Journal of Athletic Training 2020;55(2):109–115
doi: 10.4085/1062-6050-187-19

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Baseline Postural Control and Lower Extremity Injury Incidence Among Those With a History of Concussion

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Context: Lower extremity musculoskeletal (LEMSK) injury may be more prevalent among those with a history of sport-related concussion (SRC).

Objective: To investigate the relationship between baseline postural control metrics and the LEMSK injury incidence in National Collegiate Athletic Association Division I student-athletes with a history of SRC.

Setting: National Collegiate Athletic Association Division I athletes.

Design: Cohort study.

Patients or Other Participants: Of 84 total athletes (62 males), 42 had been previously diagnosed with an SRC, and 42 were matched controls based on age, sex, height, weight, and sport.

Main Outcome Measure(s): During the preseason baseline evaluation, all participants performed 3 trials of eyes-open and eyes-closed upright quiet stance on a force platform. Medical charts were assessed for all the LEMSK injuries that occurred from preseason baseline to 1 year later. Center-of-pressure data in the anteroposterior and mediolateral directions were filtered before we calculated root mean square and mean excursion velocity; the complexity index was calculated from the unfiltered data. Factorial analysis-of-variance models were used to examine differences between groups and across conditions for root mean square; mean excursion velocity, complexity index, and tests of association to examine between-groups LEMSK differences; and logistic regression models to predict LEMSK.

Results: Concussion history and injury incidence were related in the SRC group ($P = .043$). The complexity index of the SRC group was lower with eyes closed (14.08 $\pm$ 0.63 versus 15.93 $\pm$ 0.52) and eyes open (10.25 $\pm$ 0.52 vs 11.80 $\pm$ 0.57) in the mediolateral direction than for the control participants ($P < .05$). Eyes-open root mean square in the mediolateral direction was greater for the SRC group (5.00 $\pm$ 0.28 mm) than the control group (4.10 $\pm$ 0.22 mm). Logistic regression models significantly predicted LEMSK only in control participants.

Conclusions: These findings may suggest that LEMSK after SRC cannot be predicted from postural-control metrics at baseline.

Key Words: mild traumatic brain injury, center of pressure, sport-related concussion, musculoskeletal injury

Key Points

- Baseline postural-control values could not predict lower extremity musculoskeletal injury in athletes with a history of sport-related concussion.
- The complexity index identified participants with a history of sport-related concussion.

Sport-related concussion (SRC) continues to be a health epidemic that affects athletes at all levels of sport activity[1] and accounts for 5% to 9% of all sport-related injuries.[2] Unfortunately, the neurologic signs and symptoms of SRC vary by injury type and can be transient or difficult to detect (or both).[1] This has created challenges for health care professionals as they attempt to diagnose and monitor the recovery of patients with SRC. Fortunately, postural control is a cardinal sign of SRC that has been studied extensively using both clinical and instrumented techniques.[3–6]

After SRC, greater postural instability is commonly observed clinically as an increased number of errors on the Balance Error Scoring System.[7] These deficits appear to return to baseline within 2 to 5 days,[8] but the value of the Balance Error Scoring System score is limited due to the high reliable change index (7 to 8 errors)[8] and the subjectivity of the scoring criteria.[6] To overcome these concerns, postural instability can be assessed using laboratory-based force-platform technology.[3–5] These deficits can be expressed as increases in anteroposterior (AP) center-of-pressure (CoP) sway or velocity during various

visual conditions[4,5,9] or as increases in CoP regularity.[3] Although CoP measures are limited in clinical settings, they represent an indirect indication of the health of the neurologic system.[10] Furthermore, CoP measures are sensitive to lingering impairments of the postural-control system that may last up to a year after the injury.[11–13] However, these lingering postural-control impairments are not typically considered when clinical decisions are made or postconcussion rehabilitation protocols are implemented. Thus, the consequences of lingering postural-control deficits have not been studied.

Current evidence[14–19] suggests that lower extremity musculoskeletal (LEMSK) injuries are more prevalent among those with an SRC history. Specifically, within the first year after SRC recovery, affected athletes are 1.9 to 3.5 times more likely to sustain an acute lower extremity (LE) injury.[14] Unfortunately, the underlying causes of the increased LE risk have yet to be fully explored, but recent researchers[16] suggested that lingering postural-control instabilities could play a role in the increased LE injury rates.

Among the investigations that demonstrated lingering postural-control deficits after SRC during quiet upright stance, only increases in regularity or reductions in complexity of the CoP signal were observed during the Sensory Organization Test.[11,12] Sosnoff et al[11] noted increased regularity, as measured by approximate entropy, in National Collegiate Athletic Association (NCAA) Division I athletes who sustained an SRC at least 6 months earlier. Supporting this research, Schmidt et al[12] found increased regularity, measured via sample entropy and a reduction in complexity (*multiscale entropy*) in former high school football players who had a history of 2 or more SRCs. These nonlinear CoP measures may provide greater insight into the health or adaptability of the postural-control system[20] whereby increases in regularity or reductions in complexity may characterize a more constrained system that has less capacity to adapt to given strategies or tasks.[21–23]

Thus, it is possible that the increased postural-control regularity and reduced capacity of the complex network interactions involved in the regulation of physiological function (complexity) among those with a history of SRC[11,12] could result in less capacity to adapt to sport-specific tasks. This maladaptive postural-control strategy could place athletes at risk of LEMSK injury after a full clinical recovery from SRC. Prior authors[16,19] have speculated about this relationship, but it has yet to be explored.

Therefore, the purpose of our study was to prospectively investigate the relationship between baseline postural-control metrics and LEMSK injury incidence in NCAA Division I student-athletes with a history of SRC. We hypothesized that a history of SRC would be associated with LE injury. Additionally, we proposed that postural-control complexity would distinguish between those with and those without a history of SRC before the occurrence of an LE injury, whereas linear measures would not.

METHODS

Participants

A total of 84 student-athletes agreed to participate in the study. They were divided into 2 groups (n = 42 each): (1) history of concussion (SRC group; females = 11, males = 31, age = 19 ± 1 years, height = 178.6 ± 10.7 cm, weight = 81.8 ± 18.1 kg, mean number of concussions = 3 ± 1) and (2) no history of concussion (control group; females = 11, males = 31, age = 18 ± 1 years, height = 177.8 ± 11.2 cm, weight = 82.1 ± 19.1 kg). The history of medically diagnosed concussions and the number of concussions were self-reported. This resulted in 42 participants who were Division I athletes between the ages of 18 and 25 years who were currently competing in a university-sanctioned sport and had a history of SRC. The other 42 participants were perfectly matched by sport (type and position) and closely matched by age, height, weight, and sex; these control student-athletes did not have a self-reported history of concussion. For the SRC group, we calculated the time between the baseline assessment and the last concussion and categorized it as <6 months (n = 7), 6–12 months (n = 9), or >12 months (n = 26).

Table 1. Locations and Types of Musculoskeletal Lower Extremity Injuries Sustained[a]

	Group	
Injury	Sport-Related Concussion (n = 15)	Control (n = 8)
Location		
Foot	0	1
Ankle	7	3
Knee	4	1
Thigh	2	2
Hip	2	1
Type		
Strain	4	2
Sprain	9	6
Fracture	1	0
Cartilage tear	1	0

[a] The association between concussion history and injury incidence was significant ($P = .043$), which resulted in a relative risk ratio of 1.88 for lower extremity injury in the concussion group.

Student-athletes were included in the study if they had (1) complete and available paper or digital medical records and (2) a history of medically diagnosed concussion for the SRC group and no history of concussion for the control group. A concussion history was determined by reviewing the medical records for documented concussion(s) at the athlete's university along with self-report as far back as the athlete could remember. Student-athletes were excluded if they had any self-reported vestibular, metabolic, or neurologic condition (excluding concussion); chronic injury (that may have caused time loss from sport participation ≥3 months); or a preexisting condition, such as chronic ankle instability or severe lower extremity injury, that permanently affected the ability to perform upright static stance.

All procedures of this study were approved by the Georgia Southern University institutional review board, and all student-athletes signed an informed consent before participating in the research.

Procedures

Injury Surveillance. After a single athletic season, we reviewed each student-athlete's university medical charts.

These charts were reviewed for all recorded lower extremity injuries that were sustained within 1 year of the preparticipation baseline examination. A full year was evaluated, as it provided a picture of all portions of the athletic season (ie, preseason, in season, postseason, and off-season). Prior injury-history data beyond documented SRC was available at the time of data collection.

An *LE injury* was defined as a soft tissue injury (strain or sprain) or a fracture to the hip, groin, thigh, knee, lower leg, ankle, or foot area. An *acute injury* was further defined as a muscle strain, ligament sprain, or noncontact fracture or dislocation of the foot, ankle or lower leg, knee, thigh, or hip complex that occurred during a sport-related activity. A *chronic injury* was defined as a stress fracture, bursitis, or tendinitis of the foot, ankle, lower leg, knee, thigh, or hip complex. Contusions, abrasions, and lacerations were not recorded in the injury-surveillance record. Total numbers of each acute injury (total incidence) and injury type were further recorded and analyzed. Chronic injuries were noted solely for exclusion criteria and were not analyzed. Exposures were not recorded or analyzed as this information was not available at the time of testing. Thus, only the total incidence of acute LE injuries was analyzed.

Baseline Postural Control. All student-athletes performed 3 trials of eyes-open and eyes-closed upright quiet stance on a 40- × 60-cm force platform (model OR-6 series; Advanced Mechanical Technology, Inc, Watertown, MA) during preseason baseline physical examinations. The participants were asked to stand with feet together while fixating on a target that was 1.40 m away during the eyes-open task. During the eyes-closed task, to allow for postural adjustment, the trial did not start until 5 seconds after the participant closed his or her eyes. Each trial lasted 30 seconds. Any extraneous movement, such as moving the head, sneezing, or suddenly moving the arms or legs resulted in an unsuccessful trial, and the trial was repeated. Data from 10 trials were collected.

Data Analysis

The AP and mediolateral (ML) CoP data were exported via the Vicon Motion Capture System (model Nexus 1.8.5; Oxford, UK) into Excel (version 16.0.4949.1000; Microsoft Corp, Redmond, WA) for further analysis. We used custom software (MATLAB 2017; The MathWorks, Inc, Natick, MA) to apply a fourth-order, zero-phase, low-pass Butterworth filter with a cutoff frequency of 10 Hz to the raw CoP data. The root mean square (RMS) was used to calculate CoP excursions with a 2–data-point window and a 10–data-point overlap from the filtered CoP data. Mean excursion velocity (MEV) was also determined from the filtered CoP data. Both RMS and MEV were calculated as described by Prieto et al.[24]

From the unfiltered CoP data, we obtained the complexity index (CI) via the multiscale entropy[22] score by performing sample entropy (SampEn) across consecutive coarse-grain time series, corresponding to a scale factor τ.[25] The SampEn was calculated with the dimension = 2 and the tolerance range = 0.15.[25] The coarse-grain time series was determined using the following equation (Equation 1):

$$y_j^{(\tau)} = 1/\tau \sum_{i=(l-1)\tau+1}^{j\tau} x_i$$

where τ is the scale factor and j is $1 \leq j \leq N/\tau$. The length of each coarse-grain time series is N/τ. Previous authors[22] noted that approximately 600 data points were required for consistent SampEn outcomes. Therefore, we performed SampEn at 50 τ, with the final τ being 600 data points (30 000/50 = 600). Finally, the CI was calculated as the integrated numeric summation of the SampEn versus τ curve.[22] All CoP trials were averaged to obtain an aggregate value for all measures used.

Statistical Analysis

We examined all data from the force platform to ensure that they sufficiently conformed to a normal distribution without influential skewness. Descriptive statistics were generated for participants; 4 separate factorial analysis of variance (ANOVA) models (2 × 2 ANOVAs) were used to examine differences between groups (history of concussion versus no history of concussion) based on eyes open or eyes closed for each force-platform variable (RMS, MEV, and CI) in the 2 directions (ML, AP). Groups were compared in the eyes-open and eyes-closed conditions on all 3 variables in the ML and AP directions. In the event of significant interactions, the simple main effects for direction were assessed using independent *t* tests. Similar factorial ANOVA models were developed only for those participants with SRC to determine if those who sustained any injury were different from those who were injury free on all metrics in each direction, with eyes open and eyes closed, using injury status (*yes* or *no*) and force-plate metrics (RMS, MEV, and CI). We performed χ^2 tests to determine the association of the risk of LE injury and concussion history, including an examination of concussion incidence, injury incidence, and sex. Lastly, 2 logistic regression models were assessed to determine if any postural-control metrics could predict LEMSK (*yes* or *no*) within each group (SRC and control). Significance was set at $P < .05$ for all analyses.

RESULTS

Over the course of the season, 27.4% of the student-athletes had a documented injury (SRC group: acute injury = 15, no injury = 27; control group: acute injury = 8, no injury = 34). Sprains and strains of the ankle and thigh were the most common injury types and locations (Table 1). The association between concussion history and injury incidence was significant ($\chi^2_1 = 2.93$, $P = .043$) and resulted in a relative risk of 1.88 (95% confidence interval = 1.09, 3.95) for LE injury, given a history of concussion. A participant's sex did not influence this association ($P = .989$), and the number of concussions was not associated with the injury rate or incidence ($P = .791$).

Eyes-Closed Condition

With the eyes closed, the CI in the ML direction was significantly less ($F_{1,82} = 5.15$, $P = .026$) in the SRC group (14.08 ± 0.63) than in the control group (15.93 ± 0.52; Cohen d = 3.2; Table 2). Neither eyes-closed MEV nor mean RMS in the ML direction were different (*P* values >

Table 2. Baseline Center-of-Pressure Force-Platform Measures: Eyes Closed

Direction	Measure	Group, Mean ± SD	
		Sport-Related Concussion	Control
Mediolateral	Multiscale entropy, SE[a]	14.08 ± 4.08	15.93 ± 3.39
	Mean excursion velocity, mm/s	12.1 ± 4.00	12.9 ± 3.00
	Root mean square, mm	5.10 ± 1.00	4.70 ± 1.00
Anteroposterior	Multiscale entropy, SE	13.41 ± 3.47	14.52 ± 2.71
	Mean excursion velocity, mm/s	11.5 ± 3.00	12.8 ± 4.00
	Root mean square, mm	5.00 ± 1.00	5.00 ± 1.00

[a] Difference between groups ($P < .05$).

.05). Further, none of the eyes-closed postural metrics in the AP direction were different between the groups (P values $> .05$).

Eyes-Open Condition

Both the CI ($F_{1,82} = 3.99$, $P = .049$) and RMS ($F_{1,82} = 5.55$, $P = .021$) in the ML direction were different between the groups (Table 3). For the CI, mean values for the SRC group (10.25 ± 0.52) were lower than those for the control group (11.80 ± 0.57; Cohen d = 2.8), whereas RMS mean values were greater for the SRC group (5.00 ± 1.00 mm) compared with the control group (4.10 ± 1.00 mm; Cohen d = 0.91). Neither the MEV nor any of the AP-direction variables were different between groups ($P > .05$).

Logistic Regression

When predicting LEMSK, the logistic regression model for the SRC group was not significant ($P = .068$), whereas the model for the control group was significant ($P = .002$). This latter model explained 43% of the variance in LEMSK and correctly classified 83.3% of the athletes in whom eyes-closed CI in the ML direction was the only postural variable that significantly contributed to the model. These results indicate that an increase in CI in the ML direction with the eyes closed was associated with a 1.6 times (95% confidence interval = 1.019, 2.639) increased risk of LEMSK in the control group.

DISCUSSION

The purpose of our study was to investigate the relationship between baseline postural-control metrics and prospective LEMSK injury incidence in NCAA Division I student-athletes with a history of SRC. The most important findings were that baseline static upright-stance postural-control metrics were not related and did not predict LEMSK injury incidence among those with a history of SRC. Similarly, only a single postural-control metric (CI with eyes closed in the ML direction) predicted LEMSK injury before injury occurrence. Lastly, a history of SRC could be detected using the CI, and a history of SRC was associated with a 1.88 times elevated risk of LEMSK injury. In agreement with prior research,[14–19] a history of SRC was associated with an increased LE injury risk; however, baseline postural-control metrics did not predict LEMSK injury before injury occurrence among those with a history of SRC. This may suggest that postural-control metrics do not play a role in predicting LEMSK injury incidence after full clinical recovery from SRC or that quiet upright stance is not sufficiently challenging to detect prospective LEMSK.

We hypothesized that LEMSK injury would be associated with a history of SRC. This hypothesis was correct as those with a history of SRC had a 1.88 times greater likelihood of incurring an LEMSK injury compared with the control group (Table 1). These data support earlier findings[14,15] that SRC was associated with an increased risk of LE injury. Furthermore, our result closely matched that of previous authors[14] (relative risk ratio = 1.88 versus 1.97) and thus supports the growing body of literature indicating that a history of SRC is associated with LE injury. Although information about prior LEMSK injuries was not available at the time of data collection for the current study, previous LEMSK injuries appeared to be the most common factor associated with future LEMSK injuries.[26] Thus, the increase in injury rates for the SRC group could be explained by previous injury and other contributing factors, such as sex[26] or sport position. Future researchers should include previous and prospective LEMSK injury data.

We further proposed that nonlinear measures (ie, CI) would distinguish between those with and those without a history of SRC, whereas linear measures (RMS, MEV) would not. Our findings partially support this hypothesis as CI and RMS in the ML direction distinguished between those with and those without a history of SRC. No other differences were noted between groups for the AP direction

Table 3. Baseline Center-of-Pressure Force-Platform Measures: Eyes Open

Direction	Measure	Group, Mean ± SD	
		Sport-Related Concussion	Control
Mediolateral	Multiscale entropy, SE[a]	10.25 ± 3.35	11.80 ± 3.71
	Mean excursion velocity, mm/s	7.3 ± 2.00	7.3 ± 2.00
	Root mean square, mm[a]	5.00 ± 1.00	4.10 ± 1.00
Anteroposterior	Multiscale entropy, SE	12.42 ± 3.43	12.64 ± 3.11
	Mean excursion velocity, mm/s	7.6 ± 2.00	7.4 ± 2.00
	Root mean square, mm	3.6 ± 1.00	3.4 ± 1.00

[a] Difference between groups ($P < .05$).

or MEV. These findings are in agreement with earlier investigations[11,12] and indicate that the SRC group had an overall loss of complexity in the ML direction under both visual conditions, along with greater postural sway in the ML direction during the eyes-open condition. The loss of complexity could be related to either impaired ability or an adaptation in the motor program to maintain upright stance.[27]

Greater postural sway is commonly observed with neurologic disorders, specifically SRC,[3–5] yet this mainly presents in the AP direction and when testing velocity metrics. Our MEV values closely matched those of healthy control participants in prior work in both the AP and ML directions regardless of visual condition[4] and were slightly higher than normative data.[24] The RMS values in our study were smaller for the healthy control group in the eyes-open ML direction than in earlier research[4] (5 mm versus approximately 7 mm). These differences could be attributed to the lack of a filter as reported by Powers et al[4] in their CoP analysis, which could have inflated the total RMS values and could account for the difference we noted. However, a total 1-mm change between groups in the current study is not considered a clinically meaningful change despite the large effect size (Cohen $d = 0.91$).

Although the student-athletes were not clinically impaired at the time of testing, the greater sway in the ML direction could indicate a different strategy to maintain upright quiet stance[27] rather than a pathologic state. Coupled with the loss of complexity, the SRC group may have adopted a hip and ankle strategy to maintain upright stance.[3,9,28] This is not surprising as lingering postural-control deficits have been observed among those with a history of SRC, specifically in the ML direction.[11–13] In the presence of lingering postural-control deficits, athletes may rely on a feed-forward rather than a feedback control of posture or a combination of both.[28] This may help to explain the lack of differences in the AP direction. After an SRC, a combination of hip and ankle strategies are used to maintain upright stance, with the majority of the CoP differences occurring in the AP direction due to a reliance on the ankle plantar flexors and dorsiflexors to control the human inverted pendulum.[4] The lack of any significant finding in the AP direction follows a trend of prior researchers[11,12] and may provide insight into a varying postural strategy that those with a history of SRC use to maintain upright stance.

Athletes who trained using unique protocols to meet the demands of their sport (eg, surfers versus basketball players) demonstrated this ML postural strategy.[29] Athletes competing on an unstable surface (eg, surfers) tend to use direction-specific proximal-to-distal control of posture, whereas athletes performing on a stable surface use a traditional distal-to-proximal approach to control quiet stance. Neither strategy is incorrect; both strategies simply reflect the body's response to the training stimulus. Therefore, the presence of lingering postural deficits may have caused an adaptation in the stability of the motor program for quiet upright stance, which resulted in greater RMS and a loss of complexity. However, without a more challenging stimulus (eg, a translating force platform or a dynamic task), these suggestions are speculative.

Movement complexity is a fundamental inherent characteristic of all human functions.[20] Reductions in complexity may characterize a more constrained system that has less capacity to adapt to given strategies or tasks.[20–23] For example, as physiological function declines, such as during the natural aging process or in the presence of a compromised neurologic system, interactions among the elements (ie, sensory systems) within the human body deteriorate and may result in a loss of complexity.[21,22] We demonstrated a loss of complexity in the SRC group under both visual conditions in the ML direction. These data support prior observations[11,12] that those with a history of SRC displayed more regularity as measured by Approximate Entropy (ApEn) and SampEn during the Sensory Organization Test. Although we did not directly report ApEn or SampEn, CI is the integrated summation of the SampEn versus τ curve. This allows for an analysis of the complexity of physiological outputs across multiple time scales, whereas ApEn and SampEn measure regularity on a single time scale.[22] In short, multiscale entropy CI is a global approach to measuring the complexity of all physiological systems that influence the postural-control system.[23] A loss of complexity could be related to an impaired or altered state in the central nervous system or an adaptation in the motor program for certain tasks or both.

CLINICAL IMPLICATIONS

These results have 2 major clinical implications. First, CI was able to discern those with a history of SRC from those without a history of SRC at baseline. For clinical facilities with access to embedded or mobile force platforms, CI may aid clinicians in differentiating those with a history of SRC. Second, CI with eyes closed in the ML direction successfully predicted LEMSK in those without a history of SRC. This finding may suggest that those without a history of SRC who have higher CI values (ie, more irregularity and complexity) at baseline may have a higher risk of incurring LEMSK in the upcoming athletic season. If a system becomes too complex, 1 or more physiological processes may "freeze up" and lose functionality.[30] For example, if an in-season NCAA Division I track athlete is actively training for the 100-m dash but suddenly decides to play a full soccer match as a defending midfielder before the conference final, the training for the dash may have been insufficient for adequate performance during the soccer match and the influence of the multiplanar movements introduced to the athlete via the soccer match may subsequently decrease performance on the athlete's 100-m dash. Thus, if a system has to adapt to multiple environments, 1 of the environments may counteract the effect of another environment, resulting in a maladaptation to the first environment to which the athlete had previously adapted. This phenomenon may lead to a decrease in overall functionality because of too much complexity within the system. This has been observed in older adults in whom too much step-width variability was associated with a prior fall.[20] Thus, clinicians may be able to use CI as a screening tool to identify student-athletes who need rehabilitation before the athletic season. Protocols that have shown promise in reducing LEMSK injuries, such as the Fédération Internationale de Football Association (FIFA) 11,[31] could be viable options for reducing the excessive variability in the motor system.

LIMITATIONS

No study is without limitations, and our study did have limitations that should be addressed. First, the lack of exposure rates may have limited our ability to generate more robust regression models and identify potentially relevant associations between postural-control metrics and the LE injury risk in the SRC group. However, this study was sufficiently powered for the ANOVA. Future researchers should include exposure rates, along with a larger sample size, to further examine this association, yet it is challenging to find existing student-athletes with or without an SRC history while prospectively tracking injury occurrence. Second, student-athletes may not have been completely truthful regarding their history of SRC or this information may not have been properly recorded by the sports medicine staff. Third, the chart review was performed systematically, but some of the data may have been entered incorrectly, thereby potentially altering the injury information. Fourth, the majority of the SRC group had a prior diagnosed SRC >12 months earlier. Because this characterized most of the sample, the overall results of the study may have been influenced, even though the postural-control data were parametric. Fifth, no prior LEMSK injury data were available beyond the existing prospective single-year analysis. A history of injury is commonly accepted as the leading factor associated with prospective injury. Thus, future investigators should inquire about the athletes' history of LEMSK injury. Lastly, quiet upright stance may not be challenging enough for student-athlete populations. Future researchers should examine how more difficult sport-like tasks influence postural control and the LEMSK injury risk.

Overall, our results indicate that baseline static upright-stance postural-control metrics did not relate to or predict LEMSK injury incidence among those with a history of SRC. However, CI was able to successfully identify those with a history of SRC and predict those without a history of SRC who might have been at a greater risk of LEMSK at baseline. Thus, postural-control complexity may play a role in determining the LEMSK injury risk, yet more work is needed to explore this potential association.

ACKNOWLEDGMENTS

This study was partially funded by the University of Nevada, Reno Neuroscience Institute Pilot Study Program (COBRE P20GM103650) and the Nevada IDeA Network of Biomedical Research Excellence pilot program (P20GM103440; U54GM104944).

REFERENCES

1. McCrory P, Meeuwisse W, Dvorak J, et al. Consensus statement on concussion in sport-the 5th International Conference on Concussion in Sport held in Berlin, October 2016. *Br J Sports Med*. 2017;51(11):838–847.
2. Meehan WP III, d'Hemecourt P, Comstock RD. High school concussions in the 2008-2009 academic year: mechanism, symptoms, and management. *Am J Sports Med*. 2010;38(12):2405–2409.
3. Cavanaugh JT, Guskiewicz KM, Giuliani C, Marshall S, Mercer VS, Stergiou N. Recovery of postural control after cerebral concussion: new insights using approximate entropy. *J Athl Train*. 2006;41(3):305–313.
4. Powers KC, Kalmar JM, Cinelli ME. Recovery of static stability following a concussion. *Gait Posture*. 2014;39(1):611–614.
5. Guskiewicz KM, Ross SE, Marshall SW. Postural stability and neuropsychological deficits after concussion in collegiate athletes. *J Athl Train*. 2001;36(3):263–273.
6. Murray N, Salvatore A, Powell D, Reed-Jones R. Reliability and validity evidence of multiple balance assessments in athletes with a concussion. *J Athl Train*. 2014;49(4):540–549.
7. Riemann BL, Guskiewicz KM. Effects of mild head injury on postural stability as measured through clinical balance testing. *J Athl Train*. 2000;35(1):19–25.
8. McCrea M, Guskiewicz KM, Marshall SW, et al. Acute effects and recovery time following concussion in collegiate football players: the NCAA Concussion Study. *JAMA*. 2003;290(19):2556–2563.
9. Murray NG, Szekely B, Moran R, et al. Concussion history associated with increased postural control deficits after subsequent injury. *Physiol Meas*. 2019;40(2):024001.
10. Degani AM, Santos MM, Leonard CT, et al. The effects of mild traumatic brain injury on postural control. *Brain Inj*. 2017;31(1):49–56.
11. Sosnoff JJ, Broglio SP, Shin S, Ferrara MS. Previous mild traumatic brain injury and postural-control dynamics. *J Athl Train*. 2011;46(1):85–91.
12. Schmidt JD, Terry DP, Ko J, Newell KM, Miller LS. Balance regularity among former high school football players with or without a history of concussion. *J Athl Train*. 2018;53(2):109–114.
13. Buckley TA, Oldham JR, Caccese JB. Postural control deficits identify lingering post-concussion neurological deficits. *J Sport Health Sci*. 2016;5(1):61–69.
14. Lynall RC, Mauntel TC, Padua DA, Mihalik JP. Acute lower extremity injury rates increase after concussion in college athletes. *Med Sci Sports Exerc*. 2015;47(12):2487–2492.
15. Fino PC, Becker LN, Fino NF, Griesemer B, Goforth M, Brolinson PG. Effects of recent concussion and injury history on instantaneous relative risk of lower extremity injury in Division I collegiate athletes. *Clin J Sport Med*. 2019;29(3):218–223.
16. Howell DR, Buckley TA, Lynall RC, Meehan WP III. Worsening dual-task gait costs after concussion and their association with subsequent sport-related injury. *J Neurotrauma*. 2018;35(14):1630–1636.
17. Houston MN, Hoch JM, Cameron KL, Abt JP, Peck KY, Hoch MC. Sex and number of concussions influence the association between concussion and musculoskeletal injury history in collegiate athletes. *Brain Inj*. 2018;32(11):1353–1358.
18. Gilbert FC, Burdette GT, Joyner AB, Llewellyn TA, Buckley TA. Association between concussion and lower extremity injuries in collegiate athletes. *Sports Health*. 2016;8(6):561–567.
19. Herman DC, Jones D, Harrison A, et al. Concussion may increase the risk of subsequent lower extremity musculoskeletal injury in collegiate athletes. *Sports Med*. 2017;47(5):1003–1010.
20. Stergiou N, Decker LM. Human movement variability, nonlinear dynamics, and pathology: is there a connection? *Hum Mov Sci*. 2011;30(5):869–888.
21. Powell DW, Blackmore SE, Puppa M, et al. Deep brain stimulation enhances movement complexity during gait in individuals with Parkinson's disease [epub ahead of print]. *Neurosci Lett.* doi: 10.1016/j.neulet.2018.05.010.
22. Busa MA, van Emmerik REA. Multiscale entropy: a tool for understanding the complexity of postural control. *J Sport Health Sci*. 2016;5(1):44–51.
23. Lipsitz LA, Goldberger AL. Loss of "complexity" and aging: potential applications of fractals and chaos theory to senescence. *JAMA*. 1992;267(13):1806–1809.
24. Prieto TE, Myklebust JB, Hoffmann RG, Lovett EG, Myklebust BM. Measures of postural steadiness: differences between healthy young and elderly adults. *IEEE Trans Biomed Eng*. 1996;43(9):956–966.

25. Costa M, Goldberger AL, Peng CK. Multiscale entropy analysis of biological signals. *Phys Rev E Stat Nonlin Soft Matter Phys*. 2005;71(2 pt 1):021906.
26. Engebretsen AH, Myklebust G, Holme I, Engebretsen L, Bahr R. Intrinsic risk factors for acute knee injuries among male football players: a prospective cohort study. *Scand J Med Sci Sports*. 2011;21(5):645–652.
27. Williams DSB III, Murray NG, Powell DW. Athletes who train on unstable compared to stable surfaces exhibit unique postural control strategies in response to balance perturbations. *J Sport Health Sci*. 2016;5(1):70–76.
28. Winter DA. Human balance and posture control during standing and walking. *Gait Posture*. 1995;3(4):193–214.
29. Powell DW, Williams DSB III. Athletes trained using stable compared to unstable surfaces exhibit distinct postural control profiles when assessed by traditional and nonlinear measures. *Hum Mov Sci*. 2015;44:73–80.
30. Lipsitz LA. Dynamics of stability: the physiologic basis of functional health and frailty. *J Gerontol A Biol Sci Med Sci*. 2002;57(3):B115–B125.
31. Bizzini M, Junge A, Dvorak J. Implementation of the FIFA 11+ football warm up program: how to approach and convince the Football associations to invest in prevention. *Br J Sports Med* 2013;47(12):803–806.

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