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original research

Osteoarthritis Prevalence in Retired National Football League Players With a History of Concussion and Lower Extremity Injury

Robert C. Lynall, PhD, ATC*†; Brian Pietrosimone, PhD, ATC†‡§; Zachary Y. Kerr, PhD, MPH¶; Timothy C. Mauntel, PhD, ATC†§; Jason P. Mihalik, PhD, CAT(C), ATC*†; Kevin M. Guskiewicz, PhD, ATC, FNATA, FACSM*†

*Matthew Gfeller Sport-Related Traumatic Brain Injury Research Center, ‡Neuromuscular Research Laboratory, §Sports Medicine Research Laboratory, ¶Department of Exercise and Sport Science, and †Curriculum in Human Movement Science, Department of Allied Health Sciences, University of North Carolina at Chapel Hill. Dr Lynall is now at the Concussion Research Laboratory, Department of Kinesiology, The University of Georgia, Athens. Dr Mauntel is now at Walter Reed National Military Medical Center, Department of Orthopaedics, Bethesda, MD.

Context: Dynamic balance deficits have been described postconcussion, even after athletes return to play. Lower extremity (LE) musculoskeletal injury rates increase for up to 1 year after concussion, but the long-term musculoskeletal implications of concussion are unclear.

Objective: To (1) examine the association of concussion and LE injury histories with osteoarthritis (OA) prevalence in retired National Football League players and (2) examine the association of concussion and LE injury histories with OA prevalence in those ≤55 years of age.

Design: Case-control study.

Setting: Survey.

Patients or Other Participants: We administered the Health Survey of Retired National Football League Players, which collects information about demographics, OA, LE injury, and concussion history.

Main Outcome Measure(s): Twelve discrete categories were created based on concussion and LE injury history, ranging from 0 concussions and 0 LE injuries (referent group) to 3+ concussions and 2+ LE injuries. Binomial regression analysis modeled lifetime OA prevalence. Covariates were body mass index, age at the time of the survey, and total years playing professional football.

Results: Complete data were available for 2696 participants. Lifetime OA prevalence was smallest in the referent group (21.1%) and largest in the 3+ concussion and 2+ LE group (50.6%; 2.5 times the referent; 95% confidence interval [CI] = 2.1, 3.1). Participants in all concussion groups (1, 2, 3+) who reported a history of 0 LE injuries had a greater OA prevalence than the referent group. When participants were stratified by age, the ≤55 years of age, 3+ concussions, and 2+ LE injuries group prevalence ratio (3.6; 95% CI = 2.7, 5.2) was larger than that of the >55 years of age, 3+ concussions, and 2+ LE injuries group (1.8; 95% CI = 1.3, 2.4) compared with the respective referent groups.

Conclusions: Concussion with or without a history of LE injury may be an important moderator of OA. Future researchers should seek to better understand the mechanisms that influence the association among concussion, LE injury, and OA.

Key Words: mild traumatic brain injury, joint degeneration, knee arthritis

Key Points

- Retired players who described 1 or more concussions, with or without a history of traumatic lower extremity injury, reported a higher prevalence of osteoarthritis than those with no history of concussion.
- The association was strongest among those 55 years old or younger.
- Players who sustained multiple concussions had a higher prevalence of osteoarthritis than those with a history of 0 or 1 concussion.

Concussion leads to acute neurocognitive and balance deficits[1–3] that have been reported to resolve within 7 to 10 days,[4] although numerous investigators[5–8] have described impairments lasting beyond this time period and well after the athlete has returned to full activity. Gait measures[6,9–11] and virtual-reality postural-control assessments[12] indicate lingering impairments in dynamic postural control and locomotor navigation in previously concussed individuals. Persistent postconcussive neuromuscular-control alterations have been hypothesized to contribute to aberrant movement biomechanics or the inability to adequately react to a potentially injurious stimulus, which may increase the risk of a lower extremity (LE) musculoskeletal injury.[13–17] Furthermore, aberrant movement biomechanics and joint loading have been implicated as potential risk factors in the development of chronic disability and joint degeneration.[18,19] Unfortunately, few researchers have explored how concussion, alone or combined with acute LE injury, may be related to the long-term deterioration of joint health.

Aberrant gait biomechanics related to altered neuromuscular control have been implicated in the incidence and progression of osteoarthritis (OA) in the LE joints.[18,19] Additionally, traumatic joint injury is among the most predictive factors related to the risk of developing OA.[20–22] Long-term changes in biomechanics, influenced by the neuromuscular consequences of joint injury and biochemical alterations in joint homeostasis, likely hasten the onset and progression of LE joint OA.[23] Furthermore, concussion increases the risk of sustaining an acute LE joint injury[13,14] and may contribute to gross changes in LE biomechanics during gait.[6,9,24] Gait alterations have been observed when athletes return to play after concussion, but how long these alterations persist after brain injury is unknown. These neuromuscular-control alterations include greater interjoint coordination variability,[25] increased propulsive and braking forces during gait termination,[9] and greater center-of-mass medial-lateral displacement[6] than in nonconcussed control participants. Just as long-term changes in biomechanics may influence OA progression after LE injury, long-term changes in biomechanics due to concussion may also influence OA development. Thus, it is reasonable to suggest that people who have sustained both a concussion and an LE joint injury may be at higher risk of developing LE OA. Individuals who sustain a concussion and an LE injury early in life, both of which may result in the development of aberrant biomechanics, may be at higher risk of developing OA at an early age.

Currently, there is a dearth of information on the association between concussion and LE injury and the development of chronic musculoskeletal diseases such as OA. Understanding how concussion and LE musculoskeletal injury may collectively influence the progression to OA will be important for developing strategies to improve long-term health after athletic injury. Previous investigators have described an increased prevalence of early-onset OA among retired National Football League (NFL) players[26] and an association between reported history of concussion and LE injuries.[15] Retired professional football players represent a unique and available cohort of athletes who frequently sustain concussions and LE injuries, which allowed us to investigate this association.

Our primary purpose was to examine the association of concussion and LE injury histories with the prevalence of OA in retired NFL players. Our secondary purpose was to examine the association of concussion and LE injury histories with the prevalence of OA in those 55 years of age and younger. We hypothesized that (1) retired NFL players who had sustained more (2 or 3+) concussions during their careers would have a higher prevalence of OA than retired NFL players who had sustained no or fewer concussions (0 or 1) and (2) the prevalence of OA in those aged 55 and younger would be higher among those who had sustained more concussions (2 or 3+) as opposed to those who had sustained no or fewer concussions (0 or 1).

METHODS

Participants

Participants were part of the Health Survey of Retired NFL Players.[27] This ongoing survey, administered by the Center for the Study of Retired Athletes, collects information from retired NFL players to evaluate multiple aspects of health and wellbeing. The initial survey, completed in 2001, was mailed to 3647 former players who retired between 1930 and 2001. The same survey was also sent to new retirees (those who had not yet received the survey) in 2006 (n = 1272), 2009 (n = 876), 2011 (n = 374), and 2012 (n = 364). Thus, the survey was sent to 6533 retired players in total. The survey was mailed to each player 3 times, and any player who did not respond to the paper survey was contacted via phone. Participant consent was implied based on completion of the paper survey or given verbally during phone interviews. Our university's institutional review board approved the study protocol.

Procedures

The Health Survey of Retired NFL Players. This 13-page paper survey collected information about player demographics, playing history (eg, total years of football played, years of football played at various competitive levels, positions played), general medical history, joint-injury history, and overall health status. For each category, information was requested about the participant's status during and after his football career.

Concussions. We collected information about all concussions sustained during the participant's professional football career. First, each participant was asked, "Did you sustain any concussions during your professional career?" If the answer was *yes*, the participant was asked how many concussions he sustained during his NFL career.

Lower Extremity Musculoskeletal Injury. We asked for the frequency of specific LE injuries sustained throughout the participant's NFL career. Each player was specifically asked to indicate how many total injuries he had incurred in the following categories: (1) medial collateral ligament tear, (2) lateral collateral ligament tear, (3) anterior cruciate ligament tear, (4) posterior cruciate ligament tear, (5) meniscal tear, (6) hamstrings/quadriceps rupture, (7) calf/Achilles rupture, (8) ankle ligament rupture, and (9) ankle/foot fracture.[15] The injuries sustained across the 9 categories were summed.

Osteoarthritis. Participants were asked to respond *yes* or *no* to the following question: "Have you ever been told by a physician or health professional that you had/have osteoarthritis/degenerative arthritis?"

Covariates. Self-reported height and mass during the last year each participant played in the NFL were used to compute body mass index (BMI). Additionally, we calculated the total number of years each participant played NFL football and his age at the time the survey was completed.

Statistical Analysis

Coding of Variables. The main outcome, lifetime prevalence of OA, was retained as a dichotomous variable (*yes*/*no*). We used 2 main exposures, based on the numbers of NFL career concussions and LE injuries. Our first outcome was the number of LE injuries, categorized as 0, 1, or 2+. Our second outcome was the combination of the number of career concussions and LE injuries. Concussion count was categorized as 0, 1, 2, or 3+. This categorization of concussion history followed previous publications[15,27] detailing differences in long-term outcomes based on increasing concussion counts. From

Table 1. Group Size and Demographic Information

Concussions, No.	Lower Extremity Injuries, No.	Participants, No.	Mean ± SD		
				National Football League Participation	
			Age, y	Body Mass Index	Years Played
0	0	427	55.1 ± 16.4	29.1 ± 3.6	5.1 ± 3.7
	1	236	51.0 ± 13.6	29.3 ± 3.2	5.5 ± 3.6
	2+	419	51.6 ± 14.2	30.0 ± 3.8	6.0 ± 3.5
1	0	135	56.0 ± 15.8	28.7 ± 3.5	6.0 ± 3.5
	1	114	54.2 ± 14.8	29.8 ± 3.7	6.2 ± 3.3
	2+	273	51.2 ± 13.0	29.4 ± 3.4	6.2 ± 3.3
2	0	94	55.3 ± 14.9	29.7 ± 3.7	6.2 ± 3.7
	1	80	51.1 ± 12.4	29.3 ± 3.6	7.1 ± 3.4
	2+	266	52.0 ± 12.5	29.5 ± 3.4	7.2 ± 3.4
3+	0	110	50.0 ± 11.7	29.0 ± 3.7	7.3 ± 3.6
	1	101	50.6 ± 12.0	29.2 ± 3.9	7.5 ± 3.6
	2+	441	50.0 ± 11.7	30.1 ± 3.7	8.2 ± 3.3

the concussion and LE variables, 12 discrete categories were created, ranging from individuals with 0 concussions and 0 LE injuries to individuals with 3+ concussions and 2+ LE injuries. The first covariate, BMI while playing, was computed from self-reported height and weight. For the remaining covariates, total NFL years played and current age, we used the raw data.

Models. Binomial regression models were used to model the lifetime prevalence of OA. Prevalence ratios (PRs) compared the lifetime prevalence of OA among groups. Multivariate models controlled for BMI while playing, total years played, and current age. All binomial regression models used Poisson residuals and robust variance estimation.[28–30] First, we calculated prevalence ratios that used the categorized LE injury count (0, 1, or 2+); 0 LE injuries was the referent group. Second, we calculated PRs that used the second 12-category exposure, which also considered concussion count; the 0 concussion/0 LE group was the referent group. A PR higher than 1 implied that the group had a higher prevalence of OA compared with the referent group, whereas a PR of less than 1 implied that the group had a lower prevalence of OA compared with the referent group. Any PR with a corresponding 95% confidence interval (CI) that did not include 1.00 was considered statistically significant.

Age Stratification. The previously described methods were used to explore any age-related difference in the association of concussion and LE injury history with lifetime prevalence of OA. For this analysis, we stratified respondents by age: ≤55 years and >55 years. We used 55 years as this is the median age at which knee OA is diagnosed in the general US population.[31] For the study, we defined players who completed a survey and indicated they were diagnosed with OA at or before age 55 as individuals with *early-onset OA*.

RESULTS

Sample

Completed surveys were received from 3226 retired NFL players (49.3% response rate). We excluded any participants who did not report a complete concussion history (n = 234) and those who did not report data for all of our injury variables (n = 51) or for a history of OA (n = 8). Finally, we excluded participants with information missing for any of our 3 covariates (n = 237), resulting in a final data set of 2696 participants.

Descriptive Statistics

Overall, 978 participants (36.3%) reported experiencing OA during their lifetime. Of our sample, 40.1% (n = 1082) reported no concussion history, whereas 19.4% (n = 522), 16.3% (n = 440), and 24.2% (n = 652) reported a history of 1, 2, or 3+ concussions, respectively. A total of 15.8% (n = 427) reported 0 concussions and 0 LE injuries, whereas 16.4% (n = 441) reported 3+ concussions and 2+ LE injuries. The mean age of our sample at the time of the survey was 60.8 ± 11.2 years (age range = 24–95 years), although a slight majority of participants were ≤55 years of age (57.9%). Group demographics along with descriptive statistics for each covariate are detailed in Table 1.

Binomial Regression Models

The lifetime prevalence of OA increased with the number of LE injuries, ranging from 26.1% for 0 to 29.6% for 1 and 44.4% for 2+ LE injuries (Table 2). Although no difference was found in the lifetime prevalence of OA between the 1 and 0 LE injury groups (PR = 1.1; 95% CI = 1.0, 1.4), the lifetime prevalence of OA in the 2+ LE injuries group was 70% higher than that of the 0 LE injuries group (PR = 1.7; 95% CI = 1.5, 1.9). Results were retained in the multivariate model.

Univariate and multivariate PRs using the 12-category exposure variable are described in Table 3. The lifetime prevalence of OA was smallest in the 0 concussion/0 LE injuries group (21.1%) and largest in the 3+ concussions/2+ LE injuries group (50.6%). Compared with the 0 concussions/0 LE injuries group, the lifetime prevalence of OA was significantly higher in the majority of the other concussion/LE injury groups. Results were retained in the multivariate models. For example, controlling for BMI while playing, NFL years played, and current age, the lifetime prevalence of OA in the 3+ concussions/2+ LE injuries group was 2.5 times (95% CI = 2.1, 3.1) that of the 0 concussions/0 LE injuries group. In addition, multivariate analyses showed that compared with the 0 concussions/0 LE injuries group, the lifetime prevalence of OA was higher among all concussion groups in the absence of LE

Table 2. Lifetime Prevalence of Osteoarthritis and Prevalence Ratios Within Each Group of Lower Extremity Injury Counts, Overall and by Age

Group	Lower Extremity Injuries, No.	Prevalence of Osteoarthritis, % (No.)	Prevalence Ratio (95% Confidence Interval)	
			Univariate Analysis	Multivariate Analysis[a]
Overall	0	26.1 (200)	1.0 (referent)	1.0 (referent)
	1	29.6 (157)	1.1 (1.0, 1.4)	1.2 (1.0, 1.4)
	2+	44.4 (621)	1.7 (1.5, 1.9)[b]	1.7 (1.5, 2.0)[b]
Age ≤55 y	0	19.6 (78)	1.0 (referent)	1.0 (referent)
	1	26.4 (84)	1.4 (1.0, 1.8)[b]	1.3 (1.0, 1.7)[b]
	2+	43.4 (367)	2.2 (1.8, 2.8)[b]	2.1 (1.7, 2.6)[b]
Age >55 y	0	33.2 (122)	1.0 (referent)	1.0 (referent)
	1	34.3 (73)	1.0 (0.8, 1.3)	1.0 (0.8, 1.3)
	2+	45.9 (254)	1.4 (1.2, 1.6)[b]	1.4 (1.2, 1.6)[b]

[a] Controls for age at the time of the survey, body mass index while playing, and total years played in the National Football League.
[b] The lower value for the 95% confidence interval was actually above 1.0 but was rounded to 1.0.

injuries (1 concussion/0 LE injuries group PR = 1.4; 95% CI = 1.0, 1.9; 2 concussions/0 LE injuries group PR = 1.7; 95% CI = 1.2, 2.4; 3+ concussions/0 LE injuries group PR = 1.7; 95% CI = 1.2, 2.3; Figure).

The lifetime prevalence of OA among the ≤55 years group (33.9%) was smaller than that of the >55 years group (39.6%). In participants ≤55 years, the lifetime OA prevalence was higher in the 1 LE injury (26.4%; PR = 1.4; 95% CI = 1.0, 1.8) and 2+ LE injuries (43.4%; PR = 2.2; 95% CI = 1.8, 2.8; Table 2) groups compared with the 0 LE injuries group (19.5%). Lifetime OA prevalence in the >55 years group did not differ between the 1 LE injury and 0 LE injuries groups (34.3% versus 33.2%; PR = 1.0; 95% CI = 0.8, 1.3), yet the lifetime prevalence of OA in the 2+ LE injuries group was 40% higher than that of the 0 LE injuries group (PR = 1.4; 95% CI = 1.2, 1.6). Results were retained in the multivariate models.

In analyses using the 12-category exposure variable, the smaller lifetime prevalence of OA among participants ≤55 years was consistent among participants in each concussion/LE injury stratification. All univariate and multivariate PRs stratified by age are described in Table 4. Compared with the overall analyses, PRs for participants ≤55 years were larger, whereas PRs for participants >55 years, although statistically significant in some cases, were not as large as those in the ≤55 years group.

DISCUSSION

Retired NFL players with a history of 3 or more concussions and 2 or more LE injuries reported the highest prevalence of OA. Concussion plus LE injury may be associated with OA. This effect is best seen in younger individuals (≤55 years) compared with older individuals (>55 years). Therefore, a concussion history in NFL players who also reported sustaining an LE joint injury may increase the risk of developing OA early in life (≤55 years). Our results also suggest a concussion dose-response relationship, as those who sustained multiple concussions demonstrated a higher prevalence of OA than those who received 0 or 1 concussion. To a lesser degree, we observed an LE injury dose-response relationship as well, with those who incurred 2 or more injuries reporting a higher prevalence of OA.

As seen in previous research,[20–22] we observed a dose-response relationship in which larger counts of LE injuries were associated with an increased lifetime prevalence of OA. Findings were similar within strata based on age. However, when also considering concussion history, we noted that the relative risk of OA associated with concussion existed even in the absence of self-reported LE injuries. Effect estimates were attenuated among the older age stratum (ie, >55 years). However, this was partially attributable to the attenuation of PRs due to the higher lifetime prevalence of OA in the referent group for

Table 3. Lifetime Prevalence of Osteoarthritis and Prevalence Ratios Within Each Group of Concussion and Lower Extremity Injury Counts

Concussions, No.	Lower Extremity Injuries, No.	Prevalence of Osteoarthritis, % (No.)	Prevalence Ratio (95% Confidence Interval)	
			Univariate Analysis	Multivariate Analysis[a]
0	0	21.1 (90)	1.0 (referent)	1.0 (referent)
	1	24.2 (57)	1.1 (0.9, 1.5)	1.2 (0.9, 1.6)
	2+	42.5 (178)	2.0 (1.6, 2.5)[b]	2.1 (1.7, 2.5)[b]
1	0	29.6 (40)	1.4 (1.0, 1.9)[b]	1.4 (1.0, 1.9)[b]
	1	32.5 (37)	1.5 (1.1, 2.1)[b]	1.5 (1.1, 2.1)[b]
	2+	37.7 (103)	1.8 (1.4, 2.3)[b]	1.9 (1.5, 2.4)[b]
2	0	36.2 (34)	1.7 (1.2, 2.4)[b]	1.7 (1.2, 2.4)[b]
	1	28.8 (23)	1.4 (0.9, 2.0)	1.5 (1.0, 2.1)
	2+	44.0 (117)	2.1 (1.7, 2.6)[b]	2.2 (1.8, 2.8)[b]
3+	0	32.7 (36)	1.6 (1.1, 2.1)[b]	1.7 (1.2, 2.3)[b]
	1	39.6 (40)	1.9 (1.4, 2.5)[b]	2.0 (1.5, 2.8)[b]
	2+	50.6 (223)	2.5 (2.0, 3.1)[b]	2.5 (2.1, 3.1)[b]

[a] Controls for age at the time of the survey, body mass index while playing, and total years played in the National Football League.
[b] The lower value for the 95% confidence interval was actually above 1.0 but was rounded to 1.0.

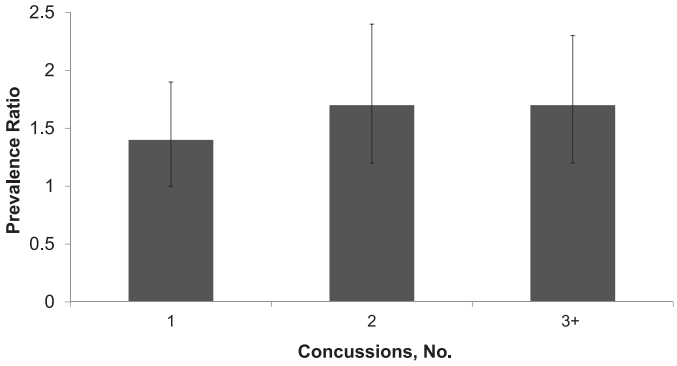

Figure. Prevalence ratios for all respondents in each concussion group (1, 2, or 3+ concussions) who reported a history of 0 lower extremity injuries as compared with the referent group (0 concussions, 0 lower extremity injuries). The prevalence of osteoarthritis in all concussion groups was statistically higher than that in the referent group. Error bars represent 95% confidence intervals for the prevalence ratios.

the older age stratum (29.2%) compared with the younger age stratum (13.8%). Although these results highlight the limitations of PRs, which are bounded by a 0% to 100% range, they also suggest that concussion history, LE injury history, and age are associated with lifetime prevalence of OA.

Before considering possible explanations for the connection between concussion and OA, it is important to acknowledge the limitations of our work. Our outcomes were all survey based, and responses could be influenced by recall bias. A previous study[15] of data from the same survey demonstrated moderate to good reliability in retired NFL players for self-reporting concussion (weighted Cohen $k = 0.59$) and LE injury history (weighted Cohen $k \geq 0.43$). Unfortunately, the Health Survey of Retired NFL Players does not ask participants to specify which joints were affected by OA. Although OA commonly develops in joints of the upper extremity (eg, the hands), hand OA rarely develops in isolation, as most patients with hand OA also have symptomatic OA of the knee or lumbar spine.[32] It is critical to acknowledge that all data were collected retrospectively and based on survey responses from retired NFL players only. We were unable to directly establish a causal association between concussion history and OA or to ascertain the exact time from injury (either concussion or LE injury) to the onset of OA. We also lacked data on concussions sustained before the participants' careers in the NFL, history of LE surgery, and BMI throughout their careers, all of which could influence OA progression. Previous authors[13–17] have linked concussion with LE injury but were unable to explore long-term outcomes. There is also evidence of a strong link between LE injury and OA.[33] We sought to build on these publications by investigating the moderating effect of concussion on OA. Acknowledging the lack of causality in our report, we believe it is important to discuss several underlying constructs that may have contributed to the increased prevalence of OA in retired football players who experienced multiple concussions. Exploring these hypotheses further will help researchers and clinicians to develop intervention strategies targeting the underlying factors contributing to the increased prevalence of OA in retired NFL players who sustained concussions during their professional careers.

Aberrant gait biomechanics after concussion may influence OA development. Specifically, gait velocity was

Table 4. Prevalence and Prevalence Ratios of Lifetime Prevalence of Osteoarthritis Within Each Group of Concussion and Lower Extremity Injury Counts Stratified by Age

Age Group	Concussions, No.	Lower Extremity Injuries, No.	Prevalence of Osteoarthritis, % (No.)	Prevalence Ratio (95% Confidence Interval)	
				Univariate Analysis	Multivariate Analysis[a]
≤55 y	0	0	13.8 (31)	1.0 (referent)	1.0 (referent)
		1	22.9 (33)	1.7 (1.1, 2.6)[b]	1.6 (1.1, 2.6)[b]
		2+	40.3 (98)	2.9 (2.0, 4.2)[b]	2.9 (2.0, 4.1)[b]
	1	0	17.9 (12)	1.3 (0.7, 2.4)	1.3 (0.7, 2.4)
		1	21.8 (12)	1.6 (0.9, 2.9)	1.6 (0.9, 2.8)
		2+	36.8 (60)	2.7 (1.8, 3.9)[b]	2.7 (1.8, 3.9)[b]
	2	0	40.5 (17)	2.9 (1.8, 4.8)[b]	2.9 (1.7, 4.7)[b]
		1	26.0 (13)	1.9 (1.1, 3.3)[b]	1.8 (1.1, 3.2)[b]
		2+	41.8 (66)	3.0 (2.1, 4.4)[b]	3.0 (2.0, 4.3)[b]
	3+	0	27.7 (18)	2.0 (1.2, 3.4)[b]	2.1 (1.3, 3.5)[b]
		1	37.7 (26)	2.7 (1.8, 4.3)[b]	2.7 (1.8, 4.2)[b]
		2+	50.9 (143)	3.7 (2.6, 5.2)[b]	3.6 (2.7, 5.2)[b]
>55 y	0	0	29.2 (59)	1.0 (referent)	1.0 (referent)
		1	26.1 (24)	0.9 (0.6, 1.3)	0.9 (0.6, 1.4)
		2+	45.5 (80)	1.6 (1.2, 2.0)[b]	1.6 (1.2, 2.1)[b]
	1	0	41.2 (28)	1.4 (1.0, 2.0)	1.4 (1.0, 2.0)[b]
		1	42.4 (25)	1.5 (1.0, 2.1)[b]	1.5 (1.0, 2.1)[b]
		2+	39.1 (43)	1.3 (1.0, 1.8)	1.4 (1.0, 1.9)[b]
	2	0	32.7 (17)	1.1 (0.7, 1.7)	1.2 (0.7, 1.8)
		1	33.3 (10)	1.1 (0.7, 2.0)	1.2 (0.7, 2.1)
		2+	47.2 (51)	1.6 (1.2, 2.2)[b]	1.7 (1.3, 2.3)[b]
	3+	0	40.0 (18)	1.4 (0.9, 2.1)	1.5 (1.0, 2.4)
		1	43.8 (14)	1.5 (1.0, 2.3)	1.5 (1.0, 2.4)
		2+	50.0 (80)	1.7 (1.3, 2.2)[b]	1.8 (1.3, 2.4)[b]

[a] Controls for age at the time of the survey, body mass index while playing, and total years played in the National Football League.
[b] The lower value for the 95% confidence interval was actually above 1.0 but was rounded to 1.0.

reduced immediately after concussion,[34–36] whereas frontal-[37] and sagittal-plane center-of-mass range of motion increased.[35] Motor-control strategy alterations appear postconcussion, as evidenced by altered peak propulsive and braking forces for up to 10 days after the injury.[9] Traditional clinical measures indicate that postural control appears to return to normal within several days of injury,[1,38] but other, more in-depth measures suggest that deficits persist beyond the athlete's return to play postconcussion.[12,39] Together, these results imply that neuromuscular control is affected acutely and these changes may persist after the return to participation postconcussion. These findings are important, as aberrant gait biomechanics, which may be linked to changes in neuromuscular control, have been implicated in the progression of OA.[18,19] Although this is still a hypothesis, an athlete who sustains multiple concussions and LE injuries at a relatively young age may alter his or her gait biomechanics. Our data suggest that concussion may be a more significant moderator of OA in those aged 55 and younger. Gait changes resulting from concussion may contribute to the increased prevalence of early-onset OA in retired NFL players. Linking potential biomechanical changes after concussion with aberrant gait biomechanics after LE injury may provide important information as clinicians and researchers seek effective interventions.

Appropriate motor control requires the efficient integration of multiple systems within a short period of time. Sensory and visual cues prompt a cognitive response. The primary motor cortex, in conjunction with several other brain regions, is responsible for planning and executing movement. Motor-evoked potentials (MEPs) provide useful information about the integrity of the motor cortex and descending motor pathways.[40] When an external stimulus is applied to the motor cortex, surface electromyography can measure outcomes related to MEPs.[40–42] Several groups[43–46] have demonstrated decreased excitability of the primary motor cortex after concussion. Specifically, compared with healthy control participants, concussed participants displayed less intracortical facilitation,[44] increased intracortical inhibition,[43] increased MEP latency, and decreased MEP amplitude.[45,46] Researchers[14,15] have hypothesized that these cortical disruptions may increase the risk of musculoskeletal injury postconcussion. These disruptions may linger for years after the concussion,[47] resulting in subtle yet important changes in functional movement. Although gross alterations in motor function are not common postconcussion, subtle persistent cortical-activation changes may alter movement biomechanics, increasing the risk of degenerative conditions such as OA. As this remains a hypothesis, future authors should prospectively investigate MEP alterations after concussion to determine a cortical recovery timeline.

Our findings cause concern for the long-term physical health of retired NFL players who have sustained multiple concussions and LE injuries. Despite our results, participation in sports provides numerous physical,[48] psychological,[49] and cognitive[50] benefits. We believe our findings underscore the need to identify cortical impairments or other mechanisms that may lead to the increased prevalence of OA in retired football players who have sustained concussions. Our cohort of retired NFL players, as well as other current and former athletes, may benefit from long-term interventions aimed at correcting aberrant biomechanics resulting from a combination of LE injuries and concussions. We believe our results may help to further identify athletes who may be at increased risk of long-term degenerative conditions such as OA. Future investigators should seek to determine the direct cause of the increased risk of musculoskeletal injury after concussion, along with the associated mechanisms by which concussion appears to moderate OA prevalence in combination with a history of joint injury. If the underlying pathophysiology behind the increased risk can be identified, intervention strategies can be developed to mitigate the increased risk of injury and increased prevalence of OA after concussion and LE injury.

CONCLUSIONS

Our results suggest that concussion may be associated with OA in retired NFL players. Participants with a history of concussion (1, 2, or 3+ injuries) and no LE injuries reported a significantly higher prevalence of OA than the referent group. This association appears to be strongest among retired players $\leq$55 years old. The number of concussions sustained may also be important, as players with multiple concussions demonstrated a higher prevalence of OA than those with 0 or 1 concussion. More work is needed to identify the underlying cortical mechanisms that may contribute to OA progression after concussion.

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Address correspondence to Robert C. Lynall, PhD, ATC, Concussion Research Laboratory, Department of Kinesiology, The University of Georgia, 330 River Road, Athens, GA 30677. Address e-mail to rlynall@uga.edu.