THE EFFECT OF CONCUSSION ON SUBSEQUENT LOWER INJURY IN CURRENT NFL PLAYERS

by

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ABSTRACT

Context: Recent literature suggests individuals may be at an elevated risk for sustaining a subsequent injury following return to play from sport related concussion (SRC). National Football League athletes are underrepresented in this literature, and as football has one of the highest concussion incidence rates in sport, it is important to examine the relationship between concussion and musculoskeletal (MSK) injuries in this cohort.

Objective: The first aim of this project was to assess the association and risk of lower extremity musculoskeletal injury (LEMSK) both prior and after concussion between team and position-matched players who sustained an SRC and players who did not during the same season. The second aim of this study was to examine the association and risk of LEMSK following concussion of position-matched players who sustained an SRC and those who sustained an injury that was non-lower extremity.

Design: Cohort study

Setting: Publicly available NFL injury report data.

Patients or Other Participants: NFL athletes

Main Outcome Measure(s): The concussion rate and the rate of LEMSK pre and post-concussion.

Results: Chi square analysis showed no association between concussion and LEMSK post-concussion ($x^2 = 2.19$, $p = 0.17$) for Aim 1 (concussed vs. non-concussed player/team matched controls) or Aim 2 ($x^2 = 2.58$, $p=0.13$) (concussed vs non-concussed and non-LEMSK). There was a significant association between concussion and LEMSK during the pre-concussion time period ($x^2=15.22$, $p<.001$) between players who sustained

a concussion and those that did not. Odds ratio pre-concussion was 0.52 (95% CI: 0.37-0.72), where the control group was more likely to sustain a LEMSK. For Aim1, the Cox proportional hazard model showed no significance for groups post-concussion, but statistical significance was identified pre-concussion ($p<.001$) where prior to sustaining a concussion, the non-concussed players were 1.72 times more likely to sustain a LEMSK than the concussed group. For Aim 2, the Cox model was not significant ($p=0.14$).

Conclusion: Concussion did not increase the likelihood of appearing on the NFL injury report for LEMSK pre or post-concussion in NFL players. Both players with and without concussions are about at equal risk to sustain a LEMSK post-concussion, and players who did not go on to sustain a concussion were more likely to sustain a LEMSK than those who sustained a concussion prior to the concussion event. Players that sustained a non-LEMSK injury prior to their matches' concussion week also did not show an increased risk to appear on the NFL injury report with a LEMSK compared to the concussed group.

Chapter 1

INTRODUCTION

Concussion is defined as "a traumatically induced transient disturbance of brain function and involves a complex pathophysiological process."[1] It is estimated that between 1.6 and 3.8 million sport related concussions occur annually in the USA.[2] Contact/collision sports such as football, men's lacrosse and soccer in the high school population have been previously identified as having prevalence rates of 6.1 – 9.2 concussions per 10,000 athlete exposures.[3] Football has among the highest incidence rate of all sports,[3–5] with reported incidence rates in FBS level football of 0.81 per 1,000 AE.[5]

The current literature suggests that most athletes return to play (RTP) from concussion in 7-10 days.[4,6,7] Presently, it's suggested that using a multifaceted battery of clinical tests is the most effective way to help clinicians identify concussions.[8–10] The battery often includes symptom recognition, postural stability, and cognitive functioning.[8] While these test have been proven to be effective for diagnosis (89-96% sensitivity), they may not be the best at guiding RTP decisions.[8,11] Recently published literature has also suggested that at the time of RTP, an athlete may not be fully recovered and neurological deficits persist beyond clinical recovery.[11–13] Kamins et al. reported that neuroimaging, blood flow measures, and EEG all show neurophysiological disturbances beyond the standard RTP timeline of 7-10 days, often lasting between 15-30 days post-concussion.[11] Similarly, gait impairments have been identified in concussed athletes which persist beyond clinical recovery.[12–14] In

theory, decreased motor function may alter the body's ability to adapt to rapid changes an athlete might be presented with when playing their sport.[15]

A football athlete needs to be able to react in a timely manner to different events that they may face on any given play. Therefore, dual task models have been used to study and recreate these challenges and these protocols have been used to identify prolonged lingering deficits after RTP.[13,14,16] As a result of these deficits being present after RTP, the athlete may be at an increased risk to develop subsequent injury.[17–23] One of the first studies examining the topic of concussion and subsequent injury found that there was a 2.2 times increased risk of subsequent injury following concussion in professional soccer players for up to 12 months post-concussion.[18] It is also important to note that in this study injury risk increased over time and subjects were found to have about a two times increased risk for subsequent injury before the concussion as well, making it uncertain as to whether the increased injury risk post-concussion is due to concussion pathology, or to an athlete's "injury proneness".[18] Multiple studies have found within the NCAA Division I setting previously concussed athletes are at an elevated risk to sustaining a subsequent lower extremity musculoskeletal injury (LEMSK) than those with no concussive history; with rates being reported to be up to 3.39 times in the year following sport-related concussion (SRC).[17,20,22–24] The majority of subjects in these studies are football athletes, which is not surprising given the previously reported SRC and musculoskeletal (MSK) injury rates.[3–5,17,20] Given the emerging evidence of lingering deficits and elevated (LEMSK) risk, it has been argued that post-concussion RTP should be lengthened.[20]

NFL studies focus on the long term sequalae of SRC and have been performed after retirement, with little investigation of active NFL players.[19,25–31] Traditionally, self-reported survey studies are used after retirement to report findings regarding concussions in the NFL.[19,25,26,28] While elevated injury rates have been noted in collegiate and high school athletes, NFL injury rates for currently active players have not been explored. Therefore, the purpose of the study was to evaluate injury patterns in active players throughout three NFL seasons (2015-2017), and examine the association of SRC and risk of LEMSK. Specifically, the first aim of this study was to assess the risk of LEMSK, via presence on the official NFL injury report, between players who suffered a SRC and position matched teammates who did not sustain a concussion that season.[32] The second aim of the study was to compare LEMSK risk, via presence on the official NFL injury report, between NFL players who suffered SRC and position matched players who had not sustained a concussion, but had sustained a non-lower extremity injurie. We hypothesized that the athletes who sustained a concussion would have a higher risk of sustaining a LEMSK for both aims.

Chapter 2

METHODS

2.1 Design

For this study, a cohort design was used to identify if NFL players who sustained a concussion were more likely to sustain a LEMSK in the same season than players with no SRC during that respective season. We systematically identified current NFL players who sustained a SRC and reviewed LEMSK reported on publicly available NFL injury reports.[33] As all data collected during this study was publicly available, the university's institutional review board approval deemed the project exempt from informed consent.

2.2 Data Acquisition

Injury report data was collected for all 32 NFL teams during the 2015, 2016, and 2017 seasons. Each week throughout the course of a season all 32 NFL teams are required to submit an official injury report to the NFL, which is then made public on NFL.com. The data compiled consisted of player status for that respective week (active/ inactive) and the injured body part. While this information was available on a variety of websites, for this study only the NFL.com was used because this is the official report provided by the league.[32]

To be included in the study, individuals had to have been active on a 53-man roster for at least one week. Players in the concussion group must have sustained a reported SRC that appeared on at least one of the NFL's injury reports during the 2015-16, 2016-17, or 2017-18 season. We adopted the NFL's definition of concussion for this study, which references the 5th consensus statement as their

definition for a concussion, which is defined as "a complex pathophysiological process affecting the brain, induced by biomechanical forces"; however concussion diagnosis and reporting was subject to individual franchise reporting accuracy.[7] Healthy was defined as a player who does not appear on the injury report over the course of the season. Non-LE injury was defined as an injury to the body excluding the hip/groin, upper leg, knee, lower leg, and ankle/foot that resulted in being listed on the respective team's injury report. Exclusion criteria included sustaining multiple concussions in the same season, or sustaining a season-ending concussion. Only regular season concussion and injury reports were analyzed; neither preseason nor postseason data were included in this study.

2.3 Procedure

A database was created for each individual season (2015/2016-2017/2018). Each of the 32 NFL teams received its own tab within the spreadsheet. Each tab contained weeks 1 through 16 on the x-axis and each player's name and position on the y-axis. (Figure 2.1) Injury reports were reviewed weekly over the course of the regular season, and injuries were documented accordingly using our injury key (Figure 2.2).

To address Aim 1, all players who suffered a concussion were identified and matched to a teammate of the same position with no reported concussion over that entire season. To address Aim 2, all players who suffered a concussion were identified and matched to a player of the same position who had not suffered a concussion that season but had sustained a non-lower extremity injury during the weeks prior to the concussed subject's RTP week (for example, if a player sustained a

concussion in week 5, the matched control must have sustained a non-LE injury prior to or in week 5). If a matched control player could not be found on the same team, a matched control was then accepted from another team. Position was narrowly defined initially (e.g., middle linebacker), but as matched control could not be identified, a broader definition (e.g., linebacker) was used. Primary position was identified based on position listed on the NFL.com website.

2.4 Statistical Analysis

Descriptive statistics were reported for all groups and all variables and were classified as categorical (injured or not injured). A chi-square analysis was performed to address both aims to compare the expected vs observed frequency of reported injury in each group. Specifically, in Aim 1 the association between appearing on the injury report for a LE injury was compared between the concussion and control (uninjured group) for two timepoints: pre-concussion, or injuries that occurred prior to the week of the concussion, and post-concussion, or injuries that occurred at or after the concussed player's RTP week. For Aim 2, the association between appearing on the injury report for a LE-injury was compared between the Concussion and Control (other injury) group post-concussion. If a significant association was identified, a post-hoc odds ratio was calculated. Also, a Cox proportional hazard model was also used to determine the risk of sustaining a LEMSK during the evaluated time frames. Again, for Aim 1, risk was observed during both pre and post-concussion time points. For Aim 2, risk was observed only post-concussion as the matched controls were required to have sustained a non-LEMSK prior to the subject's concussion week.

FIGURES

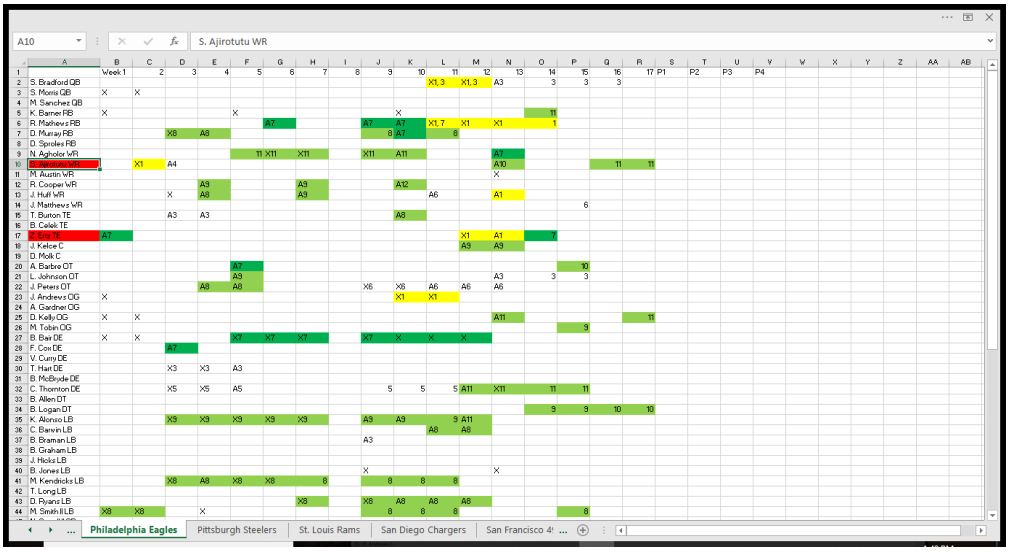

Figure 2.1: Excel NFL injury report spreadsheet

A: Active
X: Inactive
1: Concussion (if it is not yellow it is listed as a headache/head injury)
2: neck
3: shoulder
4: arm/elbow
5: hand/wrist/finger
6: chest/back/torso/ribs
7: hip/groin
8: hamstring/quad/thigh
9: knee
10: lower leg/calf
11: ankle
12: foot/heel
13: face

Figure 2.2: Injury Key

Chapter 3

RESULTS

In the 3-year experimental time frame there were 397 total concussions reported during the regular season within the NFL, via public injury reports. Of those, 322 concussions were used to identify if there was an increased probability of sustaining a LE injury in the concussed group compared to the non-concussed group for Aim 1. Forty-one subjects were excluded because they sustained season ending concussions, and another 34 were excluded for sustaining multiple concussions. Crosstabulation tables were constructed for the pre-concussion (Table 3.1) and post-concussion (Table 3.2) analyses to show the total number of subjects in each group and used to calculate post-hoc odds ratios. For Aim 1, chi-square analysis showed no association between groups post-concussion (x^2= 2.19, p= 0.166) but showed a significant difference between groups pre-concussion (x^2= 15.22, p< 0.001). The odds of the concussed group sustaining a LEMSK pre-concussion compared to the non-concussed group was .519 (95% CI 0.373- 0.723).

The Cox proportional analysis was not significant for increased risk post-concussion (p= 0.123) (Figure 3.1). However, a significant difference was found for an increased risk to sustain a LEMSK in the non-concussion group prior to the concussion week (p< 0.001) (Figure 3.2). It was calculated that there was a 1.72 times greater risk (95% CI 1.31-2.25) for the players without a concussion to sustain a LE injury during the weeks prior to the matched subjects' concussion week.

To identify the probability and risk of sustaining a LEMSK in Aim 2, 309 total subjects were analyzed. Of the 322 subjects analyzed in Aim 1, 13 additional

subjects were excluded for either having sustained a concussion in week one or not having a comparable non-LE control match for a subject. A crosstabulation table was constructed for the post-concussion (Table 3.3) analysis to show the number of subjects in each group. The chi square analysis showed no significance (x^2= 2.58, p= 0.130) between groups post-concussion. The cox proportional hazard model showed no significance (p= 0.143) between the concussed and non-LE injury group as well (Figure 3.3).

FIGURES AND TABLES

Table 3.1: Crosstabs for Pre-concussion (Aim 1)

Pre-concussion				
Concussion		**No LE-Injury (0)**	**LE Injury (1)**	**Total**
	No (0)	188	134	322
	Yes (1)	235	87	322
	Total	423	221	644
Crosstabs of the concussed and non-concussed groups for pre-concussion week				

Table 3.2: Crosstabs for Post-concussion (Aim1)

Post-concussion (Aim 1)				
Concussion		**No LE-Injury (0)**	**LE Injury (1)**	**Total**
	No (0)	237	85	322
	Yes (1)	253	69	322
	Total	490	159	644
Crosstabs of the concussed and non-concussed groups for post-concussion week.				

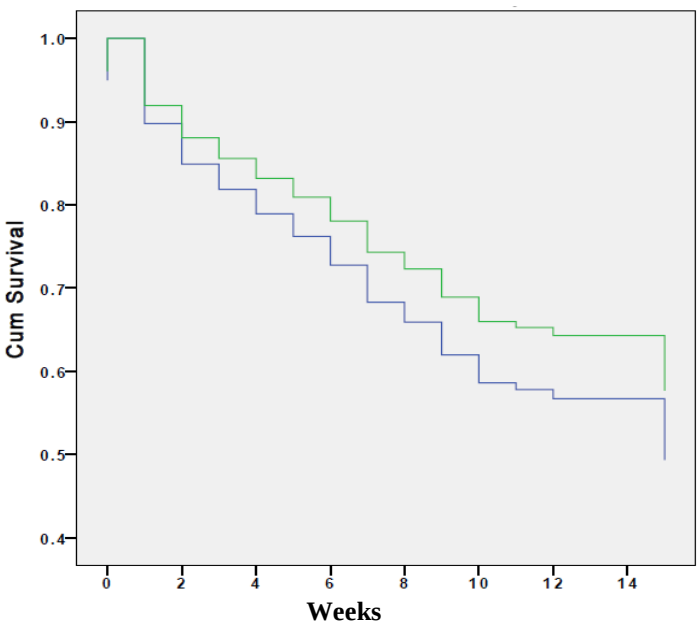

Figure 3.1: Post-concussion Survival Analysis Curve (Aim 1)

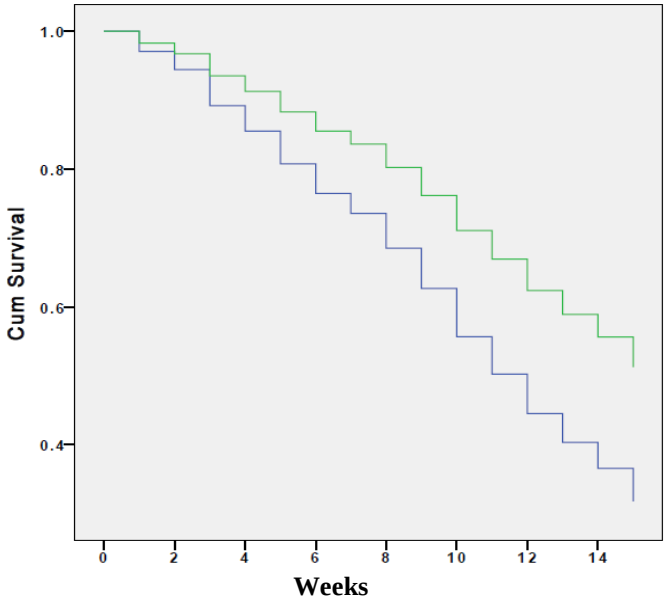

Figure 3.2: Pre-concussion Survival Analysis Curve

Table 3.3: Crosstab for Post-concussion (Aim 2)

Post-concussion (Aim 2)				
Concussion		**Other Injury (0)**	**LE Injury (1)**	**Total**
	No (0)	227	82	309
	Yes (1)	244	65	309
	Total	471	147	618
Crosstabs for the concussed vs non-LE injury groups post-concussion week				

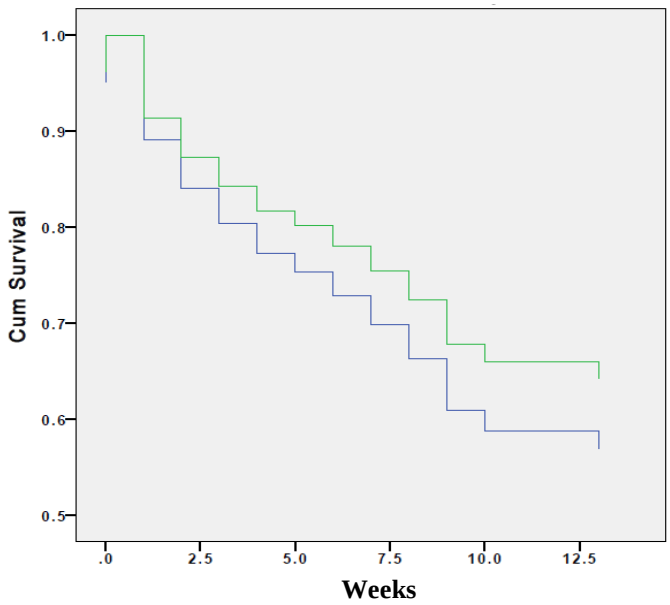

Figure 3.3: Post-concussion Survival Analysis Curve (Aim 2)

Chapter 4

DISCUSSION

Emerging scientific evidence suggests there is an elevated risk of subsequent LEMSK over the year following a concussion; however, findings to date have been primarily limited to high school and collegiate athletic settings in the U.S.[17–20,22–24] The results of this study suggest NFL players who sustained a concussion were not at an increased risk of sustaining a subsequent LEMSK based on team provided injury reports. However, we did find that players that did not sustain a concussion were 1.72 times more likely to sustain a LEMSK than the concussed subjects in the pre-concussion time period; these findings suggest LE injury in NFL players may not be associated with concussions, which is the opposite of what previous literature has suggested.[19] Further, our results suggest that individuals that will not go on to sustain a concussion during a given season may be at an increased risk to sustain LEMSK injury earlier on in the season compared to individuals who will go on to sustain a concussion.

Previous literature has suggested that post-concussion, athletes are at an increased risk (↑1.5-3.0x) to sustain LEMSK in the year following concussion.[17–24] The first study to examine subsequent injury post-concussion in professional athletes was performed by Nordstrom et al., in professional European soccer players.[18] Unlike our study, an increased risk of injury in professional soccer players was found both pre and post-concussion, eluding to a possible explanation that those concussed athletes may just be more "injury prone" and more likely to sustain any injury. This study was able to adequately control for injury type, position, and exposures;

however, they included any injury sustained pre and post-concussion and did not limit to strictly lower extremity injuries. The lone NFL subsequent injury study done by Pietrosimone et al. reported a 1.2-2.7 times increased odds of MSK injury in players who sustained one or more concussion throughout the course of their career; however it is important to note this was based on individual recall of "serious" injuries post-career.[19] Conversely, our study strived to obtain current data and only included active players. Although this study was performed using reported injury rates and not direct medical notes, NFL policy mandates all 32 teams to submit injury reports that are "credible, accurate, timely and specific within the guidelines of the policy".[33] However, it must be noted that there has been speculation that NFL teams may not be fully honest when submitting weekly injury reports.[34] Until now, past research involving an NFL cohort was not able to establish a time-based risk of LEMSK pre- or post-concussion.[19] Our results are in part reflected by a study of professional ice hockey players, which showed that hockey players who suffered a SRC were not at a higher risk to sustain a subsequent injury compared to players who experienced a knee injury.[35] While there seems to be mixed results on subsequent injury in the professional setting, both high school and collegiate settings show a definite increase in MSK injury risk post-concussion (up to 3.29 times).[17,20,22–24] Several studies have also suggested that subsequent injury is more likely in the first 90 days following return to play from concussion, which likely includes time spent in-season.[17,20,24] All studies that have identified an increased subsequent injury risk have been populations other than professional football. At this time no one consensus has been established

on subsequent MSK injury risk in professional sports, as some do show an increased risk, or, like our results, do not.[18,2135]

Injury proneness has been listed as a possible explanation to why specific groups may be more likely to sustain MSK injury pre or post-concussion.[18,35] Likewise, injury proneness may also be an explanation why our non-concussed group showed a significant elevated risk to sustain a LEMSK during the pre-concussion time frame compared to the concussed group. Both Nordstrom et al. and Nyberg et al. agreed that concussed individuals may just be more prone to injury, they did not speculate why this phenomenon may occur.[18,35] Possible reasoning behind injury proneness may include neurological deficits as a result of subconcussive impacts.[36,37] It has been previously suggested in soccer literature that repetitive subconcussive headers may be a cause for temporary vestibular and postural control impairments.[36,37] Like soccer players, football players sustain a great number of subconcussive impacts throughout the course of one season. It has been previously reported that college football players sustain approximately 1,000 head impacts per season.[38] With these large amounts of head impacts present in the current study it can be speculated that this population may be exposed to experiencing these vestibular and postural control impairments which may ultimately lead to LEMSK injury. Genetic anatomical abnormalities, such as hypermobility of joints, may also be a factor behind injury proneness.[39] Arousal levels and stress may also change the neuromuscular response to both intrinsic and extrinsic factors a player would face during competition, possibly leading to injury through changes in muscular tone and stiffness.[40] Clearly, there are a number of factors that can influence whether an

individual may sustain a LEMSK that should also be considered, in addition to the presence or absence of concussion pathology.

The current study is not without limitations. To further elaborate on the unknown accuracy of the NFL's public injury report, like in other sports, players may not accurately reported their injuries to the team medical staff which would result in inaccurate injury reports.[34] Likewise, the medical staff may not report accurately because of the uncertainty medicine provides at times. Not all players respond to injuries the same way and this could be a possible reason for potential reporting errors. Further, the injury reports were not well detailed on the specifics of each injury; we were unable to exclude chronic injuries (e.g., tendinopathy) for this reason. Due to the stringent criteria controls had to have to be matched, controls were not always able to be found for subjects in Aim 2, which lead to exclusion of concussed subjects. Additionally, concussion and/or LEMSK prior to the seasons in question were not ascertained and they may have influenced the outcome of this study.

In conclusion, our study did not show an increased the risk for LEMSK pre or post-concussion in the NFL players, which did not support our hypothesis. Our study did however find increased odds and risk for the non-concussed group to sustain a LEMSK during the pre-concussion time period. Future NFL research should focus on prospective studies tracking injuries in greater detail (e.g. chronic vs. acute) over multiple seasons, to better understand the effects of concussion in NFL players.

REFERENCES

1. Harmon KG, Drezner JA, Gammons M, et al. American Medical Society for Sports Medicine position statement : concussion in sport. *Br J Sports Med*. 2013;47:15-26.
2. Langlois JA, Rutland-Brown W, Wald MM. The epidemiology and impact of traumatic brain injury: a brief overview. *J Head Trauma Rehabil*. 2006;21(5):375-378.
3. O'Connor KL, Baker MM, Dalton SL, Dompier TP, Broglio SP, Kerr ZY. Epidemiology of sport-related concussions in high school athletes: National Athletic Treatment, Injury and Outcomes Network (NATION), 2011–2012 through 2013–2014. *J Athl Train*. 2017;52(3):175-185.
4. McCrea M, Guskiewicz KM, Marshall SW, et al. Acute effects and recovery time following concussion in collegiate football players. *J Am Med Assoc*. 2003;290(19):2556-2563.
5. Guskiewicz KM, Mccrea M, Marshall SW, et al. Cumulative effects associated with recurrent concussion in collegiate football players: The NCAA concussion study. *JAMA*. 2003;290(19):2549-2555.
6. McCrea MM, Barr WB, Guskiewicz K, et al. Standard regression-based methods for measuring recovery after sport-related concussion. *J Int Neuropsychol Soc*. 2005;11:58-69.
7. 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;0:1-10.

8. Broglio SP, Macciocchi SN, Ferrara MS. Sensitivity of the concussion assessment battery. *Neurosurgery*. 2007;60(6):1050-1057.
9. Marinides Z, Galetta KM, Andrews CN, et al. Vision Testing is Additive to the Sideline Assessment of Sports-related Concussion. *Am Acad Neurol*. 2015:25-34.
10. Patricios J, Fuller GW, Ellenbogen R, et al. What are the critical elements of sideline screening that can be used to establish the diagnosis of concussion ? A systematic review. *Br J Sports Med*. 2017;51:1-8.
11. Kamins J, Bigler E, Covassin T, et al. What is the physiological time to recovery after concussion? A systematic review. *Br J Sports Med*. 2017;51(12):935-940.
12. Buckley TA, Oldham JR, Caccese JB. Postural control deficits identify lingering post-concussion neurological deficits. *J Sport Heal Sci*. 2016;5(1):61-69.
13. Howell DR, Osternig LR, Chou LS. Return to activity after concussion affects dual-task gait balance control recovery. *Med Sci Sports Exerc*. 2015;47(4):673-680.
14. Howell DR, Osternig LR, Chou L. Dual-task effect on gait balance control in adolescents with concussion. *YAPMR*. 2013;94(8):1513-1520.
15. Wilkerson GB, Grooms DR, Acocello SN. Neuromechanical considerations for postconcussion musculoskeletal injury risk management. *Curr Sports Med Rep*. 2017;16(6):419-427.

16. Swanik CB, Covassin T, Stearne DJ, Schatz P. The relationship between neurocognitive function and noncontact anterior cruciate ligament injuries. *Am J Sports Med*. 2007;35(6):943-948.

17. Lynall RC, Mauntel TC, Padua DA, Mihalik JP. Acute lower extremity injury rates increase after concussion in college athletes. *Am Coll Sport Med*. 2015:2487-2492.

18. Nordström A, Nordström P, Ekstrand J. Sports-related concussion increases the risk of subsequent injury by about 50% in elite male football players. *Br J Sports Med*. 2014;48:1447-1450.

19. Pietrosimone B, Golightly YM, Mihalik JP, Guskiewicz KM. Concussion frequency associates with musculoskeletal injury in retired NFL players. *Am Coll Sport Med*. 2015;(16):2366-2372.

20. Brooks MA, Peterson K, Biese K, Sanfilippo J, Heiderscheit BC, Bell DR. Concussion increases odds of sustaining a lower extremity musculoskeletal injury after return to play among collegiate athletes. *Am J Sports Med*. 2016;44(3):742-747.

21. Cross M, Kemp S, Smith A, Trewartha G, Stokes K. Professional Rugby Union players have a 60 % greater risk of time loss injury after concussion : a 2-season prospective study of clinical outcomes. *Br J Sports Med*. 2016;50:926-931.

22. Gilbert FC, Burdette GT, Joyner AB, Llewellyn TA, Buckley TA. Association between concussion and lower extremity injuries in collegiate athletes. *Sport Heal A Multidiscip Approach*. 2016;XX(X):1-7.

23. Herman DC, Jones D, Harrison A, et al. Concussion may increase the risk of subsequent lower extremity musculoskeletal injury in collegiate athletes. *Sport Med*. 2016:1-8.

24. Lynall RC, Mauntel TC, Pohlig RT, et al. Lower extremity musculoskeletal injury risk after concussion recovery in high school athletes. *J Athl Train*. 2017;52(11):1062-6050-52.11.22.

25. Ford JH, Giovanello KS, Guskiewicz KM. Episodic memory in former professional football players with a history of concussion: An event-related functional neuroimaging study. *J Neurotrauma*. 2013;30(20):1683-1701.

26. Guskiewicz KM, Marshall SW, Bailes J, et al. Recurrent concussion and risk of depression in retired professional football players. *Med Sci Sports Exerc*. 2007;39(6):903-909.

27. Hanson A, Jolly NA, Peterson J. Safety regulation in professional football: Empirical evidence of intended and unintended consequences. *J Health Econ*. 2017;53:87-99.

28. Kerr ZY, Marshall SW, Guskiewicz KM. Reliability of concussion history in former professional football players. *Med Sci Sports Exerc*. 2012;44(3):377-382.

29. Multani N, Goswami R, Khodadadi M, et al. The association between white-matter tract abnormalities, and neuropsychiatric and cognitive symptoms in retired professional football players with multiple concussions. *J Neurol*. 2016;263(7):1332-1341.

30. Partridge B, Hall W. Conflicts of interest in recommendations to use

computerized neuropsychological tests to manage concussion in professional football codes. *Neuroethics*. 2014;7(1):63-74.

31. Pellman EJ, Viano DC, Tucker AM, et al. Concussion in professional football: Reconstruction of game impacts and injuries. *Neurosurgery*. 2003;53(4):799-814.
32. NFL.com. https://www.NFL.com. Updated in 2018
33. NFL. 2017 Personnel (Injury) Report Policy. 2017:1-9.
34. Craig M. ON THE NFL; Injury report is manipulated misinformation; After falling short of Patriots coach Bill Belichick in tweaking the weekly list , Vikings coach Brad Childress should listen to himself and not believe what he reads. 2006:1-4.
35. Nyberg G, Mossberg KH, Lysholm J, Ba YT. Subsequent traumatic injuries after a concussion in elite ice hockey : A study over 28 years. 2015;2(3):109-112.
36. Hwang S, Ma L, Kawata K, Tierney R, Jeka JJ. Vestibular dysfunction after subconcussive head impact. *J Neurotrauma*. 2017;34(1):8-15.
37. Haran, F J, Tierney, R, Wright, W G, Silter M. Acute changes in postural control after soccer heading. *Int J Sports Med*. 2004;34:350-354.
38. Gysland SM, Mihalik JP, Register-Mihalik JK, Trulock SC, Shields EW, Guskiewicz KM. The relationship between subconcussive impacts and concussion history on clinical measures of neurologic function in collegiate football players. *Ann Biomed Eng*. 2012;40(1):14-22.
39. Malfait F, Hakim AJ, De Paepe A, Grahame R. The genetic basis of the joint

hypermobility syndromes. *Rheumatology*. 2006;45(5):502-507.

40. Yerkes RM, Dodson JD. The relation of strength of stimulus to rapidity of habit-formation in the kitten. *J Anim Behav*. 1908;5(18):459-482.

Appendix A

LITERATURE REVIEW

Introduction

It has been estimated that 1.6-3.8 million sport related concussions occur annually in the US.[2] This number has been speculated to continue to rise due to the increased concussion awareness that has been published more frequently by main stream media. As a result of the increased attention, it's believed that athletes and parents are more likely to report concussions if suspected. It is estimated that more than 2 million high school athletes participate in collision/ contact sports a year,[3] which have also been documented to have the highest incidence rates.[41]

A concussion is defined as "a complex pathophysiological process affecting the brain, induced by biomechanical forces" according to the 5th Berlin consensus statement.[7] Every four years top leaders in the field of concussion come together to discuss new and improved finding in the field and come to a general understanding about current practices, prevention strategies and management of concussion. The international concussion consensus statement is a result of the meeting and is available for all who have an interest in the most current concussion findings. The mechanism of injury is described as; "sport related concussion may be caused either by a direct blow to the head, face, neck or elsewhere on the body with an impulsive force transmitted to the head".[7]

History

The history of concussions date back centuries before it became the term for which we know today. Rhazes (900 A.D.), an Arabic physician was the first to

describe concussion as a separate entity from other TBIs, stating it as an "abnormal physiologic state rather than severe brain injury or a generic descriptor of brain injury".[42] Lanfrancus (1306) who was considered the first "modern" physician to teach that symptoms of a concussions were a temporary paralysis of the brain functions which could rapidly disappear post injury.[42] The next great breakthrough for the modern term occurred when "The Learned Doctor Read" (16th century) stated common signs and symptoms we associate with mTBIs today. The signs and symptoms included a singing of the ears after the wound is received, falling after the blow, swooning for a time, slumbering after the wound is received, dazzling of the eyes, and a giddiness which passes rapidly.[42] As time has evolved so has the understanding about concussions.

Epidemiology

It is estimated that at least 10 million TBIs occur annually worldwide.[2] This is an alarming number which like SRC, it's believed to continue to grow as reporting rates continue to rise. The leading causes for TBI's are falls, motor vehicle crashes, struck by or against events, and assaults.[2] Males are approximately two times more likely to sustain a concussion than females.[2] Ages 0-4 and 15-19 years old were found to be more likely to sustain a TBI than other age group.[2] Also, it's projected that only 7% of all head injuries that go to the hospital are SRC.[43] Since it has been reported that 90% of TBIs are mild,[44] it's not difficult to see this statistic being relatively accurate.

While SRC is only a subcategory of TBI, there is a massive demand to understand more about it as its estimated that annually in the US 3 million children

participate in youth football,[45] and 8 million athletes take part in high school athletics.[3] These are two of the larger groups that research has observed, but collegiate football has been a staple for concussion research in the US throughout the years. In one study done by Kerr et al. incidence rates in collegiate athletics were found to be 0.89, 0.78, and 0.75 per 1,000 athlete exposures (AE) for wrestling, ice hockey and football respectively.[46] In high school athletics rates were observed as 9.21, 6.65, and 6.11 per 10,000 AE for football, boys lacrosse and girls soccer respectively.[3]

Football specifically, is considered a contact/collision sport,[7] which have been shown to consistently produce higher incidence rates.[41] In collegiate football incidence rates have been seen to be between 0.75-1.19 per 1,000 AE.[5,45,46] When rates were broken down by position it was identified that linebackers (0.99 per 1,000 AE), and offensive linemen (0.95 per 1,000 AE) were most likely to sustain a concussion in college football.[5] It was also found in one study that wide receivers and running backs on average sustained the highest magnitudes.[47]

Neurometabolic Cascade

Upon injury a series of cerebral events occur which ultimately lead to transient neurological dysfunction; this is known as the neurometabolic cascade.[48,49] Following the injury non-specific cell depolarization and initiation of action potentials occur because of damage to the neuronal membranes, axons, and potassium channels within individual brain cells.[48,49] This amplifies the ionic flux that occurs next, and increased levels of potassium exit the cell while sodium and calcium enter the cell.[48,49] In order to try and restore homeostasis the cells membrane pumps are

activated and require large amounts of ATP for functioning.[48,49] Hyperglycolysis is the result of the increase in energy needed to power the membrane pumps. Oxidative membrane impairments are also a product of brain trauma which ultimately leads to hyperglycolysis.[48,49] As large amounts of ATP are created through hyperglycolysis lactate is formed as a waste product, and in turn excess lactate is accumulated.[48,49] As a product of the previous steps calcium is accumulated in the mitochondria and ultimately leads to mitochondria dysfunction.[48,49] Because of the mitochondria dysfunction ATP is unable to be produced and leads to cell apoptosis.[48,49]

Signs and Symptoms

Signs and symptoms play an integral role in the diagnosis of concussions. Some signs and symptoms include headache, dizziness, difficulty concentrating, feeling slowed down, sensitivity to light, decreased memory and personality changes.[5,50] The most common symptom seen throughout is headaches, which are seen in approximately 71-95% of sport related concussions.[3,5,51] Some of the least common signs and symptoms seen are loss of consciousness (LOC) and post-traumatic amnesia (PTA) which ranged from 2.2-8.79% and 7.69-24.1% respectively.[3–5,44] Although LOC and PTA are not symptoms that are as likely to be seen post-concussion as headaches are, it is still believed that when these signs are observed the athlete will conversely experience greater cerebral dysfunction.[44] Brown et al. found that significant differences persist between sexes with symptom reporting. Most athlete's experience a resolution of signs and symptoms within 7 days post-injury.[5] Females are 43% more likely than males to reports a symptom at baseline.[52]

Also, post-concussion, total symptom scores were significantly higher in females than males for high school but not college athletes.[52]

Symptom reporting plays a pivotal role in potential injury recognition. It is well known that concussions are underreported, and symptoms are the gateway to this issue. McCrea et al. has reported that only 47.3% of concussions were reported over the course of one high school football season surveying 1,532 athletes.[41] Of those that did not report their symptoms to the appropriate personnel, 66% stated the reason why they didn't was because they "did not think the injury was serious enough".[41] Another study done by Delaney et al. saw that only 23.4% of football players and 19.8% of soccer players realized the symptoms they suffered represented a concussion, which is a result of a lack of concussion education.[53] Other reasons such as perceived coach support,[54] "not wanting to leave the game", and "did not want to let teammates down" are also believed to play a part in the lack of reporting.[41] This directly sheds light on the importance of symptom recognition and reporting.

Diagnosis

When diagnosing a concussion, a multimodal assessment has been shown to be the standard.[9,10,55] It offers a method to objectify findings that directly correlate to cerebral function. Areas that are important to assess have been established as being postural control and cognitive functioning. These tests have been created to specifically examine these aspects of brain functioning to help clinicians identify if dysfunction is present. The most widely used tests are the balance error scoring test (BESS), immediate post-concussion assessment and cognitive test (ImPACT), Standardized Assessment of Concussion (SAC), and Sport Concussion Assessment

Tool, 5th edition (SCAT5).[8,9] While these test can help clinicians to diagnose it has been a common consensus that they should not solely be used to diagnose or determine return to play (RTP).[6,9,10]

BESS

When assessing postural control one of the most commonly used test is the BESS test.[56] The test is able to give clinicians an objective assessment tool that is inexpensive and practical as it only requires a foam pad.[57] Upon examination subjects are asked to stand in three different stances (feet together, single leg, tandem) for twenty seconds each on two different surfaces (firm and foam).[56] Each trial starts with the subject being instructed to close their eyes and place their hands on their hip and the evaluator scores the subject based on the amount of errors made during the trials.[56] Each error is documented as one point, with the maximum number of obtainable points for each trail being ten.[56]

The BESS test has been acknowledged to have a high sensitivity (.34) and a low to moderate specificity (.91) from the time of injury to a week post-injury.[6] It has also been identified in previous studies that there is a learning effect associated with the BESS test.[58] In a study conducted by Finnoff et al., intrarater and interrater reliability were examined. It was determined that while the BESS test as a whole did not provide adequate (≥ 0.75) ICC reliability for interrater (0.57) and intrarater (0.74) scores, some stance positions did produce an adequate ICC reliability.[56] Minimal detectable change (MDC) scores were calculated as well; intrarater (7.3 points) and interrater (9.4 points).[56]

ImPACT

ImPACT is a computerized test that evaluates neurocognitive function and is comprised of six neurocognitive testing categories (verbal memory, visual memory, visual motor speed, reaction time, impulse control, and cognitive efficiency).[8,9] According to one study by Schatz et al. the categories' combined sensitivity and specificity were 81.9% and 89.4%, respectively.[59]

SAC

The SAC test is a question-based tool to examine neurocognitive function, specifically at the time of injury. It's comprised of four domains (orientation, immediate memory, concentration, and delayed recall) that are scored out of a total of 30 possible points.[44] The orientation, concentration, and delayed recall sections are out of five possible points and the immediate memory section is out of 15.[44] The SAC has been found to have high sensitivity (.94) and moderate specificity (76%).[60] McCrea et al. reported that the average baseline measures for collegiate football players was about 27 points, would decrease at time of concussion and then return to baseline by the 48 hour mark.[6] As a result of that trend it has been strongly advised that clinicians should not use this test as a RTP guideline.[44]

SCAT5

The SCAT5 is a multidimensional concussion evaluation tool best used for sideline assessment.[7] A new version of the SCAT is created every four years at the International Conference on Concussion in Sport in Berlin, Germany. The assessment includes a symptom checklist, reaction time, gait/balance assessment, video-observable signs and oculomotor screening.[7] The video-observable signs section is

new to the SCAT testing module and looks to identify a greater number of significant head impacts.[7] While it is stated in the 5th Consensus Statement on Concussion in Sport that the SCAT5 is best used for differentiating concussed from non-concussed individuals at the time of injury; it also states that the symptom checklist section can be very useful during the recovery phase.[7]

King Devick

The King Devick (KD) test involves reading off three separate series of numbers from left to right. Each series of numbers is its own trail which are each timed.[9] When performing the test subjects are instructed to say the numbers displayed as quickly as possible without making any errors. This assessment requires vision acuity and saccades to challenge cognitive processing.[9]

Short Term Recovery

Symptom Recovery

During the recovery phase symptoms can take the longest amount of time to return to baseline values, with most athlete's symptom resolution occurring between 5-7 days.[4,6] McCrea et al. also found that in this study symptoms were most severe during the first 2 days post injury and then steadily declined.[4] While the current standard of care in most concussion protocols around the world involves starting the return to play aspect once an athlete is "asymptomatic"; this is not always a feasible feat.[61] More often than not people naturally are experiencing common signs and symptoms in their everyday lives that are also seen in concussed individuals. Some of these include "feeling tired", having a headache, impatient. These symptoms can be influenced by a number of other factors that is occurring in someone's life.[61] Alla et

al. stated that achieving full resolution of symptoms might not always be obtainable and clinicians should use their clinical expertise and opinion to start return to play progressions rather than waiting for full symptom resolution.[61]

Postural Recovery

In concussed athletes, the BESS test is most commonly used to assess their postural control.[56] McCrea et al. found that most college aged athletes return to baseline in the BESS test at 3-5 days post-injury.[6] It is well documented that this test has a learning effect, so by the time it's performed the third or fourth time the athlete has had multiple days to practice the same stances.[58] For that reason on average athletes performed better at day five post-injury than baseline.[4,6] This is why the general consensus for BESS is it should not be used as a RTP guideline.[4,6,9]

Cognitive Recovery

Cognitive recovery is believed to occur once an athlete has achieved asymptomatic status while and after performing cognitive tasks such as school work, neurocognitive testing, etc. That timeline can vary depending on which indicator is used to help the clinician verify recovery. McCrea et al. found that collegiate athletes returned to baseline measures on the SAC test by the 48 hour mark post-concussion.[6] By day 7 the athletes were well below their baseline values indication a learning effect.[6] When using more challenging methods such as ImPACT it is difficult to identify a recovery timeline because if athletes are already experiencing symptoms the difficulty and variability of the test will most likely exacerbate the symptoms and inevitably delay the RTP process.[55]

Long Term Recovery

CTE

Chronic traumatic encephalopathy (CTE) is a neurodegenerative disease that is often seen later on in individuals' lives that have a history of sustaining numerous head injuries.[62] The exact relationship between SRC and CTE is currently unknown, but is speculated that repetitive head traumas lead to a cascade of neurological events that ultimately lead to this disease.[7,62] The mean age reported of onset was 42.8 years of age, with the average time to onset post-retirement happening at 8 years.[62] Because history has shown that this disease occurs most often post-retirement athletes are now more likely to be spending time with their families and friends more. As a result, families and friends have reported some common signs and symptoms being more irritable, angry, apathetic, showing cognitive difficulties, poor episodic memory and executive functioning.[62] Since there is no current test available to diagnose CTE in individuals while still living; athletes brains are studied post-mortem. The common biomarker discovered in abundance in individuals that were found with this disease is a protein called tau.[62] A different variation of this protein is also commonly found in the brains of individuals that had Alzheimer disease, which is another neurodegenerative disease.[62]

Return to Play (RTP)

Most athletes return to play from SRC between 7-10 days post-injury.[4,63] Before return to sport, the athlete must go through a concussion protocol which simply is a planned routine that intends to stress the brain to simulate activities of daily living and sport.[7] If the athlete at any point in the progression becomes

symptomatic; that activity is stopped and athlete will not resume the progression until 24hrs. He/she has to return to where step in which the symptoms were produced.[7] According to the 5th concussion consensus statement upon diagnosis of a concussion the athlete should rest (approximately 24-48 hours).[7] This step is argued by Buckley et al. study that showed a single day of prescribed cognitive and physical rest in the acute post-concussion period was not effective at reducing recovery time.[64] It is recommended the athlete then starts to perform light exercise and some cognitive activities (i.e.: schoolwork).[64,65] From this point the steps of the progression vary based on the physician's orders and/or the clinician's philosophies but continue to stress the athlete's brain increasingly more during each step until arriving at a full practice.[66] It has been well documented and expressed that not all concussions are the same and time to return to play will vary by athlete.[11]

Subsequent Injury

One of the biggest predictor of injury is previous injury.[67] With concussions, this statement is no different. Guskiewicz et al. reported that collegiate football players with a history of three or more concussions were three times more likely to sustain a concussion than a player with no concussion history; this is known as "the dose response".[5]

Another well-known subsequent injury occurrence is second impact syndrome. It is defined as "an athlete who has sustained an initial head injury, most often a concussion, sustains a second head injury before symptoms associated with the first have fully cleared''.[68] This in some cases can lead to death but the injury is rarely reported worldwide.[68] Upon the second impact the brain is believed to swell

due to the increased cerebral blood flow because of additional trauma to the area.[68] In the same fashion as a subdural or epidural hematoma the swelling has nowhere to perfuse to and will lead to death due to increased intracranial pressure.[68]

It has been well documented that concussion may put athletes at an increased risk to sustain a subsequent MSK injury. Rates have been reported to be as high as 3.29, up to 1 year post concussion.[17,18,20,22,23,69] Research done by Herman et al. is the first study to control both participation (starting status and position played) and identify a clear relationship between concussion and subsequent lower extremity injury.[23] Previous articles were unable to achieve both, with most only able to acquire the latter. It's also important to note that increased subsequent injury rates were found in concussed athletes up to a year post-injury[17,18], and in one study concussed athletes were about 4 times as likely to sustain subsequent injury during 6-12 months post-injury compared to non-concussed athletes.[18] Also, Lynall et al. found that in his study high school athletes were 34% more likely to sustain a subsequent lower extremity after concussion than matched controls without concussion.[24] This is important to note because this study is the first to identify the trend of subsequent injury post-concussion in high school athletes. Cross et al. followed professional rugby players over the course of two seasons and found that players who returned to play from concussion during the same season was at a 60% increased risk to sustain a time-loss injury than those without concussions.[21]

It is understood that there is no definitive explanation as to why increased subsequent injury rate is seen in concussed individuals. Instead, all suggest that further research should explore deeper for definitive reasons whys. The most common

explanation used was concussed athletes may just be more injury prone in general.[17,18,22] Also, the possibility of detraining and not being able to give the body adequate time it needs to return to the condition it was pre-concussion was another suggestion.[20] Previous research has also found that neurophysiological changes in the primary motor cortex beyond the time of return-to-sport from concussion exsits.[69] A majority of the current literature that is published on subsequent injury post-concussion focuses on the collegiate setting and lower extremity injuries exclusively.[17,20,22,23,69] There're very limited amount of studies focusing on professional rates. Pietrosimone et al. looked at the relationship between concussion history and lower extremity injury.[19] The results did show there was an increased odds ratio for reporting more lower extremity injuries as the number of concussions sustained increased. But a big limitation was that this was a retrospective study using questionnaires answered by retired NFL athletes and is unable to give real time relevance.[19] All studies found that concluded athletes are at an increased risk to develop lower extremity injury post-concussion suggests further research needed to be done to identify why this trend occurs.

Conclusion

Being able to identify those who are at an increased risk to sustain a MSK injury because of concussion(s) is very important in the world of athletics. Understanding SRC is the foundation to identifying that risk. Because football is the driving force behind the topic of concussions in the US right now, looking at subsequent injury and concussion in football is a must. There are ample studies out

currently examining subsequent injury rates in the collegiate and high school settings but very little focusing on the NFL. Those that do focus on players that are retired.

As the topic of concussions continue to grow it is inevitable that rule changes in sports at various levels will continue to be seen. Throughout the past decade we have already seen our fair share of rule changes. Youth soccer in the US has banned headers for adolescents. The NCAA has eliminated "two a day" practices from football pre-seasons; partly in an effort to reduce the number of athlete exposures. The NFL has moved up the kickoff to the 35-yard line in an effort to force more touchbacks and less kickoff returns. These are all good advances from a medical standpoint and takes player safety into account first.

The NFL is a multi-billion-dollar corporation that has a major following. Studies that provide proof to potential situations where athletes may be at increased risk to harm themselves would directly affect the NFL's revenue. It's believed this could partially be a reason to why currently the literature cannot capture this setting. It is very important to be able to identify problems in real time to put a stop to trends.

Identifying if a relationship between subsequent injury and concussion in the NFL is very important and would ultimately help add evidence to pursue why we tend to see a relationship between the two categories in lower settings. It's also believed that the NFL holds more "elite" talent and in theory should decrease error because of competition level. In both the high school and collegiate settings; teams might not play opponents that are necessarily of the same talent caliber. When this happens the team with greater talent is at an obvious advantage and could in turn increase the risk of injury for both teams. The same can be said if the roles were reversed.

Overall, there are many topics within concussions that require further extensive research to understand the concept completely. Going forward understanding why neurocognitive impairments seem to extend way beyond the RTP point post-concussion is a pivotal part in eventually understanding subsequent injury after concussion.

REFERENCES

1. Harmon KG, Drezner JA, Gammons M, et al. American Medical Society for Sports Medicine position statement : concussion in sport. *Br J Sports Med*. 2013;47:15-26. doi:10.1136/bjsports-2012-091941.
2. Langlois JA, Rutland-Brown W, Wald MM. The epidemiology and impact of traumatic brain injury: a brief overview. *J Head Trauma Rehabil*. 2006;21(5):375-378. doi:00001199-200609000-00001 [pii].
3. O'Connor KL, Baker MM, Dalton SL, Dompier TP, Broglio SP, Kerr ZY. Epidemiology of sport-related concussions in high school athletes: National Athletic Treatment, Injury and Outcomes Network (NATION), 2011–2012 through 2013–2014. *J Athl Train*. 2017;52(3):175-185. doi:10.4085/1062-6050-52.1.15.
4. McCrea M, Guskiewicz KM, Marshall SW, et al. Acute effects and recovery time following concussion in collegiate football players. *J Am Med Assoc*. 2003;290(19):2556-2563. doi:10.1001/jama.290.19.2556.
5. Guskiewicz KM, Mccrea M, Marshall SW, et al. Cumulative effects associated with recurrent concussion in collegiate football players: The NCAA concussion study. *JAMA*. 2003;290(19):2549-2555.
6. McCrea MM, Barr WB, Guskiewicz K, et al. Standard regression-based methods for measuring recovery after sport-related concussion. *J Int Neuropsychol Soc*. 2005;11:58-69.
7. 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;0:1-10. doi:10.1136/bjsports-2017-097699.

8. Broglio SP, Macciocchi SN, Ferrara MS. Sensitivity of the concussion assessment battery. *Neurosurgery*. 2007;60(6):1050-1057. doi:10.1227/01.NEU.0000255479.90999.C0.

9. Marinides Z, Galetta KM, Andrews CN, et al. Vision Testing is Additive to the Sideline Assessment of Sports-related Concussion. *Am Acad Neurol*. 2015:25-34.

10. Patricios J, Fuller GW, Ellenbogen R, et al. What are the critical elements of sideline screening that can be used to establish the diagnosis of concussion ? A systematic review. *Br J Sports Med*. 2017;51:1-8. doi:10.1136/bjsports-2016-097441.

11. Kamins J, Bigler E, Covassin T, et al. What is the physiological time to recovery after concussion? A systematic review. *Br J Sports Med*. 2017;51(12):935-940. doi:10.1136/bjsports-2016-097464.

12. Buckley TA, Oldham JR, Caccese JB. Postural control deficits identify lingering post-concussion neurological deficits. *J Sport Heal Sci*. 2016;5(1):61-69. doi:10.1016/j.jshs.2016.01.007.

13. Howell DR, Osternig LR, Chou LS. Return to activity after concussion affects dual-task gait balance control recovery. *Med Sci Sports Exerc*. 2015;47(4):673-680. doi:10.1249/MSS.0000000000000462.

14. Howell DR, Osternig LR, Chou L. Dual-Task Effect on Gait Balance Control

in Adolescents With Concussion. *YAPMR*. 2013;94(8):1513-1520. doi:10.1016/j.apmr.2013.04.015.

15. Wilkerson GB, Grooms DR, Acocello SN. Neuromechanical considerations for postconcussion musculoskeletal injury risk management. *Curr Sports Med Rep*. 2017;16(6):419-427. doi:10.1249/JSR.0000000000000430.

16. Swanik CB, Covassin T, Stearne DJ, Schatz P. The Relationship Between Neurocognitive Function and Noncontact Anterior Cruciate Ligament Injuries. *Am J Sports Med*. 2007;35(6):943-948. doi:10.1177/0363546507299532.

17. Lynall RC, Mauntel TC, Padua DA, Mihalik JP. Acute Lower Extremity Injury Rates Increase after Concussion in College Athletes. *Am Coll Sport Med*. 2015:2487-2492. doi:10.1249/MSS.0000000000000716.

18. Nordström A, Nordström P, Ekstrand J. Sports-related concussion increases the risk of subsequent injury by about 50% in elite male football players. *Br J Sports Med*. 2014;48:1447-1450. doi:10.1136/bjsports-2013-093406.

19. Pietrosimone B, Golightly YM, Mihalik JP, Guskiewicz KM. Concussion Frequency Associates with Musculoskeletal Injury in Retired NFL Players. *Am Coll Sport Med*. 2015;(16):2366-2372. doi:10.1249/MSS.0000000000000684.

20. Brooks MA, Peterson K, Biese K, Sanfilippo J, Heiderscheit BC, Bell DR. Concussion Increases Odds of Sustaining a Lower Extremity Musculoskeletal Injury After Return to Play Among Collegiate Athletes. *Am J Sports Med*. 2016;44(3):742-747. doi:10.1177/0363546515622387.

21. Cross M, Kemp S, Smith A, Trewartha G, Stokes K. Professional Rugby Union players have a 60 % greater risk of time loss injury after concussion : a

2-season prospective study of clinical outcomes. *Br J Sports Med*. 2016;50:926-931. doi:10.1136/bjsports-2015-094982.

22. Gilbert FC, Burdette GT, Joyner AB, Llewellyn TA, Buckley TA. Association Between Concussion and Lower Extremity Injuries in Collegiate Athletes. *Sport Heal A Multidiscip Approach*. 2016;XX(X):1-7. doi:10.1177/1941738116666509.
23. Herman DC, Jones D, Harrison A, et al. Concussion May Increase the Risk of Subsequent Lower Extremity Musculoskeletal Injury in Collegiate Athletes. *Sport Med*. 2016:1-8. doi:10.1007/s40279-016-0607-9.
24. Lynall RC, Mauntel TC, Pohlig RT, et al. Lower Extremity Musculoskeletal Injury Risk After Concussion Recovery in High School Athletes. *J Athl Train*. 2017;52(11):1062-6050-52.11.22. doi:10.4085/1062-6050-52.11.22.
25. Ford JH, Giovanello KS, Guskiewicz KM. Episodic Memory in Former Professional Football Players with a History of Concussion: An Event-Related Functional Neuroimaging Study. *J Neurotrauma*. 2013;30(20):1683-1701. doi:10.1089/neu.2012.2535.
26. Guskiewicz KM, Marshall SW, Bailes J, et al. Recurrent concussion and risk of depression in retired professional football players. *Med Sci Sports Exerc*. 2007;39(6):903-909. doi:10.1249/mss.0b013e3180383da5.
27. Hanson A, Jolly NA, Peterson J. Safety regulation in professional football: Empirical evidence of intended and unintended consequences. *J Health Econ*. 2017;53:87-99. doi:10.1016/j.jhealeco.2017.01.004.
28. Kerr ZY, Marshall SW, Guskiewicz KM. Reliability of concussion history in

former professional football players. *Med Sci Sports Exerc*. 2012;44(3):377-382. doi:10.1249/MSS.0b013e31823240f2.

29. Multani N, Goswami R, Khodadadi M, et al. The association between white-matter tract abnormalities, and neuropsychiatric and cognitive symptoms in retired professional football players with multiple concussions. *J Neurol*. 2016;263(7):1332-1341. doi:10.1007/s00415-016-8141-0.
30. Partridge B, Hall W. Conflicts of interest in recommendations to use computerized neuropsychological tests to manage concussion in professional football codes. *Neuroethics*. 2014;7(1):63-74. doi:10.1007/s12152-013-9182-z.
31. Pellman EJ, Viano DC, Tucker AM, et al. Concussion in professional football: Reconstruction of game impacts and injuries. *Neurosurgery*. 2003;53(4):799-814. doi:10.1227/01.NEU.0000083559.68424.3F.
32. NFL.com. NFL.com.
33. NFL. 2017 Personnel (Injury) Report Policy. 2017:1-9.
34. Craig M. ON THE NFL; Injury report is manipulated misinformation; After falling short of Patriots coach Bill Belichick in tweaking the weekly list , Vikings coach Brad Childress should listen to himself and not believe what he reads. 2006:1-4.
35. Nyberg G, Mossberg KH, Lysholm J, Ba YT. Subsequent traumatic injuries after a concussion in elite ice hockey : A study over 28 years. 2015;2(3):109-112.
36. Hwang S, Ma L, Kawata K, Tierney R, Jeka JJ. Vestibular Dysfunction after Subconcussive Head Impact. *J Neurotrauma*. 2017;34(1):8-15.

doi:10.1089/neu.2015.4238.

37. Haran, F J, Tierney, R, Wright, W G, Silter M. Acute Changes in Postural Control After Soccer Heading. *Int J Sports Med*. 2004;34:350-354. doi:10.1136/bjsm.2003.004887.

38. Gysland SM, Mihalik JP, Register-Mihalik JK, Trulock SC, Shields EW, Guskiewicz KM. The Relationship Between Subconcussive Impacts and Concussion History on Clinical Measures of Neurologic Function in Collegiate Football Players. *Ann Biomed Eng*. 2012;40(1):14-22. doi:10.1007/s10439-011-0421-3.

39. Malfait F, Hakim AJ, De Paepe A, Grahame R. The genetic basis of the joint hypermobility syndromes. *Rheumatology*. 2006;45(5):502-507. doi:10.1093/rheumatology/kei268.

40. Yerkes RM, Dodson JD. The relation of strength of stimulus to rapidity of habit-formation in the kitten. *J Anim Behav*. 1908;5(18):459-482. doi:10.1037/h0073415.

41. McCrea M, Hammeke T, Olsen G, Leo P, Guskiewicz K. Unreported Concussion in High School Football Players. *Clin J Sport Med*. 2004;14(1):13-17.

42. McCrory, Paul R., Berkovic SF. Concussion: The history of clinical and pathophysiological concepts and misconceptions. *J Am Heart Assoc*. 2001;57(12):2283-2289.

43. Quayle KS, Holmes JF, Kupperman N. Epidemiology of Blunt Head Trauma in Children in U . S . Emergency Departments. *N Engl J Med*.

2014;371(20):1945-1947. doi:10.1056/NEJMc1410735.

44. McCrea M, Kelly JP, Randolph C, Cisler R, Berger L. Immediate Neurocognitive Effects of Concussion. *Neurosurgery*. 2002;50(5):1032-1042.

45. Houck Z, Asken B, Bauer R, Pothast J, Michaudet C, Clugston J. Epidemiology of Sport-Related Concussion in an NCAA Division I Football Bowl Subdivision Sample. 2015:2269-2275. doi:10.1177/0363546516645070.

46. Kerr ZY, Roos KG, Dalton SL, Broglio SP, Marshall SW, Dompier TP. Epidemiologic Measures for Quantifying the Incidence of Concussion in National Collegiate Athletic Association Sports. *J Athl Train*. 2017;52(3):167-174. doi:10.4085/1062-6050-51.6.05.

47. Mihalik JP, Bell DR, Ed M, Marshall SW, Ph D. Measurement of Head Impacts in Collegiate Football Players: An Investigation of Positional and Event-Type Differences. *Neurosurgery*. 2007;61(3):1229-1235. doi:10.1227/01.NEU.0000280147.37163.30.

48. Giza CC, Hovda DA. The New Neurometabolic Cascade of Concussion. *Neurosurgery*. 2014;75(4):24-33. doi:10.1227/NEU.0000000000000505.

49. Giza CC, Hovda DA. The Neurometabolic Cascade of Concussion. 2001;36(3):228-235.

50. Wojtys EM, Hovda D, Landry G, et al. Current concepts. Concussion in sports. *Am J Sports Med*. 1999;27(5):676-687. http://www.ncbi.nlm.nih.gov/pubmed/10496590.

51. Feddermann-Demont N, Echemendia RJ, Schneider KJ, et al. What domains of clinical function should be assessed after sport-related concussion ? A

systematic review. 2017:903-918. doi:10.1136/bjsports-2016-097403.

52. Brown DA, Elsass JA, Miller AJ, Reed LE, Reneker JC. Differences in Symptom Reporting Between Males and Females at Baseline and After a Sports-Related Concussion: A Systematic Review and Meta-Analysis. *Sport Med*. 2015;45(7):1027-1040. doi:10.1007/s40279-015-0335-6.

53. Delaney JS, Lacroix VJ, Leclerc S, Johnston KM. Concussions Among University Football and Soccer Players. *Clin J Sport Med*. 2002;12:331-338.

54. Baugh CM, Daneshvar DH, Stern RA. Perceived Coach Support and Concussion Differences between Freshmen and Non- Freshmen College Football Players. *J Law, Med Ethics*. 2014:314-322.

55. Alsalaheen B, Stockdale K, Pechumer D, Broglio SP, Marchetti GF. A Comparative Meta-Analysis of the Effects of Concussion on a Computerized Neurocognitive Test and Self-Reported Symptoms. *J Athl Train*. 2017;52(7). doi:10.4085/1062-6050-52.7.05.

56. Finnoff JT, Peterson VJ, Hollman JH, Smith J. Intrarater and Interrater Reliability of the Balance Error Scoring System. *Am Acad Phys Med ans Rehabil*. 2009;1(1):234-243. doi:10.1080/1091367X.2011.568368.

57. Riemann BL, Guskiewicz KM, Shields EW. Relationship between Clinical and Forceplate Measures of Postural Stability. *J Sport Rehabil*. 1999;8(2):71-82. doi:10.1123/jsr.8.2.71.

58. Valovich McLeod TC, Perrin DH, Guskiewicz KM, Shultz SJ, Diamond R, Gansneder BM. Serial administration of clinical concussion assessments and learning effects in healthy young athletes. *Clin J Sport Med*. 2004;14(5):287-

295. doi:10.1097/00042752-200409000-00007.

59. Schatz P, Pardini JE, Lovell MR, Collins MW, Podell K. Sensitivity and specificity of the ImPACT Test Battery for concussion in athletes. *Arch Clin Neuropsychol*. 2006;21(1):91-99. doi:10.1016/j.acn.2005.08.001.

60. Barr WB, McCrea M. Sensitivity and specificity of standardized neurocognitive testing immediately following sports concussion. *J Int Neuropsychol Soc*. 2001;7(6):S1355617701766052. doi:10.1017/S1355617701766052.

61. Alla S, Sullivan SJ, Mccrory P. Defining asymptomatic status following sports concussion : fact or fallacy ? 2012:562-569. doi:10.1136/bjsm.2010.081299.

62. Gavett, Brandon E., Stern, Robert A., McKee AC. Chronic Traumatic Encephalopathy: A Potential Late Effect of Sport-Related Concussive and Subconcussive Head Trauma. *Clin Sports Med*. 2011;30(1):179-188.

63. Guskiewicz KM, Ross SE, Marshall SW. Postural Stability and Neuropsychological Deficits after Concussion in Collegiate Athletes. *J Athl Train*. 2001;36(3):263-273.

64. Buckley TA, Munkasy BA, Clouse BP. Acute Cognitive and Physical Rest May Not Improve Concussion Recovery Time. *J Head Trauma Rehabil*. 2016;31(4):233-241. doi:10.1002/aur.1474.Replication.

65. Majerske CW, Mihalik JP, Ren D, et al. Concussion in Sports: Postconcussive Activity Levels, Symptoms, and Neurocognitive Performance. *J Athl Train*. 2008;43(3):265-274.

66. Sye G, Sullivan SJ, Mccrory P. High school rugby players' understanding of

concussion and return to play guidelines. *Br J Sports Med*. 2006;40:1003-1005. doi:10.1136/bjsm.2005.020511.

67. Schultz AB, Taaffe DR, Blackburn M, et al. Musculoskeletal screening as a predictor of seasonal injury in elite Olympic class sailors. *J Sci Med Sport*. 2016;19(11):903-909. doi:10.1016/j.jsams.2016.02.011.

68. McCrory P, Davis G, Makdissi M. Second Impact Syndrome or Cerebral Swelling after Sporting Head Injury. *Am Coll Sport Med*. 2012;11(1):21-23.

69. DuBose DF, Herman DC, Jones DL, et al. Lower Extremity Stiffness Changes following Concussion in Collegiate Football Players. *Med Sci Sports Exerc*. 2016:167-172. doi:10.1249/MSS.0000000000001067.