

Estimated age of first exposure to American football and outcome from concussion

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Abstract

Objective

To examine the association between estimated age at first exposure (eAFE) to American football and clinical measures throughout recovery following concussion.

Methods

Participants were recruited across 30 colleges and universities as part of the National Collegiate Athletic Association (NCAA)–Department of Defense Concussion Assessment, Research and Education Consortium. There were 294 NCAA American football players (age 19 ± 1 years) evaluated 24–48 hours following concussion with valid baseline data and 327 (age 19 ± 1 years) evaluated at the time they were asymptomatic with valid baseline data. Participants sustained a medically diagnosed concussion between baseline testing and postconcussion assessments. Outcome measures included the number of days until asymptomatic, Immediate Post-Concussion Assessment and Cognitive Testing (ImPACT) composite scores, Balance Error Scoring System (BESS) total score, and Brief Symptom Inventory 18 (BSI-18) subscores. The eAFE was defined as participant's age at the time of assessment minus self-reported number of years playing football.

Results

In unadjusted regression models, younger eAFE was associated with lower (worse) ImPACT Visual Motor Speed ($R^2 = 0.031$, $p = 0.012$) at 24–48 hours following injury and lower (better) BSI-18 Somatization subscores ($R^2 = 0.014$, $p = 0.038$) when the athletes were asymptomatic. The effect sizes were very small. The eAFE was not associated with the number of days until asymptomatic, other ImPACT composite scores, BESS total score, or other BSI-18 subscores.

Conclusion

Earlier eAFE to American football was not associated with longer symptom recovery, worse balance, worse cognitive performance, or greater psychological distress following concussion. In these NCAA football players, longer duration of exposure to football during childhood and adolescence appears to be unrelated to clinical recovery following concussion.

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Glossary

AFE = age at first exposure; **BESS** = Balance Error Scoring System; **BIC** = Bayesian information criterion; **BSI** = Brief Symptom Inventory; **CARE** = Concussion Assessment, Research and Education; **eAFE** = estimated age at first exposure; **GSI** = Global Severity Index; **ImPACT** = Immediate Post-Concussion Assessment and Cognitive Testing; **LOC** = loss of consciousness; **NCAA** = National Collegiate Athletic Association; **NFL** = National Football League; **PTA** = posttraumatic amnesia; **RGA** = retrograde amnesia; **SES** = socioeconomic status.

Earlier age at first exposure (AFE) to American football has been associated with worse neuropsychological test performance,¹ microstructural differences in the corpus callosum measured by diffusion tensor imaging,² and smaller volumes measured in the thalamus³ among retired National Football League (NFL) players recruited from the Diagnosing and Evaluating Traumatic Encephalopathy Using Clinical Tests (DETECT) study. In contrast, a separate study of retired NFL players found no association between earlier estimated AFE (eAFE) and neurocognitive, neurologic, or neuro-radiologic outcomes.⁴ A recently published large-scale survey study of retired NFL players (n = 3,506) found that greater number of seasons playing professional football was associated with greater risk for later-in-life mental health and cognitive difficulties; however, there was no association between AFE to football and increased risk for later in life depression, anxiety, or cognitive impairment.⁵ Moreover, 5 large-scale cross-sectional cohort studies of current collegiate and high school American football players and other contact sport athletes found no association between eAFE and baseline preseason neurocognitive function.^{6–10}

It has been hypothesized that exposure to football at an early age might interfere with neurodevelopmental maturation and increase vulnerability to neurologic consequences later in life.^{1,11} If so, it is possible that neurobiological changes from early participation in American football, if present, are sufficiently compensated for in otherwise healthy individuals, but when faced with injury (i.e., concussion), earlier eAFE may associate with longer symptom recovery, worse cognitive performance, or greater psychological distress. Cognitive reserve is a theory suggesting some individuals may have more efficient utilization of brain networks, which attempts to explain why there does not appear to be a strong and immutable relationship between degree of brain neuropathology and clinical manifestations in many older adults.^{12,13} This theory was originally proposed to explain individuals' resistance and resilience to the clinical manifestations of Alzheimer disease.^{14,15} A preliminary study suggested cognitive reserve may forestall clinical manifestations of chronic traumatic encephalopathy.¹⁶ Cognitive reserve is thought to be influenced by both genetic^{12,13} and lifestyle factors.^{17–19} Earlier eAFE to American football may gradually reduce cognitive reserve via exposure to repetitive head impacts. If so, a concussion within the context of reduced cognitive reserve could result in greater clinical manifestations (e.g., greater symptoms or worse cognitive performance).

This study examined the association between eAFE to American football and clinical measures throughout recovery following concussion. The null hypothesis for the present study is that earlier eAFE to American football is not associated with worse cognitive performance, greater psychiatric distress, or longer recovery time following concussion.

Methods

Standard protocol approvals, registrations, and patient consents

All participants provided written informed consent that was approved by their local institution and the US Army Human Research Protection Office. This study was conducted in accordance with the standards of ethics outlined in the Declaration of Helsinki.

Participants

National Collegiate Athletic Association (NCAA) American football players were recruited across 30 colleges and universities as part of the NCAA–Department of Defense Concussion Assessment, Research and Education (CARE) Consortium. A detailed description of the CARE Consortium methods is provided elsewhere.²⁰ Inclusion criteria included male American football players who sustained a sport-related concussion and completed a multimodal concussion assessment battery at baseline, postinjury (i.e., within 6 hours), within 24–48 hours of injury, and at the time they reported being asymptomatic. For individuals who sustained multiple concussions throughout their participation in the CARE Consortium, only their first concussion was included in analyses. According to the CARE Consortium, “a concussion is defined as a change in brain function following a force to the head, which may be accompanied by temporary loss of consciousness, but is identified in awake individuals with measures of neurologic and cognitive dysfunction. Identification, assessment, and diagnosis of concussive events were completed by the research and medical staff at each site.”^{20,21}

Instrumentation

Baseline and postconcussion assessments from the Clinical Study Core were used in the analyses,^{20,22,23} including Immediate Post-Concussion Assessment and Cognitive Testing (ImPACT), the Balance Error Scoring System (BESS), and the Brief Symptom Inventory 18 (BSI-18). Although many participants completed the entire battery of assessments, there were individuals with missing tests/time points. Each

assessment has been described in detail elsewhere but is described briefly below.^{20,22,23}

Immediate Post-Concussion Assessment and Cognitive Testing

The ImPACT is the most widely used computerized neuropsychological test for concussion evaluation.²⁴ The outcome measures are 4 composite scores, including (1) verbal memory, (2) visual memory, (3) visual motor speed, (4) reaction time, and the Post-Concussion Symptom Scale. Higher verbal memory, visual memory, and visual motor speed scores reflect better neurocognitive performance, and lower reaction time scores and symptom total scores are better.

Balance Error Scoring System

The BESS is commonly used following concussion for the assessment of acute and subacute postural control.²⁵ The outcome measure is the number of errors recorded by the administering clinician (range for scoring is 0–60, where a lower score reflects better balance).

Brief Symptom Inventory 18

The BSI-18 is a brief symptom inventory that requires respondents to rate their level of distress over the past 7 days. The BSI-18 provides subscores on 3 dimensions (somatization, depression, and anxiety); the range of possible scores for each is 0–24, for which lower is better. In addition, a composite score, the Global Severity Index (GSI), is calculated on a range of 0–72, for which lower is better.

Demographic and medical history

The Demographics and Personal and Family Medical History unique case report form captures demographic data, information on current and previous sport history, concussion history, and preexisting personal and family medical history. These data were used to compute eAFE (see below). In addition, race (White or other), ethnicity (Hispanic or Latino, not Hispanic or Latino, not reported), socioeconomic status (see below), concussion history (yes/no), neurodevelopmental history (i.e., yes/no for either learning disability or attention-deficit/hyperactivity disorder), psychiatric history (yes/no), history of depression (yes/no), migraine history (yes/no), headache history (i.e., nonmigraine headaches, yes/no), setting (competition or practice/training), loss of consciousness (LOC; yes/no), posttraumatic amnesia (PTA; yes/no), retrograde amnesia (RGA; yes/no), delayed removal from play or additional head trauma (yes/no), and initial postinjury (<6 hours) symptom severity were considered as potential predictors in the adjusted models (table 1).²⁶

Estimated age at first exposure

The eAFE was defined as the participant's age at the time of baseline assessment minus the number of years the participant reported playing football. The minimum eAFE for this study was 5 years because this is the youngest age for Pop Warner youth football. Participants with reported eAFE prior to age 5 were excluded. The eAFE in our study assumes

that participants played football continuously from when they first began. In addition, participants who reported their athletic eligibility year, instead of their total number of years playing football, or participants who did not provide their years of sport participation were excluded.

Socioeconomic status

A participant's parental socioeconomic status (SES) was calculated using the Hollingshead Four Factor Index of Social Status.^{9,27} Each participant reported maternal and paternal education and occupation, which were assigned numeric values: education (0–7) and occupation (0–9).²⁷ A score of 0 corresponded to unemployed/unknown/unreported, which may be a result of a participant being a member of a single parent household. Education and occupation scores were then multiplied by 3 and 5, respectively, and summed to obtain a composite score (range 0–66) for each parent separately and then averaged to derive a single SES score per athlete.²⁷ Predefined SES groups were applied based on published cutoffs: low (0–19), middle-low (20–29), middle (30–39), middle-high (40–54), and high (55–66).²⁷

Statistical analyses

For all outcome measures (i.e., ImPACT composite scores, BESS total score, and BSI-18 subscores), we computed change scores (i.e., postinjury score – baseline score). Positive change scores reflect an increase from baseline to postinjury and negative change scores reflect a decrease from baseline to postinjury. In the context of our analyses, lower ImPACT verbal memory, visual memory, and visual motor speed composite scores are worse. Higher ImPACT reaction time composite scores, PCSS symptom severity scores, BSI-18 subscores, and BESS scores are worse. We also examined the association between eAFE to American football and the time to become asymptomatic. Being asymptomatic was determined through self-reported symptom evaluations. We ran unadjusted regression models to examine the association between eAFE to American football and clinical measures throughout recovery following concussion. The dependent variable for each analysis was the change score or the time to become asymptomatic and the independent variable was eAFE (continuous). Then we ran adjusted regression models. The best model was chosen among all possible models based on the Bayesian information criterion (BIC). Minimizing the BIC returns the best fitting model, while penalizing the complexity of the model. Potential independent variables included race, socioeconomic status, concussion history, migraine history, setting, LOC, PTA, RGA, delayed removal from play or additional head trauma, and initial postinjury symptom severity. Other terms were removed due to low frequency (table 1). After computing the best fit model, we added eAFE to the model. The gain in R^2 with eAFE represents the added value of including eAFE in the model. Effect size was interpreted by Cohen f^2 of 0.02 as a small effect, 0.15 as a medium effect, and 0.35 as a large effect. Significance was defined a priori as $p < 0.05$. Although we computed 21 regression models, we did not apply a Bonferroni correction

Table 1 Participant preinjury and injury characteristics and distributional statistics for change scores

	24–48 hours	Asymptomatic
Age, y	19 ± 1	19 ± 1
eAFE, y	10 ± 3	10 ± 3
Race: White	121 (41.2)	137 (41.9)
Race: other	173 (58.8)	190 (58.1)
Ethnicity: Hispanic or Latino	14 (4.8)	13 (4)
Ethnicity: not Hispanic or Latino	203 (69)	234 (71.6)
Ethnicity: not reported	77 (26.2)	80 (24.5)
SES: high	79 (27.1)	89 (27.2)
SES: mid-high	87 (29.9)	101 (30.9)
SES: middle	48 (16.5)	54 (16.5)
SES: mid-low	27 (9.3)	32 (9.8)
SES: low	50 (17.2)	48 (14.7)
Positive concussion history	138 (46.9)	148 (45.3)
Positive ADHD	28 (9.5)	36 (11)
Positive LD	10 (3.4)	15 (4.6)
Positive neurodevelopmental history	34 (11.6)	44 (13.5)
Positive psychiatric history	5 (1.7)	4 (1.2)
Positive depression history	3 (1)	3 (0.9)
Positive migraine history	27 (9.2)	28 (8.6)
Positive headache history	4 (1.4)	4 (1.2)
Setting: competition	78 (26.5)	88 (26.9)
Setting: practice/training	216 (73.5)	239 (73.1)
LOC	18 (6.1)	21 (6.4)
PTA	49 (16.7)	57 (17.4)
RGA	27 (9.2)	30 (9.2)
Delayed removal from play or additional head trauma	120 (40.8)	129 (39.4)
Postinjury symptom severity	29 ± 21	28 ± 21
BESS difference	1 ± 8	–1 ± 7
BSI-18 somatization difference	1 ± 3	0 ± 2
BSI-18 anxiety difference	1 ± 2	0 ± 2
BSI-18 depression difference	1 ± 3	0 ± 2
BSI-18 GSI difference	3 ± 7	–1 ± 5
ImPACT RT difference	0.04 ± 0.18	–0.01 ± 0.11
ImPACT symptom severity difference	15 ± 19	–1 ± 7

Table 1 Participant preinjury and injury characteristics and distributional statistics for change scores (*continued*)

	24–48 hours	Asymptomatic
ImPACT verbal memory difference	–0.3 ± 13.8	4.5 ± 11.3
ImPACT visual memory difference	–4 ± 13.3	–1 ± 11.6
ImPACT visual motor speed difference	–1 ± 6.9	1.6 ± 5.7

Abbreviations: ADHD = attention-deficit/hyperactivity disorder; BESS = Balance Error Scoring System; BSI = Brief Symptom Inventory; eAFE = estimated age at first exposure; GSI = Global Severity Index; ImPACT = Immediate Post-Concussion Assessment and Cognitive Testing; LD = learning disability; LOC = loss of consciousness; PTA = posttraumatic amnesia; RGA = retrograde amnesia; RT = reaction time; SES = socioeconomic status. Continuous variables (i.e., age, eAFE, postinjury symptom severity, and change scores) are represented as mean ± SD. Nominal variables are presented as frequency (i.e., n [%]).

(e.g., $0.05/21 = 0.002$) to maximize our chances of refuting the null hypothesis. All analyses were conducted using JMP version 14 (SAS Institute, Cary, NC).

Data availability

CARE Consortium data are available through FITBIR (fitbir.nih.gov/).

Results

There were 294 NCAA American football players (age 19 ± 1 years) who were evaluated 24–48 hours following sport-related concussion and had valid baseline data and postinjury symptom severity scores and 327 (age = 19 ± 1 years) who were evaluated at the time they were asymptomatic and had valid baseline data and postinjury symptom severity scores (table 1).

Results of unadjusted regression models suggested that younger eAFE was associated with lower (worse) ImPACT visual motor speed change scores ($R^2 = 0.031$, $p = 0.012$) at 24–48 hours and lower (better) BSI-18 somatization subscore change scores ($R^2 = 0.014$, $p = 0.038$) when the athletes were asymptomatic (table 2 and figures 1 and 2). eAFE was not associated with number of days until becoming asymptomatic, other ImPACT composite scores, BESS total score, or other BSI-18 subscores (table 2). Results of the adjusted models were the same as the results of the unadjusted models (table 3). Effect sizes were small (i.e., ImPACT visual motor speed composite score, $f^2 = 0.036$; BSI-18 somatization subscore, $f^2 = 0.014$).

Measures included in the best fit models are provided in table 4. Most models were weak, accounting for less than 5% of the variance in the change scores (table 3). At 24–48 hours, postinjury symptom severity was the best predictor of BSI-18 somatization, BSI-18 anxiety, BSI-18 GSI, and

Table 2 Results of the unadjusted regression models for change scores

	24–48 hours			Asymptomatic		
	N	R ²	P Value	N	R ²	P Value
BESS difference	267	0.000	0.804	298	0.006	0.190
BSI-18 somatization difference	274	0.001	0.636	301	0.014	0.038
BSI-18 anxiety difference	274	0.000	0.720	301	0.010	0.080
BSI-18 depression difference	274	0.004	0.279	301	0.000	0.797
BSI-18 GSI difference	274	0.001	0.692	301	0.010	0.090
ImPACT verbal memory difference	204	0.019	0.051	235	0.006	0.223
ImPACT visual memory difference	204	0.000	0.951	235	0.003	0.405
ImPACT visual motor speed difference	204	0.031	0.012	229	0.000	0.796
ImPACT RT difference	204	0.000	0.760	234	0.004	0.365
ImPACT symptom severity difference	198	0.005	0.304	223	0.001	0.694
Days until asymptomatic				322	0.000	0.791

Abbreviations: BESS = Balance Error Scoring System; BSI = Brief Symptom Inventory; GSI = Global Severity Index; ImPACT = Immediate Post-Concussion Assessment and Cognitive Testing; RT = reaction time.

ImPACT Symptom Severity change scores ($R^2 = 0.222$, $R^2 = 0.157$, $R^2 = 0.278$, $R^2 = 0.425$, respectively).

Discussion

This is the first study to examine whether NCAA American football players who have longer lifetime exposure to the sport have worse clinical outcome following concussion. The short- and long-term effects of repetitive neurotrauma on brain health are a fundamental concern facing the sports medicine community and there has been speculation that playing football during critical periods of neurodevelopment (i.e., before age 12) may be associated with greater later-life cognitive, behavioral, and mood dysfunction.^{2,5} There is a steadily growing body of literature, however, that is not supporting this theory.^{4–10,28,29} For example, Caccese et al.⁷ assessed neurocognitive performance in over 4,000 NCAA American football players and reported that earlier eAFE was not associated with worse baseline neurocognitive performance. Roberts et al.⁵ surveyed 3,506 former NFL players and found no association between AFE and increased risk for later-in-life anxiety, depression, or self-reported cognitive impairment. The present study examined whether potential

latent differences in brain health in those with longer lifetime exposure to football might manifest in greater or prolonged symptoms, cognitive deficits, or balance problems following a concussion. Our findings suggest that younger eAFE was associated with worse ImPACT visual motor speed at 24–48 hours and better BSI-18 somatization subscore at the time athletes were asymptomatic. The effect sizes for these conflicting findings were small, and had we corrected for multiple comparisons, the effects would not have been significant. These findings do not support the theory that repetitive head trauma associated with playing football during childhood and adolescence may disrupt developmental processes and decrease cognitive reserve, resulting in worse outcomes following concussion during the collegiate years.

The extent to which preinjury and injury severity characteristics are associated with clinical recovery from concussion is not well understood, and the literature is mixed.²⁶ Based on small measure estimates for preinjury and injury severity characteristics, our findings suggest these factors have marginal practical relevance. Most research to date supports an association between greater acute/subacute symptoms and clinical recovery from concussion.²⁶ Our findings suggest that postinjury (<6 hours) symptom severity was a strong predictor of change scores (i.e., BSI-18 somatization, $R^2 = 0.222$; anxiety, $R^2 = 0.157$; GSI, $R^2 = 0.278$; ImPACT symptom severity, $R^2 = 0.425$) at 24–48 hours, but not at the time the athletes were asymptomatic. Greater acute symptom severity (i.e., symptom severity reported <6 hours following sport-related concussion) may be associated with worse clinical concussion outcomes 24–48 hours following sport-related concussion.

Figure 1 Association between estimated age at first exposure (eAFE) and Immediate Post-Concussion Assessment and Cognitive Testing (ImPACT) visual motor speed change score (i.e., 24–48 hours after injury – baseline) with linear fit in red

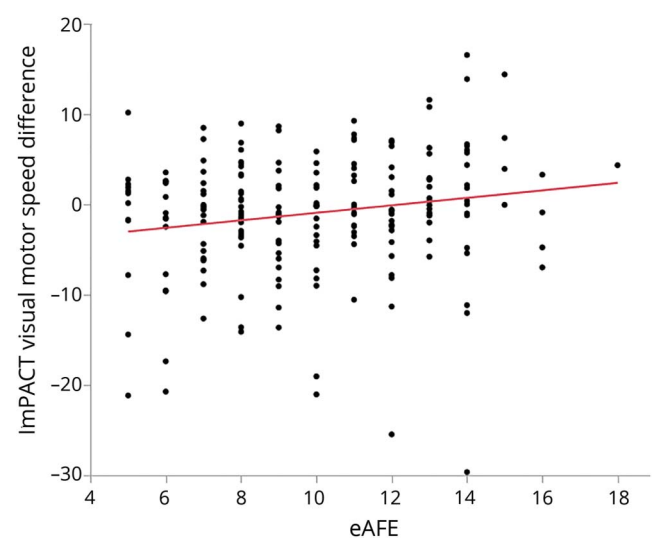
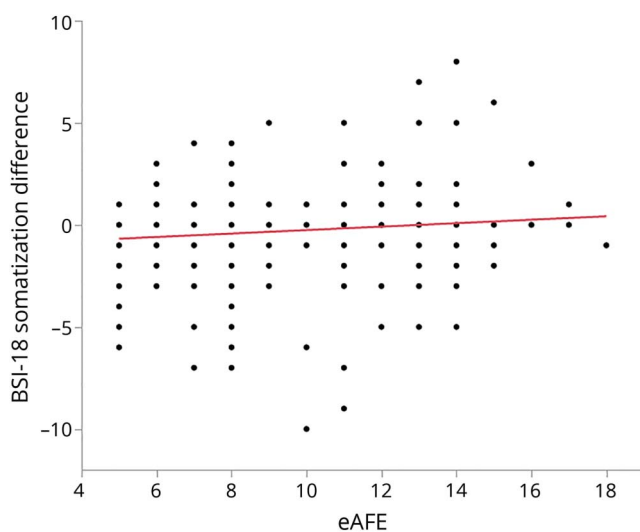


Figure 2 Association between estimated age at first exposure (eAFE) and Brief Symptom Inventory (BSI-18) somatization change score (i.e., asymptomatic – baseline) with linear fit in red



Our findings should be interpreted in the context of this study's limitations. First, like previous work, the eAFE was based on athletes' self-reporting and assumed that athletes played continuously from when they first began. Specifically, student athletes were asked to report the number of years of

participation in American football. However, until we can prospectively examine the effects of a lifetime of exposure to repetitive blows to the head on clinical outcomes, these self-reported data appear to be our best option and are essential to research.³⁰ Second, our cohort was limited to American tackle football, but concussions are common among other contact and collision sports (e.g., ice hockey, soccer, and lacrosse)^{31,32} and the current findings may not extend to those populations, but should be further investigated. Finally, we did not account for exposure to other contact sports or player position because this may change over time and many individuals play multiple positions, especially at earlier ages; however, these may be important unmeasured variables and should be investigated further.

Longer exposure to playing American tackle football during childhood and adolescence was not associated with worse clinical outcomes following concussion in collegiate student athletes and earlier eAFE was not associated with longer concussion recovery. This study adds to a body of research with current collegiate athletes^{6–10} and former professional athletes^{4,5,28,29} showing no association between more years of playing football and worse cognitive or psychological health.

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Table 3 Results of the adjusted models for change scores

	24–48 hours				Asymptomatic			
	N	Best fit, ^a R ²	With eAFE, ^b R ²	eAFE effect, <i>p</i> Value	N	Best fit, ^a R ²	With eAFE, ^b R ²	eAFE effect, <i>p</i> Value
BESS difference	267	0.027	0.027	0.896	294	0.038	0.043	0.194
BSI-18 somatization difference	274	0.254	0.256	0.518	301	0.010	0.024	0.040
BSI-18 anxiety difference	270	0.161	0.161	0.951	298	0.021	0.032	0.064
BSI-18 depression difference	271	0.024	0.027	0.318	300	0.015	0.015	0.685
BSI-18 GSI difference	267	0.342	0.343	0.751	298	0.021	0.032	0.070
ImPACT verbal memory difference	204	0.041	0.056	0.081	234	0.028	0.035	0.211
ImPACT visual memory difference	202	0.012	0.013	0.596	226	0.042	0.045	0.362
ImPACT visual motor speed difference	202	0.128	0.151	0.023	223	0.013	0.014	0.803
ImPACT RT difference	204	0.068	0.068	0.999	232	0.011	0.015	0.344
ImPACT symptom severity difference	198	0.430	0.430	0.807	222	0.016	0.017	0.650
Days until asymptomatic					317	0.015	0.015	0.985

Abbreviations: BESS = Balance Error Scoring System; BSI = Brief Symptom Inventory; eAFE = estimated age at first exposure; GSI = Global Severity Index; ImPACT = Immediate Post-Concussion Assessment and Cognitive Testing; RT = reaction time.

^a R² of the model selected from all possible models to best represent the data.

^b R² of the model with eAFE added.

Table 4 Measure estimates, standard errors (SEs), and test results for measures input into best fit models

Outcome	Measure	Estimate	SE	t	p Value
24–48 hours					
BESS difference	Postinjury symptom severity	0.063	0.023	2.69	0.008
BSI-18 somatization difference	Postinjury symptom severity	0.069	0.008	9.15	<0.001
	Race: other	0.560	0.164	3.40	0.001
BSI-18 anxiety difference	Postinjury symptom severity	0.042	0.006	7.02	<0.001
	Positive neurodevelopmental history	−0.229	0.203	−1.13	0.260
BSI-18 depression difference	LOC	1.001	0.390	2.56	0.011
BSI-18 GSI difference	Postinjury symptom severity	0.185	0.017	10.82	<0.001
	Race: other	0.975	0.370	2.64	0.009
	Positive concussion history	−0.433	0.366	−1.18	0.238
	SES: middle, middle-high, high, middle-low, and low	−1.044	0.368	−2.84	0.005
	LOC	1.850	0.750	2.47	0.014
ImPACT verbal memory difference	Postinjury symptom severity	−0.129	0.044	−2.94	0.004
ImPACT visual memory difference	SES: all-low	−1.856	1.213	−1.53	0.128
ImPACT visual motor speed difference	Postinjury symptom severity	−0.104	0.021	−4.97	<0.001
	Positive neurodevelopmental history	−1.965	0.766	−2.56	0.011
ImPACT RT difference	Postinjury symptom severity	0.002	0.001	3.84	<0.001
ImPACT symptom severity difference	Postinjury symptom severity	0.563	0.046	12.13	<0.001
	SES: high-middle-low	2.205	1.763	1.25	0.212
Days until asymptomatic	Postinjury symptom severity	−0.038	0.030	−1.27	0.206
	Positive migraine history	−0.630	1.125	−0.56	0.576
	SES: all-low	−1.314	0.879	−1.50	0.136
Asymptomatic					
BESS difference	LOC	−2.075	0.861	−2.41	0.017
	PTA	1.617	0.568	2.85	0.005
BSI-18 somatization difference	Race: other	0.213	0.122	1.75	0.081
BSI-18 anxiety difference	PTA	0.305	0.121	2.52	0.012
BSI-18 depression difference	SES: high, middle-low, middle-high, middle, and low	−0.242	0.115	−2.10	0.036
BSI-18 GSI difference	PTA	0.855	0.339	2.52	0.012
ImPACT verbal memory difference	Positive migraine history	−3.124	1.209	−2.58	0.010
ImPACT visual memory difference	Positive neurodevelopmental history	2.749	1.159	2.37	0.019
	SES: high, middle-high, middle-low, middle, and low	−1.727	0.801	−2.16	0.032
ImPACT visual motor speed difference	Positive neurodevelopmental history	−0.837	0.486	−1.72	0.087
ImPACT RT difference	SES: middle-high, low-high, middle, and middle-low	−0.011	0.007	−1.61	0.109
ImPACT symptom severity difference	Positive migraine history	−1.226	0.654	−1.87	0.062

Abbreviations: BESS = Balance Error Scoring System; BSI = Brief Symptom Inventory; GSI = Global Severity Index; ImPACT = Immediate Post-Concussion Assessment and Cognitive Testing; LOC = loss of consciousness; PTA = posttraumatic amnesia; RT = reaction time; SES = socioeconomic status.

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Name	Location	Contribution
Jaclyn B. Caccese, PhD	The Ohio State University College of Medicine, Columbus	Designed and conceptualized study, analyzed the data, drafted the manuscript for intellectual content
Zac Houck, MS	University of Florida, Gainesville	Designed and conceptualized study, revised the manuscript for intellectual content
Thomas W. Kaminski, PhD, ATC	University of Delaware, Newark	Designed and conceptualized study, revised the manuscript for intellectual content
James R. Clugston, MD, MS, CAQSM	University of Florida, Gainesville	Designed and conceptualized study, revised the manuscript for intellectual content
Grant L. Iverson, PhD	Harvard Medical School, Boston; Spaulding Rehabilitation Hospital, Charlestown; Spaulding Research Institute, Charlestown; MassGeneral Hospital for Children, Boston; Home Base, A Red Sox Foundation and Massachusetts General Hospital Program, Boston	Designed and conceptualized study, revised the manuscript for intellectual content
Kelsey N. Bryk, MS	University of Delaware, Newark	Major role in the acquisition of data, revised the manuscript for intellectual content
Jessie R. Oldham, PhD	Boston Children's Hospital, The Micheli Center for Sports Injury Prevention, Waltham, MA	Major role in the acquisition of data, revised the manuscript for intellectual content
Paul F. Pasquina, MD	Uniformed Services University of the Health Sciences; Walter Reed National Military Medical Center, Bethesda, MD	Designed and conceptualized study, revised the manuscript for intellectual content
Steven P. Broglio, PhD, ATC	Michigan Concussion Center, University of Michigan, Ann Arbor	Designed and conceptualized study, revised the manuscript for intellectual content

Appendix (continued)

Name	Location	Contribution
Thomas W. McAllister, MD	Indiana University School of Medicine, Indianapolis	Designed and conceptualized study, revised the manuscript for intellectual content
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Appendix (continued)

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Continued

Appendix (continued)

Name	Location	Contribution
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Laura Lintner DO	Winston-Salem University, NC	Major role in the acquisition of data, revised the manuscript for intellectual content
Thomas A. Buckley, EdD, ATC	University of Delaware, Newark	Designed and conceptualized study, revised the manuscript for intellectual content

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