table 4.

Demographic and Injury Characteristics.

There were no significant differences between groups for either demographic characteristics or initial injury presentation. None of the participant's characteristics was a significant predictor of subsequent MSK. There were no LOC participants in the No-LEMSK group therefore a binary logistic regression could not be calculated.

	No-LEMSK Group (N=30)	LEMSK Group (N=36)	Regression Outcome	
			p-Value	Exp(B) (95% CI Exp B)
Sex	60.0% Female (18/28)	50% Female (18/36)	0.799	0.836 (.211 – 3.311)
Concussion History (Yes/No)	50% Yes (15/30)	41.7% Yes (15/36)	0.563	2.046 (0.181 – 23.159)
Concussion History (Number)	0.8 ± 1.0 (Range: 0 – 3)	0.5 ± 0.6 (Range: 0 – 2)	0.210	0.321 (0.054 – 1.896)
Sport Type (Collision/Contact vs Non-Contact)	43.3% Non-Contact (13/30)	27.8% Non-Contact (10/36)	0.244	0.426 (0.102 – 1.789)
Prior Injury in the Previous Year (Yes/No)	32.1% No Prior Injury (9/28)	36.1% No Prior Injury (13/36)	0.795	0.858 (0.270 – 2.723)
Days Lost	Mean: 22.0 + 23.1 Days Median: 14.5 Days (Range: 5 – 119)	Mean: 15.6 ± 10.2 Days Median: 13.5 Days (Range: 5 – 46)	0.994	0.999 (0.863 – 1.157)
Post Traumatic Amnesia (Yes/No)	6.7% PTA (2/30)	11.1% PTA (4/36)	0.366	0.302 (0.023 – 4.042)
Loss of Consciousness	0% LOC (0/30)	8.3% LOC (3/36)	N/A	N/A