THE RELATIONSHIP BETWEEN CONCUSSION FACTORS AND LOWER EXTREMITY INJURY RATES IN COLLEGIATE ATHLETES

by

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LIST OF ABBREVIATIONS

SRC	Sport Related Concussion
SA	Student Athlete
MSK	Musculoskeletal
LE	Lower Extremity
UE	Upper Extremity
GM	General Medical
NC	Non-contact
SAC	Standardized Assessment of Concussion
GSC	Graded Symptom Checklist
BESS	Balance Error Scoring System
ImPACT	Immediate Post-Concussion Assessment and Cognitive Testing
CRT	Clinical Reaction Time
KD	King Devick Test
TG	Tandem Gait
LOC	Loss of Consciousness
PTA	Post Traumatic Amnesia

ABSTRACT

Background: Athletes with concussions have been shown to have functional postural control and dual tasking deficits long after the traditionally established timeline of clinical recovery. Concern has recently grown over the potential consequences of these deficiencies, and whether or not they adversely impact an athlete's future risk of injury. **Purpose:** The purpose of this study was twofold: to retrospectively identify a relationship between sport related concussion (SRC) and subsequent lower extremity (LE) musculoskeletal (MSK) injury in collegiate student-athletes (SA), and identify if concussion presentation characteristics detected through clinical concussion tests at baseline and return to play (RTP) predicted subsequent MSK injury. **Methods:** Electronic medical records of 24 Division-1 Collegiate Athletic Association (NCAA) SA with a history of SRC were examined and compared to 27 matched control athletes 365 days prior to the date or SRC and 365 days following RTP using a Cox proportional hazard model to calculate an odds ratio for each injury type. Clinical test results were compared to MSK injury risk using a stepwise linear regression model. **Results:** Within one year of RTP, the study group was 2.95 (95% Confidence Interval (CI), 1.5, 5.78) times more likely than the control group to sustain any MSK injury, 2.09 (95% CI, 1.07, 4.06) times more likely to experience any LE injury, and 2.25 (95% CI, 1.16, 4.36) times more likely to have a LE Non-Contact (NC) injury. The group with concussion was not significantly more likely to sustain a time-loss LE injury (1.86, 95% CI, 0.96, 3.61), a contact LE injury (1.56, 95% CI 0.81, 3.03), a LE overuse injury (1.55, 95% CI, 0.8, 3.02), upper

extremity injury (1.67, 95% CI 0.86, 3.25) or general medical complaint (1.78, 95% CI 0.92, 3.46). No relationship was found between increased incidence of MSK injury and any analyzed component of the institutional concussion test battery at baseline or RTP, which included GSC, SAC, BESS, ImPACT, KD, CRT, and TG. **Conclusion:** These preliminary results suggest SA were at an increased risk of MSK, LE, and LE NC injury in the year following a SRC event, and the current clinical test battery for concussion has no value in predicting this risk of injury. Further research is needed to examine the of clinical tests to detect underlying deficits that may be related to increased MSK injury risk.

Chapter 1

INTRODUCTION

Due to potential long term neurological complications following the injury, concussions have become a focal point of many in the sports medicine field.[1] Although no definition has been universally adopted across medical and athletic governing bodies, in their 5th Consensus Statement the International Conference on Concussion In Sport (5th CIS) describes a sport-related concussion (SRC) as "a traumatic brain injury induced by biomechanical forces."[1] Due to the heterogeneous nature of concussion, a battery of assessment tools including cognitive, balance, visual, and self-reported symptoms have been developed to evaluate this injury.[1] (Appendix A, Table 1)

Although the current battery of concussion testing is highly sensitive acutely post-concussion, the sensitivity declines to a clinically insignificant level after only seven days, indicating limited utility in their diagnostic role in concussion recovery.[2] Classically, concussions have been described to have a relatively short recovery period with as many as 90% of athletes returning to activity within 7 to 10 days, however more recent research addressing multiple domains of concussion indicates full recovery may take as long as two months.[1,3] This suggests physiological recovery far outlasts clinical recovery, and that clinicians may be returning SA to activity despite incomplete physiological recovery.[4]

Factors such as gender, reported symptoms, and re-emergence of symptoms have been found to predict prolonged recovery from SRC, however the specific

mechanism of why these factors influence recovery is poorly understood.[5] College-age females have been found to report more symptoms and perform worse on vestibular, occulomotor, and neurocognitive testing than their male counterparts following SRC.[6–8] Independent of gender, increased symptom burden has been associated with prolonged recovery (longer than 28 days) from SRC.[9] Self-reported balance deficits, dizziness, and fogginess have been associated with delayed recovery from SRC, although the underlying cause of this relationship is unknown at this time.[10,11] LOC has traditionally been associated with worse outcomes following SRC, but this theory has mixed support in current literature and no studies exist relating LOC to long term postural control deficits.[5,12] PTA has been linked to greater deficits following SRC as well as delayed recovery in measures of symptoms, cognition, and balance, but consistent support in literature for PTA as a predictor of poor outcome is lacking.[5,13] At this time, it is unknown what immediate characteristics of SRC may specifically predispose an athlete to well-documented motor control deficits following SRC.

Detections in acute and chronic gait alterations suggest athletes continue to have functional deficits long after they are cleared for RTP. Altered gait termination strategies and increased postural sway, resulting in a more conservative gait strategy, have been documented in patients acutely post-concussion by numerous studies over the past decade.[14,15] SA with concussion display lower gait speed and display more conservative movement variations of their center of mass in the first 48 hours after a concussive injury, but improvements have been observed within two weeks of injury.[16] In contrast, a separate investigation noted persistent alterations in gait stability and speed as long as four weeks post-injury, while alterations in gait strategy have been documented for as long as 2 months post-concussion.[17–20] These findings

suggest inherent limitations within the current clinical concussion testing battery, particularly with regard to motor control and dual-tasking challenges. Poor neuromuscular control has previously been implemented in LE MSK injury, suggesting these deficits may have larger consequences in an athletic population.[21] Given the complex cognitive and physical demands placed on athletes, the current assessment and RTP standard may be falling short of identifying athletes who have not yet returned to normal thresholds of motor control.[22,23] These studies have consistently documented the need for further examination of functional consequences of these deficits.[18]

Emerging retrospective evidence links a SA's history of SRC with increased rates of lower extremity musculoskeletal injury following both diagnosed and suspected concussions.[24–26] Although there is not enough evidence to predict an individual's risk of future injury, this positive association between SRC and injury has been well documented in collegiate and professional athletes who have sustained at least one SRC.[27,28] SA who sustain a SRC are 2.10 to 3.39 times more likely than their non-recently concussed peers to sustain a LE injury in the 90 days following clearance from their concussive injury.[24,25,29,30] SA continue to be at a higher risk of injury as long as one year following full clearance from SRC, and have been reported to be 1.64 times more likely to sustain a LE acute injury than their non-concussed peers, and 1.97 times more likely after a SRC than before.[24] Higher incidences of lateral ankle sprains ($P= 0.012$), knee injuries ($P=0.031$), and lower extremity muscle strains ($P= 0.003$) were significantly associated with self-reported SRC compared to athletes who did not report a history of SRC over the course of a year as well.[26] Lower extremity MSK injury prior to SRC has been linked to increased risk of SRC, but current literature has

not consistently supported this relationship.[24,25,27] The NCAA Injury Surveillance Program reports a moderate time-loss (inability to participate in organized athletic activity for one or more days) injury rate of 13.79 per 1,000 game exposures and 3.98 per 1,000 practice exposures, however the implications of these injuries vary in clinical significance and may range from a mild sprain to a season-ending ligament tear.[31] A history of SRC has recently been associated with higher prevalence of osteoarthritis in retired professional athletes, suggesting underlying effects of SRC may be detrimental to long term health outcomes long after athletic participation has ended.[32]

With over 10,000 concussions sustained by collegiate athletes every year, the implications of a relationship between SRC and lower extremity MSK injury are significant.[33] Additionally, understanding predictors of injury could improve diagnostic procedures currently implemented by health care providers to prevent premature RTP clearance, potentially reducing future MSK injury risk. The purpose of this study is to examine the relationship between SRC and subsequent MSK injury risk, and identify whether or not characteristics of SRC presentation detected through clinical concussion tests at pre-season baseline and return to play predicted subsequent MSK injury. We hypothesize SRC will be associated with a higher rate of subsequent LE MSK injury, and that prolonged deficits on symptom scores and measures of balance and motor control assessed using a common concussion testing battery will also be associated with increased risk of MSK injury.

Chapter 2

METHODS

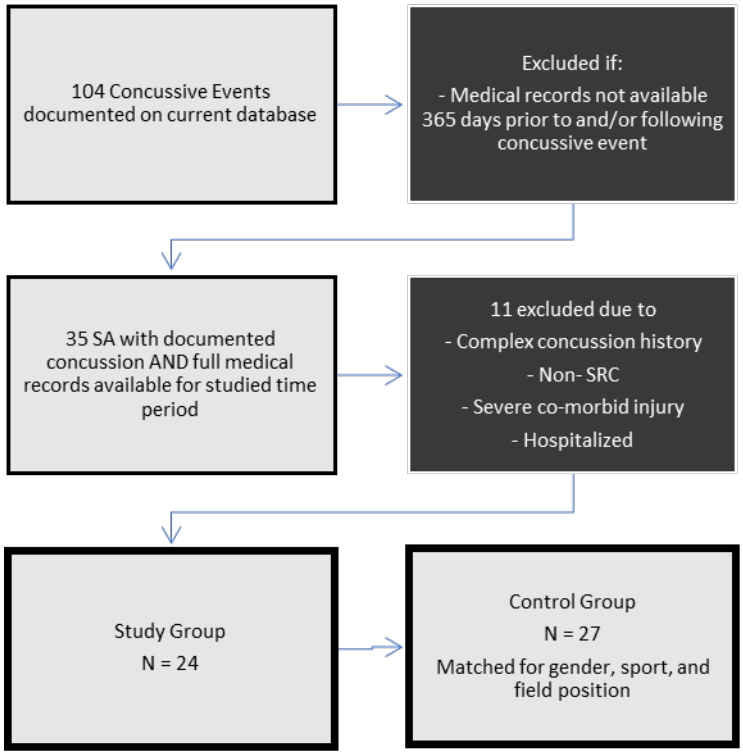
2.1 Research Design

A retrospective cohort design was used to identify a relationship between sport related concussion (SRC) and subsequent lower extremity (LE) musculoskeletal (MSK) injury in collegiate student-athletes (SA), and identify if concussion presentation characteristics detected through clinical concussion tests at baseline and return to play (RTP) predicted subsequent MSK injury.

Fifty-one NCAA D1 athletes were subjects in this study. The study group (CONC) consisted of Twenty-Four SA who were diagnosed with a concussion by a team physician and followed a return-to-play protocol implemented by certified athletic trainers (AT) before receiving written clearance to return to sport from a physician. Each of the selected SA sustained a SRC during organized athletic activity between August 1, 2015 and March 1, 2017 and were active on their roster for 365 days before and after their concussion. SA were paired with controls (Non-CONC) with no history of concussion during their collegiate career, matching for gender, sport, and position group when possible. In one instance, a sport-matched control was not available, so the study subject was matched with someone of the same gender who had similar sport-specific risks and demands. The subject selection process is detailed in Figure 1. At least one control subject was matched to each participant with concussion using publicly available demographic information. Participants provided

written informed consent as approved by the University of Delaware Institutional Review Board of (IRB #816044-4).

Figure 1. Subject Selection Methods



2.2 Procedure

The institutional concussion policy is in line with NCAA and 5th CIS guidelines, and emphasizes the resolution of symptoms and return to baseline levels of neurocognition and balance before and during incremental increases in physical exertion and risk of physical contact (when applicable). The protocol is implemented

at multiple time points during recovery, and is used to detect transient cognitive, visual, and vestibular deficits present following a concussion, ensuring athletes are not prematurely returned to activity before these deficits have resolved.[1] The SA was supervised by a physician or AT at all points during this process. Once the SA passed through the final stage of this protocol, they received final clearance from a university physician before returning to organized athletic participation. Injury data from both groups were collected using SportsWareOnLine (Computer Sports Medicine Inc, Stoughton, Massachusetts, USA), the institutional electronic medical record system. All MSK injuries in the CONC group occurring 365 days prior to the day of their concussion event and 365 days after they returned to full athletic participation were manually recorded using Microsoft Excel. Injuries were recorded for each Non-CONC participant during the same time period as the study subject to account for potential practice and game exposure. Demographic and injury information from each group is found in Table 1.

The NCAA Injury Surveillance System defines MSK injury as one that "occurred as a result of participation in an organized intercollegiate practice or competition, and required medical attention by a team certified athletic trainer or physician, and resulted in restriction of the SA participation or performance for one or more calendar days beyond the day of injury."[34] Although time loss is a clinically significant measure of injury severity, all injuries were included for statistical analysis. Each injury event was categorized by time loss (TL) and whether the injury affected lower extremity (LE), upper extremity (UE) or general medical health (GM), as well

as by whether their mechanism of injury was contact (C), non-contact (NC), or overuse (O).

Table 1: Subject Demographic and Injury Information

	Study	**Controls**
Female, n	13	15
Male, n	11	12
Age, y, mean	20.19	18.75
Weight, kg, mean	75.57	74.96
Height, cm, mean	174.33	175.28
Pre-SRC Injuries:	45	56
Total	27	26
Lower Extremity (Total)	15	13
Non-Contact	8	5
Contact	4	8
Overuse	14	26
Upper Extremity (Total)	1	6
Non-Contact	6	12
Contact	7	8
Overuse	4	4
General Medical (Total)	5	5
Post-SRC Injuries:	54	38
Total	30	17
Lower Extremity (Total)	16	4
Non-Contact	9	9
Contact	5	4
Overuse	13	13
Upper Extremity (Total)	6	1
Non-Contact	6	7
Contact	1	5
Overuse	11	8
General Medical (Total)	10	9

2.3 Data Analysis

For the purposes of this study, a non-overuse injury is defined as one that occurred after a single action or event and resulted in any level of restricted participation at practice, while overuse injury is defined as an injury that occurred due to repetitive insults, motions or actions.[35] Both time-loss and non time-loss injuries were included in analyses not specific to time-loss. A general medical complaint was categorized as an illness, infection, or systemic complaint without MSK origins. All injury data used for this study was documented by AT's, and includes the following: mechanism of injury (contact or non-contact), location of injury (foot, ankle, shin, knee, thigh, or hip), type of injury (fracture, strain, sprain, etc), categorization of injury (acute or chronic), and days of no participation. Injury data was organized using the structure outlined in Figure 2. The independent variable used in Aim 1 was a SA's history of SRC. The dependent variables used in Aim 1 were the number and type of MSK injuries sustained by a SA after their concussive injury. The independent variables used in Aim 2 were the number of LE MSK injuries sustained by SA's following SRC. The dependent variables were the presentation of an SA's SRC quantified using scores from the baseline and post-SRC concussion battery, and rate of MSK injury.

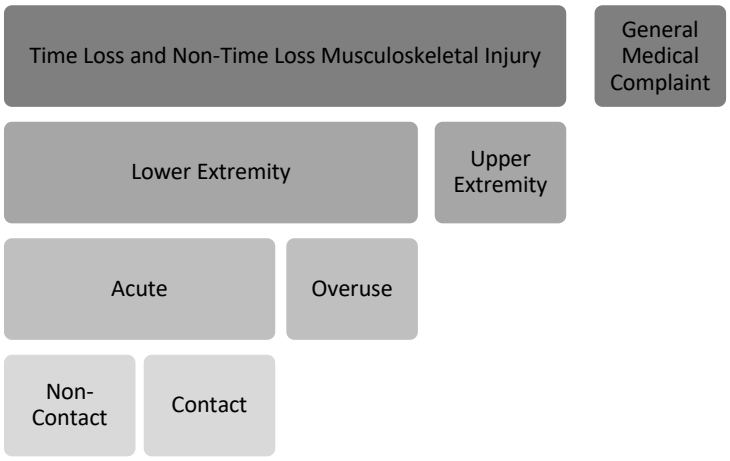


Figure 2: Injury Data Organizational Structure

2.4 Statistical Analysis

To address Aim 1, a Cox proportional hazard model was used to compare the risk of sustaining a type of MSK injury after a SRC compared to a non-concussive injury in matched controls. Multiple models were independently utilized to identify risk of LE injury, NC LE injury, contact LE injury, overuse LE injury, UE injury, GM complaint, and total MSK injury (including all LE and UE injuries). These models included both time-loss and non time-loss injuries; a separate model was used to identify the risk of a time-loss LE MSK injury. To address Aim 2, CONC group data from Aim 1 were cross referenced with change scores on various tests within the clinical concussion battery between baseline and RTP, including GSC, SAC, BESS, ImPACT, CRT, and TG using a stepwise logistic regression model to identify determinants of an individual's concussive injury that may correlate with a future higher injury risk. Pre-season baseline scores from the study and control groups were

compared using the primary study group, as well as a larger sample of available data (97 athletes with one or more SRC, 91 controls), with no differences found between any group.

Chapter 3

RESULTS

3.1 Musculoskeletal Injury Rates Following SRC

Participants with concussion were 2.95 (95% CI 1.5, 5.78, p=0.002) times more likely than the Non-CONC group to sustain a MSK injury within 365 days of clearance from SRC (Refer to Table 2 and Figure 1 in Appendix A) based on this preliminary data. These participants were 2.09 (95% CI 1.07, 4.06, p= 0.029) times more likely to sustain any type of LE MSK injury, and 2.25 (95% CI 1.16, 4.36, p= 0.016) times more likely than the control group to sustain a LE NC injury within a year of RTP. (Refer to Figures 2 and 3 in Appendix A) The CONC group were not statistically more likely to sustain a time-loss LE, LE contact, LE overuse, UE, or GM injury within 365 days of SRC clearance. No group differences were found for any injury classification in the year prior to a SRC event. (Refer to Table 2 in Appendix A)

Table 2: Group Injury Risk Within 365 Days of RTP after SRC

Injury Classification	Group	Survival Rate at 365d	Odds Ratio at 365d (95% CI)	P value
LE MSK TL	Non-CONC	0.67	1.86 (0.96,3.61)	0.064
	CONC	0.48		
Any MSK	Non-CONC	**0.47**	**2.95 (1.5, 5.78)**	**0.002**
	CONC	**0.11**		
LE	Non-CONC	**0.57**	**2.09 (1.07, 4.06)**	**0.029**
	CONC	**0.3**		
LE NC	Non-CONC	**0.81**	**2.25 (1.16, 4.36)**	**0.016**
	CONC	**0.61**		
LE C	Non-CONC	0.77	1.56 (0.81, 3.03)	0.184
	CONC	0.77		
LE O	Non-CONC	0.87	1.55 (0.8, 3.02)	0.187
	CONC	0.81		
UE	Non-CONC	0.73	1.67 (0.86, 3.25)	0.125
	CONC	0.59		
GM	Non-CONC	0.8	1.78 (0.92, 3.46)	0.084
	CONC	0.67		

LE = Lower Extremity, MSK = Musculoskeletal, TL= Time Loss, NC= Non-Contact, C= Contact, O= Overuse, UE= Upper Extremity, GM= General Medical

3.2 Determinants of SRC and Musculoskeletal Injury

There were no differences between participants who sustained concussions and those who did not at baseline for any of the dependent variables examined. (Table 3) No difference was found between baseline and RTP scores on concussion battery tests for the CONC group. (Table 4) No significant relationship was found between scores on baseline clinical battery tests and changes between baseline and RTP scores and MSK injury within 365 days of clearance. (Table 5).

Table 3: Demographics for Full Concussion Battery Group at Baseline

	Sustained Concussion	**No Concussion**
Sex Female (n) Male (n)	64 39	54 37
	Mean (±SD)	**Mean (±SD)**
Height (cm)	174.33 (± 11.94)	175.28 (± 12.48)
Mass (kg)	75.58 (± 20.21)	74.95 (± 18.58)
Age (y)	19.9 (± 1.52)	18.74 (± 1.15)
GSC	3.86 (± 8.1)	2.14 (± 2.7)
SAC	27.36 (± 1.71)	27.14 (± 1.74)
BESS	13.97 (± 7.1)	14.77 (± 6.68)
KD (s)	39.18 (± 8.09)	41.13 (± 7.23)
CRT	205.24 (± 23.95)	206.94 (± 22.3)
Tandem Gait (s)	10.31 (± 1.64)	10.37 (± 1.54)
ImPACT Motor Speed	40.46 (± 5.88)	40.03 (± 7)
ImPACT Reaction Time	0.59 (± 0.07)	0.6 (± 0.09)
ImPACT Impulse Control	6.17 (± 4.98)	5.9 (± 4.29)

Table 4: Baseline, RTP, and Change Values of Concussion Battery for CONC Group

Clinical Concussion Battery Component	Mean Baseline (+ SD)	Mean RTP (+ SD)	Mean Base-RTP Change Score (95% CI)
GSC	4.09 ± 9.83	0.61 ± 1.3	4.64 (-1.26, 10.56)
SAC	27.5 ±1.53	27.7 ± 1.7	-0.17 (-1.07, 0.74)
BESS	14.82 ± 7.51	11.05 ± 5.49	3.89 (0.81, 6.96)
CRT	208.64 ± 21.3	206.19 ± 24.9	-0.04 (-11.18, 11.10)
Tandem Gait (s)	10.05 ± 1.75	9 ± 1.05	1.29 (0.57, 2.00)
KD (s)	39.49 ± 6.97	38.77 ± 6.79	1.4 (-1.4, 4.2)
ImPACT Motor Speed	39.9 ± 5.9	39.27 ± 6.05	0.33(-1.51, 2.16)
ImPACT Reaction Time	0.6 ± 0.06	0.59 ± 0.06	0 (-0.04, 0.03)
ImPACT Verbal Memory	86.43 ± 10.34	87.44 ± 10.74	0.13 (-5.88, 6.15)
ImPACT Visual Memory	80.57 ± 12.32	79.81 ± 12.71	2.53 (-4.93, 10)
ImPACT Impulse Control	5.87 ± 4.71	5.19 ± 2.72	0.67 (-1.19, 2.52)
ImPACT Symptoms	5 ± 11.27	1 ± 2.94	2.13 (-1.04, 5.31)
ImPACT Cognitive Efficiency	0.3 ± 0.12	0.36 ± 0.11	-0.06 (-0.12, 0.01)

Table 5: Regression Values for Change Scores Between Baseline and RTP for CONC Group

Clinical Concussion Battery Component	Odds Ratio (95% CI)	P value
GSC	1.10 (0.72-1.7)	0.666
SAC	0.42 (0.14-1.26)	0.123
BESS	0.91 (0.72 - 1.16)	0.452
CRT	0.99 (0.92 – 1.06)	0.743
Tandem Gait	0.66 (0.26-1.65)	0.369
KD	1.11 (0.89-1.39)	0.366
ImPACT Motor Speed	0.87 (0.56-1.37)	0.556
ImPACT Verbal Memory	1.27 (0.92, 1.74)	0.143
ImPACT Visual Memory	1.18 (0.94, 1.49)	0.16
ImPACT Impulse Control	0.58 (0.26-1.30)	0.188
ImPACT Symptoms	1.02 (0.79, 1.34)	0.862

Chapter 4

DISCUSSION

This study examined the relationship between MSK injury and SRC, and evaluated injury determinants and diagnostic test scores used in the institutional concussion test battery to predict future MSK injury risk. CONC athletes were significantly more likely to sustain any MSK injury, any LE, and LE NC injury than athletes from the Non-CONC group within 365 days of their RTP. No relationship was found between scores on the concussion battery and subsequent risk of injury over the same time period. Increasing awareness of prolonged motor control deficits following SRC indicate our current diagnostic methods of evaluating concussion may not be sensitive enough to detect all lingering effects of a concussive injury.

The main finding of this study was a nearly threefold elevated risk of subsequent MSK following a sports-related concussion. The findings of this study align with previous literature describing increased MSK injury risk in collegiate athletes.[24,25] Recently concussed athletes were found to be at 2.48 and 3.39 times more likely to sustain LE injury within 90 days of RTP, and 1.64 times more likely within one year of RTP.[24,25,29] Although the data approaches significance, athletes in this study were no more likely to have a time loss injury than their non-concussed peers before or after SRC; this suggests that athletes in our subject pool may not have been at an increased risk for severe injury following SRC, but were more likely to sustain minor non time-loss MSK injuries. No conclusive precedent in the literature has been set regarding TL injury: athletes have been found to be 60% more likely to sustain a

time loss injury, but without an aggregate increase in total time loss compared to non-concussed subjects over a competitive season.[30] Conversely, another study recently found concussion increased the risk of TL injury but had no effect on the odds of a high school athlete sustaining a NTL MSK or LE injury.[32] The results of the present study align with a third publication that found SRC is not associated with significantly higher time loss secondary to MSK injury compared to healthy peers.[29] Given that no significant difference was found in the rate of any type of MSK injury prior to SRC, our findings do not support the theory suggested in a previous study that athletes who sustain SRC are simply more injury-prone.[27]

There are significant gaps in our understanding of exactly why this relationship between SRC and MSK injury risk exists. Neurometabolic abnormalities have been described as long as four years following concussion, but the specific consequences of these changes is not understood at this time.[36–40] Impairments in activation of the primary motor cortex may be the source of lingering neuromuscular control deficits manifested through abnormalities in gait and muscle activation.[2,17,19,29] Deceased reaction time secondary to disrupted cortical pathways may predispose an athlete to injury, however contemporary clinical measures of reaction time were not a predictor of injury in this study.[24] Impaired neuromuscular function secondary to LE injury has also been implicated in subsequent NC LE injuries.[21,28,41] These abnormalities have been identified as potential underlying factors for elevated MSK risk, however the explicit mechanism for their effect remains unclear.

Although multiple studies before us have established a correlation between SRC and increased MSK injury risk, this is the first study to examine the predictive value of clinical concussion battery scores in increased MSK injury rates. In this

preliminary study, we found no significant relationship between scores on commonly used diagnostic concussion tests and future risk of MSK injury. Furthermore, no significant differences were found between baseline scores of athletes who went on to sustain a SRC and their healthy peers. Athletes in this study also showed improvement in both BESS and Tandem Gait scores between Baseline and RTP, suggesting a possible learning effect over the course of their SRC recovery. RTP data was used as a comparison point as we found no difference between CONC and non-CONC groups at baseline, indicating any potential change from baseline to RTP could highlight an individual test's ability to detect future risk of MSK injury. Based on these results, future research examining concussion battery test scores at different time points is warranted.

Independently of concussion, poor neurocognitive performance has been associated with increased risk of injury in previous studies, as well as poor neuromuscular control and attention deficits.[21,42,43] Although poor performance on components of ImPACT have been associated with increased risk of subsequent ACL injury, specialized equipment is frequently required to detect subtle changes in neuromuscular control.[18] When interpreted alongside contemporary literature, our results suggest the current tests we use to measure SRC recovery are unable to detect deficits that may lead to increased risk of injury.

These preliminary results suggest contemporary measures of concussion recovery are unable to detect deficiencies that persist long after currently accepted standards of clinical recovery, potentially contributing to an increased risk of MSK injury. Given the well-established link between SRC and subsequent injury, our findings strengthen the argument that gaps in our understanding of concussion may

have adverse effects on athlete health and susceptibility to injury for at least a year after RTP.

Chapter 5

LIMITATIONS

This study was not without limitations; most notably, our sample size was lower than desired. Comparable studies have included 100 total subjects or more, and a sample of 104 subjects would be required to adequately power this study using preliminary odds ratios for LE TL MSK injury within a year of SRC reported in prior literature as well as results from this study.[32] This was due to limitations in access to our EMR, which significantly restricted our subject pool and was a factor we were unable to control or modify. The institution changed EMR databases in 2014, which limited this study to only sophomores, juniors, or redshirted seniors who sustained concussions between August 2015 and March 2017 and did not transfer, join, or quit the team within one year of their injury. Our study size was further reduced by excluding non-SRCs, which accounted for approximately of the concussive events during the studied time period. Due to the strict exclusion criteria applied, we were unable to expand our sample based on the resources available. In addition, although we matched for sport, position, height, and weight when possible, playing time could not be accounted for. It has been well documented that collegiate athletes are more likely to sustain injury during competition than practice, so potential risk for injury may have varied. (Murphy 2003) This limitation may be accounted for by the lack of differences found between CONC and Non-CONC groups prior to SRC. We were also unable to examine the role of LOC in future MSK risk, as only one athlete in our sample had confirmed LOC as a result of their SRC. Finally, this was a retrospective

study which relied on clinical data from a range of athletic trainers, physicians, and researchers. Variable documentation styles and missing data points at critical follow-up points limited our ability to examine certain determinants. Long-term longitudinal studies with larger sample sizes are needed to thoroughly evaluate different risk factors for injury.

Chapter 6

CONCLUSION

The purpose of this study was twofold: to assess the relationship between SRC and MSK injury, and examine possible determinants associated with concussive injury and future MSK injury. Within the studied cohort, CONC participants were 2 to 3 times more likely to suffer a subsequent MSK in the year following post-concussion RTP than closely matched non-concussed participants. However, no relationship was identified between any components of the clinical concussion testing battery and increased risk of MSK injury. These findings support earlier studies reporting an increased risk of MSK injury after SRC, and suggest the contemporary clinical battery is not sensitive enough to detect the underlying cause of this increased risk. This is the first known study of its kind examining the predictive value of components of the concussion test battery to predict subsequent MSK injury, and future research should build on this preliminary result to evaluate for potential relationships.

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Appendix A

ADDITONAL FIGURES AND TABLES

Table A1: Description of Concussion Battery Tests

Measure	Description	Functional Domain
Sport Concussion Assessment Tool 5 (SCAT5)[44]	Combination of GSC and SAC; consists of a sideline assessment, neurological screen, balance examination, graded symptom checklist and cognitive assessment.	Neurocognitive Postural Stability Symptom
Graded Symptom Checklist (GSC)[2]	22 item list of common concussion symptoms. Self-reported severity using a Likert scale of 0-6.	Symptoms
Standardized Assessment of Concussion (SAC)[45]	Series of questions to assess immediate and delayed memory, symptoms, orientation, and concentration.	Neurocognitive
Balance Error Scoring System (BESS),[46]	A series of three stances on stable and unstable surfaces.	Postural Stability
Tandem Gait[47]	Alternating heel to toe gait used to walk across a three metre line before returning to the starting position in the same method as quickly as possible.	Postural Stability
King-Devick[48]	A series of numbers on a set of cards or screen is read aloud in linear order without error as quickly as possible.	Occulomotor

Clinical Reaction Time[49]	Patient grasps a dropped measuring stick as quickly as possible.	Reaction Time
Immediate Post-Concussion Assessment and Cognitive Testing (ImPACT)[50]	Computer-based neurocognitive test. Measures attention span, working memory, sustained and selective attention time, non-verbal problem solving, and reaction time	Neurocognitive

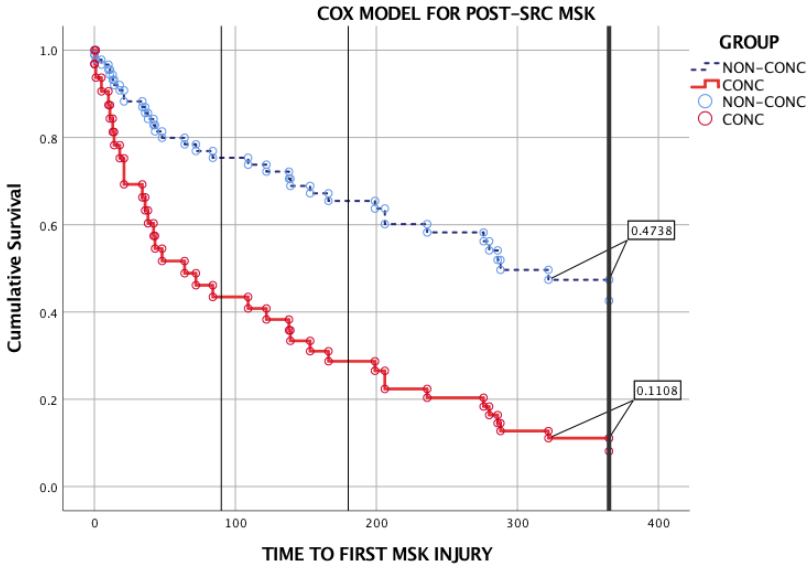

Figure A1: Cox Model for Post-SRC MSK Injury

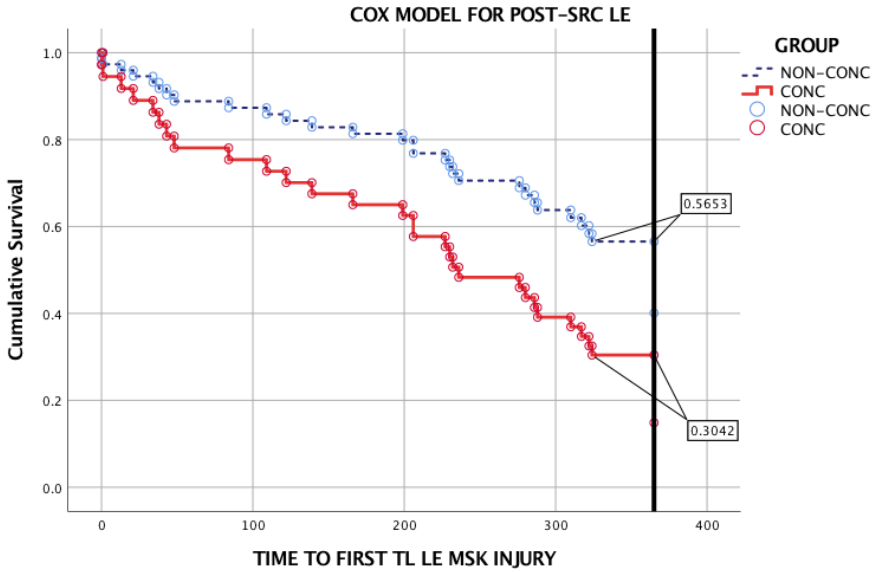

Figure A2: Cox Model for Post-SRC LE Injury

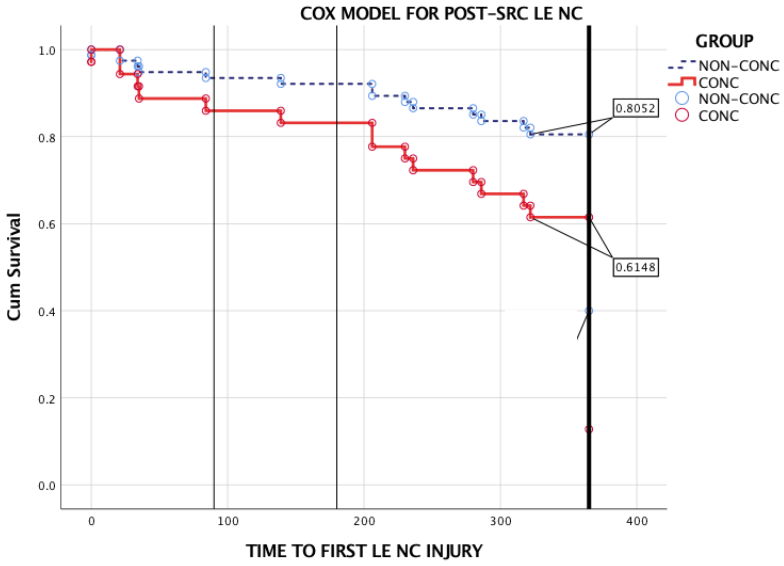

Figure A3: Cox Model for Post-SRC LE NC Injury

Appendix B

LITERATURE REVIEW

B.1 Epidemiology

Concussions are a significant public health concern, with an estimated 1.6 to 3.8 million concussions occurring in the United States each year. [51] Although no definition has been universally adopted across medical and athletic governing bodies, the International Conference on Concussion in Sport describes a concussion as a "traumatic brain injury induced by biomechanical forces" typically resulting in "the rapid onset of short-lived impairment of neurological function that resolves spontaneously." [1] Approximately 10,560 SRC occur in varsity National Collegiate Athletic Association (NCAA) sports annually, 9% of which are recurrent, however underreporting continues to be a persistent issue. [51–53] Approximately 30% to 50% of concussions are unreported by student-athletes (SA), indicating the incident rate of 4.47 per 10,000 athlete exposures (AE) may be significantly lower than reality.[33,54,55] Although NCAA football boasts the highest overall number of SRC annually (3,417), when AE is taken into consideration, men's wrestling has the highest rate of SRC with 10.92 per 10,000 AE, followed by men's and women's ice hockey (7.91 and 6.87 per 10,000 AE, respectively) and football (6.71 per 10,000 AE).[33]

Classically, sport related concussions (SRC) have been associated with a relatively short recovery period with as many as 90% of athletes returning to activity in 7 to 10 days, however more recent research addressing multiple domains of concussion indicate full recovery may take as long as three to four weeks.[23,56] Growing concern about the lasting effects of SRC is not new, but while knowledge of the neurophysiological and functional effects of SRC has improved, much remains to be understood in how to predict future risk of prolonged symptoms. Given the prevalence of SRC, professionals in collegiate settings are challenged to manage these injuries in line with the most up to date research.[57].

B.2 Physiology

Although SRC does not result in macroscopic neural damage following an external biomechanical force to the brain, resulting microstructural damages create a domino effect of impairments.[1,58] Following the initial impact, a metabolic cascade occurs which creates a chemical imbalance within affected neurons in the brain, causing the signs and symptoms commonly associated with SRC.[59,60] Disruption of neural membranes creates a sharp efflux of potassium, significantly increasing glucose demand within neurons to regain homeostasis.[61] However, cerebral blood flow significantly decreases following concussion for about eight days, negatively affecting the ability of mitochondria to perform aerobic metabolism, thus exacerbating the energy crisis.[59,62,63] Increased energy demands paired with impaired aerobic

metabolism results in an accumulation of lactate, resulting in acidosis, membrane damage, changes in electrophysiology, and altered blood brain barrier permeability.[60,64] This environment within the cell impairs neural function significantly, further exacerbating the effects of the primary mechanism of intracranial pressure and biomechanical strain. [65,66]

Much remains to be understood about the precise clinical effects of this neurometabolic crisis, however some larger connections have been made in relation to motor control deficits following SRC. Most neurometabolic studies are conducted using animal models for ethical reasons, but the use of previously concussed human models has increased in recent years.[59] Cerebral blood flow decreases in athletes post-SRC for at least eight days, demonstrating a clear gap between clinical and neurophysiological recovery.[62] Compromised metabolic integrity of the primary motor cortex (PMC) has been documented in clinically asymptomatic athletes following SRC, although much remains to be understood about the specific clinical implications of these pathophysiological changes.[22] Imbalances in GABA and Glutamate, neurotransmitters responsible for modulating inhibition and excitation in neural pathways, have been detected in concussed athletes one year following SRC, but these imbalances appear to even out within two years and the specific cause or effect of this relationship has not been established.[67] Impaired subclinical information processing is documented in asymptomatic athletes at least nine months following their injury, with cumulative effects evident in those who sustained multiple SRC.[68] In addition, SRC has been associated with subclinical neurophysiological abnormalities associated with

stimulus perception and cognitive processes, potentially resulting in mild but long-term functional deficits. [69] Despite recent progress in measuring these abnormalities, no consensus has been found regarding the ideal biomarker or neurophysiological standard with which to identify SRC, meaning no physiological standard for measuring recovery exists.[4] As a result, clinicians must rely on a range of clinical tests and professional judgment to formulate a diagnosis of SRC at this time. [1]

B.3 Signs and Symptoms

Due to its multifaceted nature, SRC presents with a range of signs and symptoms. Symptoms fall into one of three categories: somatic (e.g. headache), cognitive (e.g. feeling like in a fog), and emotional (e.g. lability).[1] Following SRC, symptom presentation can vary significantly within individuals. Headache is consistently reported as the most common symptom; however blurred vision, confusion, and "feeling slowed down" are commonly reported as well. [33,70–72]Although occurring in a very small percentage of athletes with SRC, the most common physical signs of SRC are loss of consciousness (LOC) and post traumatic amnesia (PTA), occurring in approximately 6% and 24% of cases, respectively.[63] Typically, symptoms resolve in seven to ten days, however athletes with a history of multiple SRC are twice as likely to experience prolonged symptoms than an athlete with no prior history.[63] The physiological basis of this increased risk is not fully understood at this time,

however this “dose response” relationship has been documented in multiple studies.[63,73,74]

B.4 Diagnosis

SRC is a multifaceted injury requiring a comprehensive battery of tests and refined clinical judgment to diagnose.[1,75] The most recent consensus statement on concussion in sport describes five clinical domains through which diagnosis should be made: symptoms, physical signs, balance impairment, behavioural changes, cognitive impairment, and sleep/wake disturbance.[1] SRC should be suspected if signs or symptoms from at least one of these domains are present, and athletes should immediately be removed from play and thoroughly evaluated by a trained healthcare provider before further participation. An athlete diagnosed with SRC should not be permitted to return to play on the day of injury. Due to normal variances within the population, such as biological sex or how well-rested an individual is, pre-season baseline testing of each athlete is important to establish an ideal value for each respective test an athlete may take during the diagnostic and RTP phases their recovery.[8,76–78] No difference has been found in baseline scores of athletes with a history of one or more concussions compared to athletes without a history of SRC.[76,79] The current standard of clinical evaluation with symptom, neurocognitive, and balance assessment has been refined significantly over the past decade, with a large

number of clinical tests developing over time to measure multiple facets of impaired function.[80]

B.4.1 Balance Assessment

The Balance Error Scoring System, or BESS, was developed to detect disturbances in static balance using a series of three postures.[45] This test is widely used for concussion assessment, largely because every consensus statement released by the International Conference on Concussion in Sport since 2004 has recommended it.[1,81–84] Despite these well publicized endorsements, the test does not differentiate between deficits caused by SRC or other causes, such as fatigue or chronic ankle instability.[85] In addition, due to poor interrater reliability and human error (ICC of 0.57 for total score), the BESS is only valid when significant differences exist between baseline and post-injury values and may not be useful for detecting more subtle changes shown to exist with more sophisticated equipment.[15,46,86] Poor sensitivity at time of injury (0.34) suggests the BESS is not able to effectively identify athletes with SRC, however its high specificity (0.91) does support its use in ruling out this injury.[2]

The Tandem Gait (TG) trial evaluates dynamic balance, speed, and coordination, and is a recommended item on the Sport Concussion Assessment Tool.[1,47] Athletes are instructed to walk with an alternating heel to toe gait down a three meter line before turning 180 degrees and returning to the starting point as quickly as possible.[87] TG has excellent reliability (intra class correlation 0.97), is

simple to administer, and has been shown to be a valuable component of the SRC testing battery.[47,88]

B.4.2 Vision Assessment

The King-Devick (KD) test is used to detect suboptimal occulomotor, visual, and cognitive abilities following concussion.[48] The test consists of reading aloud a series of single digit numbers from three cards on paper or screen, and is scored using the total time required for an individual to complete the entire series without error.[89] While limited evidence supporting the use of this assessment exists outside of KD-affiliated research, KD testing has been found to be a reliable method to identify patients with head trauma. Despite this, recent changes in cost and accessibility of the test may diminish its use in settings without a substantial budget dedicated to SRC diagnostic testing.

B.4.3 Symptom Assessment

The Graded Symptom Checklist (GSC) is a 22-item list of common SRC symptoms paired with a Likert scale ranging from 0 to 6 (least severe to most severe) with a maximum score of 132. The GSC has been modified and expanded over the years, but includes symptoms from all recognized domains of SRC, including headache, dizziness, confusion, and feeling in a fog. An early version of the GSC was found to have extremely high sensitivity and specificity at time of injury (0.89 and

1.00, respectively) with only 17 items.[2] GSC scores typically do not return to baseline values until several days after other components of the testing battery, suggesting underlying physiological deficits linger beyond resolution of functional impairments. [2]

B.4.4 Neurocognitive Assessment

The Standardized Assessment of Concussion (SAC) contains orientation, immediate memory, concentration and delayed recall challenges that address the cognitive impairment domain of SRC.[1,90] Because the SAC takes about five minutes to administer, it is a practical sideline assessment tool for many traditional sports. While its sensitivity and specificity are both high at time of injury (0.8 and 0.91, respectively), sensitivity drops to 0.31 within 24 hours of injury, suggesting the SAC may be of limited utility in the diagnostic process if an athlete does not immediately report to their healthcare provider.[2]

The Immediate Post-Concussion Assessment and Cognitive Testing (ImPACT) tool is a widely used computer-based test that assesses neurocognitive function. Composite scores for verbal memory, visual memory, visual-motor processing speed and reaction time are generated and compared to baseline scores to determine the presence of significant deficits post-SRC. Approximately 89% of athletic trainers in the university setting use ImPACT as part of their standard SRC assessment battery.[91] ImPACT is used across all levels of sport, and has been found to have good convergent validity.[92] ImPACT is a computer-based test designed to be administered

in a distraction-free environment, but its results are adversely affected by real world testing environments, such as baseline testing administered in a group setting. Although measures have been put in place to prevent sandbagging, between 10% and 35% of athletes are able to intentionally underperform on the test, which could increase the likelihood he or she improperly meets a baseline threshold following SRC.[93,94] Although no test is perfect, with a sensitivity of 81.9% and specificity of 89.4%, ImPACT provides useful information in both the diagnostic and RTP phases of SRC management. [50]

B.4.5 Sport Concussion Assessment Tool

The Sport Concussion Assessment Tool 5th Edition (SCAT5) was released following the 5th International Conference on Concussion in Sport in 2017 and is considered by many as the standard pen and paper diagnostic tool.[1] The SCAT5 combines the SAC and GSC to form a multifaceted tool consisting of a sideline assessment, neurological screen, balance examination, graded symptom checklist and cognitive assessment. [44] Although comprehensive, the SCAT5 does not function as a stand-alone tool to diagnose SRC, and no clinical threshold for what constitutes a passed or failed test has been established in current literature.

B.4.6 Comprehensive Testing Battery

When multiple tests are used in the diagnostic process, sensitivity of the current SRC testing battery is excellent. Sensitivity ranges from 0.89 to 0.96 when symptom evaluation, postural control, and cognitive testing are used together at time of injury.[2,95] This value drops significantly after the first 24 hours post-injury, suggesting these tests are of limited diagnostic use in detecting SRC beyond the immediate aftermath of the injury.[2] Sensitivity drops to 0.69 the day following injury, and decreases to 0.14 after a week has passed.[2] Specificity of the testing battery remains relatively constant throughout the recovery process, increasing from 0.89 at time of injury to 0.93 seven days after the impact, with a brief decrease 48-72 hours post-injury (0.84).[2] This data reveals a major flaw in the current standard SRC testing battery: if clinicians cannot accurately identify a SRC one week after injury using the only tools available, the concept of a gold standard for SRC recovery is inherently flawed.

B.5 Standard of Care

Refinement of concussion testing over the past decade has substantially improved clinicians' abilities to diagnose concussions; however the specificity of the current concussion testing battery leaves significant gaps in our understanding of recovery from SRC.[4,50] The current standard of care requires an athlete to be asymptomatic before beginning a return to play protocol, but this symptom free waiting period has been found to have no influence on clinical recovery, post-injury

performance, or risk of a repeat concussion in collegiate or high school athletes.[1,75,96,97] This is contradicted by other studies which show high levels of activity following SRC exacerbate symptoms and delay neurocognitive recovery.[98] As a result of these shortcomings, current best practice guidelines continue to prioritize subjective clinical judgment over objective diagnostic test results in determining appropriate SRC management.[1]

Gaps between "clinical recovery" and neurological function have been established for some time.[64] Based on functional magnetic resonance imaging (fMRI) changes, the brain's ability to overcome functional deficiencies is attributed to neural plasticity, allowing a still-recovering athlete to perform at baseline levels when challenged with relatively simple tasks found in a standard SRC test battery.[99] Numerous studies have found athletes return to baseline values on SAC and BESS tests before they return to an asymptomatic status, indicating performance on these tests alone should not determine RTP progression.[2] Neurometabolic abnormalities have been reported as long as 4 years post concussion, but little is understood about what, if any, specific long-term consequences these abnormalities will have. [37–40,100] These neurometabolic disturbances may negatively affect gait stability and control following SRC, as alterations in gait have been documented two months following SRC, surpassing return to baseline on current tests of static and dynamic balance by a large margin.[2,17,19]

B.6 SRC and Gait Stability

Acute deficiencies in postural stability after SRC have been documented since 2000, but increased postural sway has been identified following SRC in both acutely symptomatic and asymptomatic athletes who have cleared full RTP testing. [14,17] Postural sway is a traditional measure of postural control, and is quantified using distributional measures of horizontal deviation from the center of gravity.[101] Athletes apply different postural control strategies following SRC, altering their postural sway in a way that increases postural instability as a result of these adaptations.[101,102] The mechanisms behind these specific alterations are not fully understood, but Buckley et al hypothesized athletes limit motor systems after SRC to reduce variability in posture.[19] These static and dynamic balance impairments have been associated with conservative gait pattern, identified by an individual's reduced gait velocity and increased time in double stance phase.[103]

Athletes who have sustained a SRC are more likely to adopt a more conservative gait pattern than those who have not, and a positive correlation has been found between frequency of concussions and increasingly conservative gait patterns.[15,47] Buckley et al used force plates to conclude gate termination strategy in athletes is altered for at least 10 days post-SRC, finding athletes who successfully pass traditional balance tests have lingering motor control deficits well after they return to activity.[18] Links between executive function and gait are well established in healthy populations, and history of SRC has been found to impair an athlete's ability to perform a cognitive task while walking in both the acute recovery phase and two

months post injury compared to non-concussed peers.[103,20,104] TG times are significantly slower for two weeks in concussed athletes compared to healthy controls when a simple cognitive task is added.[87] Following SRC, athletes struggle to regulate dividing attention between motor tasks and external distractions, impairing gait pattern as well as cognitive ability when compared to healthy controls.[20,103] Despite this, the vast majority of athletes reach full symptom resolution within seven days of injury and are cleared to return to full activity using a battery of evaluative tests within ten days[63,70] This information exposes weaknesses within the current concussion testing battery, particularly with regard to motor control and dual-tasking challenges. Given the complex cognitive and physical demands placed on collegiate and professional athletes, the current assessment and RTP standard may be falling short of identifying athletes who have not yet returned to normal thresholds of motor control. The main components of our current neurocognitive and balance test battery are not sensitive enough to identify athletes with postural control impairments that may predispose them to future injury following a SRC.

B.7 SRC and LE Injury

Despite not fully understanding the underlying mechanisms of these lingering motor control deficits, researchers have correctly theorized these deficits result in higher injury rates in athletes following SRC. Many collegiate and professional sports require quick, powerful movements, and any impairment of an athlete's ability to control

these movements could place them at a higher risk of injury. Injuries to the lower Extremity (LE) account for over 50% of injuries sustained in NCAA sports as a result of the dynamic closed chain LE demands placed on the majority of athletes.[34]

B.8 LE Injury in Collegiate Sports

A strong correlation exists between SRC and increased rates of LE injury in collegiate athletes following their full return to play when compared to athletes without a history of SRC. [24–26,29,79,96] Recently-concussed athletes (RCA) are 2.48 and 3.39 times more likely than their non-recently concussed peers to sustain a LE injury in the 90 days following clearance from their concussive injury. [25,29] More alarming is that RCA continue to be at a higher risk of injury as long as one year following full clearance, and have been reported to be 1.64 times more likely to sustain a LE acute injury than their non-concussed peers, and 1.97 times more likely after a SRC than before.[24] Higher incidences of lateral ankle sprains (P= 0.012), knee injuries (P=0.031), and lower extremity muscle strains (P= 0.003) were significantly associated with self-reported SRC compared to athletes who did not report a history of SRC over the course of a year as well.[26]

Several factors of this relationship, including time loss and dose response, are not fully understood at this time. Despite the increased risk of injury, athletes who sustain LE injuries following a SRC have not been found conclusively to have significantly higher gross time loss as a result of those injuries than athletes

without.[29,30] Athletes with a SRC missed on average 9 days per LE acute injury, whereas athletes without a history of SRC missed 15 days.[29] Although this finding was not statistically significant, 6 days of time loss is clinically significant for an in-season athlete, as multiple games can occur between the 9 and 15 day period. Additionally, athletes were 60% more likely to suffer a time loss injury of any type within a competitive season than their non-concussed peers, but there were no specific differences in gross time loss between groups.[30] The risk of LE injury increases if an athlete has more than one documented SRC, changing from an 18-63% increased risk over the course of a year in athletes with one concussive event to 73-165% increases in athletes with at least three documented SRC. [28]

B.9 LE Injury in Professional Sports

Effects of SRC on LE injury rate in professional athletes are consistent with data collected on collegiate athletes.[24–30,79] Professional European soccer players with SRC were 4.07 times more likely to suffer a LE injury than matched controls in the 6 to 12 month period following their SRC.[27] This risk was greater than both the first three months after SRC (1.56 times) and three to six months after (2.78 times). Professional Rugby Union players were 60% more likely to sustain a time loss injury if they sustained a SRC than if they did not over the course of a season.[30] This statistic is even more alarming given the extremely high incidence of SRC in professional European rugby reported in this study: approximately 8.9 SRC per 1,000 hours of

match play.[30] An increased risk of LE injury was identified in retired National Football League players who sustained SRC during their professional career.[28] The number of LE injuries sustained by retired NFL athletes was proportional to the number of SRC sustained, with increased likelihood of injury ranging from 18-63%, 15-126%, and 73-165% in players with one, two, and three or more SRC respectively.[28]

B.10 Conclusion

SRC is a complex, multifaceted injury that requires specialized care provided by a highly trained health professional. With other 10,000 SRC annually in the NCAA, increasing awareness of the relationship between SRC and LE injury has garnered curiosity about how to detect changes in a clinical setting to identify athletes at a higher risk of LE injury. Further research in this realm of SRC is warranted, as no predictive factor has been identified at the time of writing.

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