Age of First Exposure to American Football and Behavioral, Cognitive, Psychological, and Physical Outcomes in High School and Collegiate Football Players

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Background: Although some studies have observed a relationship between age of first exposure (AFE) to American football and long-term outcomes, recent findings in collegiate athletes did not observe a relationship between AFE and more intermediate outcomes at early adulthood. This, however, requires independent replication.

Hypothesis: There will be no association between AFE to football and behavioral, cognitive, emotional/psychological, and physical functioning in high school and collegiate athletes.

Study Design: Cross-sectional study.

Level of Evidence: Level 3.

Methods: Active high school and collegiate football players (N = 1802) underwent a comprehensive preseason evaluation on several clinical outcome measures. Demographic and health variables that significantly differed across AFE groups were identified as potential covariates. General linear models (GLMs) with AFE as the independent variable were performed for each clinical outcome variable. Similar GLMs that included identified covariates, with AFE as the predictor, were subsequently performed for each clinical outcome variable.

Results: After controlling for covariates of age, concussion history, race, and a diagnosis of ADHD, earlier AFE (<12 vs ≥12 years) did not significantly predict poorer performance on any clinical outcome measures (all $P > 0.05$). A single statistically significant association between AFE group and somatization score was recorded, with AFE <12 years exhibiting lower levels of somatization.

Conclusion: In a large cohort of active high school and collegiate football student-athletes, AFE before the age of 12 years was not associated with worse behavioral, cognitive, psychological, and physical (oculomotor functioning and postural stability) outcomes.

Clinical Relevance: The current findings suggest that timing of onset of football exposure does not result in poorer functioning in adolescence and young adults and may contribute to resilience through decreased levels of physically related psychological distress.

Keywords: adolescence; sports; football; concussion; traumatic brain injury; age of first exposure

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Societal concern regarding participation in football has significantly increased in recent years, with particular focus on the long-term adverse outcomes associated with football participation at all levels of play.^{6,27,49} This movement has resulted in legislation to set minimum ages on participation in youth football³² and decreased participation in youth and high school football nationally, with 19.2% less enrollment from 2011-2016 among children aged 6 to 17 years.⁴⁵ A recent survey of counseling practices and attitudes of pediatricians revealed virtually universal concern regarding concussion and contact sport participation among parents (97%).¹⁷ Furthermore, the study showed that 77% of pediatricians would not allow their child to play tackle football and 52% would not allow their child to play full contact sports at all.¹⁷

Prior studies have demonstrated an association between prior participation in elite-level football and a number of negative behavioral, emotional, and cognitive outcomes, suggestively attributable to chronic traumatic encephalopathy.^{35-38,47,52} The potential of adverse outcomes associated with football participation has been extended down from the elite level, with some evidence to suggest that initial participation at an earlier age, or age of first exposure (AFE), may eventually be associated with adverse long-term outcomes. Stamm et al⁵¹ reported that AFE before the age of 12 years was associated with poorer performance on cognitive measures of executive functioning, learning, and single-word reading in a small cohort of 42 former NFL players in the 5th to 7th decade of life. Similarly, Alosco et al¹ revealed an association between AFE before the age of 12 years and self-reported executive functioning, apathy, and depressive symptoms. In a separate study, through retrospective informant telephone interviews, Alosco et al¹ also reported that AFE before the age of 12 years was associated with earlier onset of neuropsychiatric behavioral and cognitive symptoms among 246 former tackle football players with a mean age of approximately 50 years. A number of reviews have highlighted several limitations of these studies, such as uncontrolled confounding variables and retrospective study designs possibly introducing recall bias, which could artificially inflate associations between reported concussion histories and self-reported behavioral, cognitive, and emotional symptoms.^{18,25,30,31}

While several studies have examined the relationship between AFE and multiple long-term outcomes in former athletes, no research to date has examined the relationship between AFE and more intermediate outcomes at early adulthood. Preliminary evidence suggests the process instigating long-term adverse outcomes may occur earlier in development. For example, immediate effects of participation in youth football have been observed, with changes noted in white matter integrity,¹⁴ cortical/deep gray matter,²⁰ and default mode network connectivity,⁵⁴ which were associated with changes in memory scores in nonconcussed high school football players over the course of a single season. The purpose of the current study was to examine the relationship between AFE and intermediate (ie, in adolescence/young adulthood) behavioral, cognitive, emotional/psychological, and physical (oculomotor functioning and postural stability) outcomes.

METHODS

Participants

Participants were football players from 9 high schools and 4 colleges in southeastern Wisconsin enrolled in the prospective Project Head to Head 1 (PH2H1) and/or Project Head to Head 2 (PH2H2) studies between August 2012 and September 2017 (see also LaRoche et al²⁹ and Nelson et al^{39,40}). PH2H1 and PH2H2 were both composed of samples from the same 9 high schools and 4 colleges in southeastern Wisconsin. Essentially, PH2H2 was an extension of PH2H1, with more clinical variables included as part of the battery administered. To maintain generalizability, exclusion criteria involved only 2 factors, invalid performance on computerized neurocognitive tests (CNT) and those in which AFE could not be obtained (ie, age or years of participation was not provided). Athletes who failed to produce a valid CNT profile at baseline ($n = 53$) or who did not list their age ($n = 57$) were excluded from the analysis, yielding a final sample of 1802 football players for analysis (Figure 1). Comparison between the maintained sample and those excluded did not significantly differ across AFE groups, $\chi^2(1) = 1.57$, $P = 0.21$.

Procedures

Baseline assessments took place before the start of regular football activity, lasted approximately 90 minutes, and were conducted at each athlete's school. No participants reported a recent concussion (ie, within 3 months). Baseline testing group sizes ranged from 1 to 20 athletes, with multiple research assistants present for each to allow for 1-on-1 proctoring of the assessments. Each athlete was read a standardized script at the beginning of the baseline testing session and before each of the CNTs about the importance of valid baseline tests. Adult athletes and parents of minor athletes completed informed consent, and minor participants completed assent before their first evaluation. All study procedures were approved by the Medical College of Wisconsin's institutional review board.

Measures

Data were collected through assessment procedures noted above and an in-depth historical interview; which included demographics, sport participation, and medical and concussion history (Table 1). AFE was the primary independent variable of interest and was derived from participants' response to questions about the number of years participating in the sport and current age (ie, years of participation subtracted from age). Following precedent from prior literature,^{1,2,46,50,51} AFE was dichotomized into 2 groups: AFE before the age of 12 years ($AFE < 12$) and at/ after the age of 12 years ($AFE \geq 12$). However, sensitivity analyses described below were also performed to ensure that dichotomizing this variable did not mask otherwise significant effects. Given that a number of factors, such as attention-deficit/hyperactivity disorder (ADHD),^{3,8,9,19,59} race,^{22,56} age,^{7,11,42} and self-reported concussion history,^{12,42} have been associated with several clinical outcome

Figure 1. Flowchart of final sample for analysis.

variables noted above, a number of demographic, sport, and medical history variables were examined as potential covariates (Table 1).

Outcomes of interest included a number of clinical symptom report, neuropsychological, and physical measures. Self-report symptom measures included the Sport Concussion Assessment Tool (SCAT)³⁴ and subscales of the Brief Symptom Inventory-18 (BSI-18; somatization, depression, and anxiety),¹⁵ a measure of internalizing psychopathology and somatic symptoms. For a subset of the sample that comprised the second phase of the study (ie, PH2H2), the Brief Sensation Seeking Scale,²³ and Disinhibition-11, an 11-item content-representative subset of the 20-item Externalizing Spectrum Inventory⁴³ with high correlation with the original measure ($r \approx 0.90$; C. Patrick, personal communication, September 21, 2017) were added to the battery as well (Table 2).

Cognitive and neuropsychological measures included the Standardized Assessment of Concussion (SAC),³³ the Immediate Post-Concussion and Cognitive Testing (ImPACT)²⁴ online neurocognitive test battery, Trail Making Test A and B (TMT A and B),⁴⁴ Wechsler Test of Adult Reading, and Wechsler Adult Intelligence Scale-IV (WAIS-IV) Processing Speed Index subtests (Coding and Symbol Search).⁵⁷ Similar to above, the TMT and WAIS-IV subtests were added to phase II of the study, with these latter measures included to provide more measurement of cognitive domains known to be affected by concussion (ie, executive functioning and processing speed).¹⁰ Physical functioning included a measure of postural stability (the Balance Error Scoring System)²¹ and oculomotor functioning (ie, King Devick Test; Table 2).⁴¹ Additionally, self-reported standardized American College Testing (ACT) scores were included as a

measure of academic achievement. A select number of participants had not yet completed the ACT (eg, sophomore in high school) or did not report their score ($n = 476$).

Analyses

Our primary interest was to determine if AFE was predictive of any clinical outcomes assessed in this high school and collegiate athlete sample. However, we anticipated that AFE may be related to demographic or other variables and that such confounding variables might obscure relationships between AFE and outcomes of interest. Potential covariates included several demographic and medical history variables collected. To select variables for inclusion as covariates in our primary analyses, the 2 AFE groups were compared on demographic and history variables, with t tests used for continuous and chi-squared analyses for categorical data. Variables that significantly differed across groups were then entered into a multivariable binary logistic regression with AFE <12 and ≥ 12 as the dependent variable to reduce redundancy of covariate predictors and removal of variables that were no longer significant, controlling for extraneous variance in the subsequent regressions.

Next, we undertook our primary analyses of interest. First, single-predictor general linear models (GLMs) were computed to estimate the effect of AFE (<12 and ≥ 12) on several clinical outcome variables in high school and collegiate football players. Outcome variables included the various measures of behavioral, cognitive, emotional/ psychological, and physical functioning highlighted above. Second, the same models were run again, this time including as independent variables both AFE and covariates identified earlier as demonstrating significant differences across the 2 AFE groups. Although dependent

Table 1. Demographic information for total sample and AFE groups

	n	Total Sample ^a (n = 1802)	AFE < 12 ^a (n = 1249)	AFE ≥ 12 ^a (n = 553)	P ^b	d/Phi ^{c,d}
Age, y	1802	17.99 ± 1.83	17.81 ± 1.78	18.41 ± 1.88	<0.001	0.328
Race	1802				<0.001	0.117
White		1359 (75.4)	983 (78.8)	376 (68.0)		
Black/African American		361 (20.0)	218 (17.5)	143 (25.9)		
Asian, Native Hawaiian/ Pacific Islander, American Indian, unknown		82 (4.6)	48 (3.8)	57 (5.5)		
Height, inches	1799	71.31 ± 2.78	71.23 ± 2.74	71.49 ± 2.85	0.063	
Weight, lbs	1801	200.63 ± 42.74	198.92 ± 41.70	204.49 ± 44.77	0.013	0.040
ADHD+	1800	171 (9.5)	106 (8.5)	65 (11.8)	0.030	0.044
Prescribed medication for ADHD ^e	93	56 (60.2)	40 (64.5)	16 (51.6)	0.231	
LD	1799	32 (1.8)	22 (1.8)	10 (1.8)	0.950	
IEP	1786	107 (6.0)	72 (5.8)	35 (6.4)	0.600	
Autism spectrum disorder	1795	3 (0.2)	2 (0.2)	1 (0.2)	0.919	
History of meningitis	1069	4 (0.4)	1 (0.1)	3 (0.9)	0.056	
Balance disorder	1063	1 (0.1)	1 (0.1)	0 (0.0)	0.503	
Seizure disorder	1801	13 (0.7)	11 (0.9)	2 (0.4)	0.229	
Vision problems (not glasses)	1798	31 (1.7)	21 (1.7)	10 (1.8)	0.844	
Hearing problems	1798	53 (2.9)	32 (2.6)	21 (3.8)	0.156	
Stroke	1797	2 (0.1)	1 (0.1)	1 (0.2)	0.552	
Diabetes	1799	12 (0.7)	10 (0.8)	2 (0.4)	0.289	
Psychiatric diagnosis (Y/N)	1800	12 (0.7)	10 (0.8)	2 (0.4)	0.290	
Mood Disorder/Depression/ Bipolar		3	1 (10.0)	2 (100.0)		
Anxiety		8	8 (80.0)	0 (0.0)		
Psychotic disorder/ Schizophrenia		1	1 (10.0)	0 (0.0)		
Migraine disorder	1793	88 (4.9)	54 (4.3)	34 (6.2)	0.097	
Headache (nonmigraine) disorder	1802	89 (4.9)	65 (5.2)	24 (4.3)	0.435	
Sleep disorder	1789	20 (1.1)	12 (1.0)	8 (1.5)	0.355	
Household SES (range: 8-66)	1739	46.49 ± 10.36	46.92 ± 10.00	45.52 ± 11.10	0.013	0.132
Total No. of prior concussions	1802	0.78 ± 1.27	0.83 ± 1.32	0.68 ± 1.15	0.065	
Duration since last concussion, mo	752	33.87 ± 76.95	34.99 ± 88.44	30.83 ± 26.03	0.514	

Table 1. (continued)

	n	Total Sample ^a (n = 1802)	AFE < 12 ^a (n = 1249)	AFE ≥ 12 ^a (n = 553)	P ^b	d/Phi ^{c,d}
Number of diagnosed SRCs	1797					
0		1183 (65.8)	797 (64.0)	386 (70.1)	0.041	0.060
1		418 (23.3)	304 (24.4)	114 (20.7)		
2+		196 (10.9)	145 (11.6)	51 (9.3)		
Number of undiagnosed SRCs	1785				0.092	0.052
0	1555	1555 (87.1)	1067 (86.1)	488 (89.4)		
1	153	153 (8.6)	118 (9.5)	35 (6.4)		
2+	77	77 (4.3)	54 (4.4)	23 (4.2)		
Number of diagnosed non-SRCs	1775	0.06 ± 0.24	0.06 ± 0.23	0.05 ± 0.25	0.814	
Typical number of hours of sleep	1794	7.35 ± 1.01	7.39 ± 1.03	7.26 ± 0.95	0.009	0.131
Average weekly alcoholic drinks	238	5.51 ± 6.74	5.29 ± 6.85	6.06 ± 6.48	0.432	
Prescription medications (Y/N)	1798	334 (18.6)	239 (19.2)	95 (17.2)	0.429	
Antidepressants	1802	6 (0.3)	3 (0.2)	3 (0.5)	0.304	
Antianxiety	1802	18 (1.0)	12 (1.0)	6 (1.1)	0.807	
Antipsychotic	1802	0 (0.0)	0 (0.0)	0 (0.0)	1.000	
Narcotic/Pain	1802	33 (1.8)	20 (1.6)	13 (2.4)	0.274	
Nonnarcotic/Pain	1802	5 (0.3)	4 (0.3)	1 (0.2)	0.604	
Sleep aid/Sedative	1802	90 (5.0)	69 (5.5)	21 (3.8)	0.121	
Psychostimulant	1802	32 (3.0)	25 (3.4)	7 (2.1)	0.260	
Allergy	1802	41 (3.8)	27 (3.7)	14 (4.2)	0.650	
Asthma	1802	44 (4.1)	29 (3.9)	15 (4.5)	0.644	
Acid reflux	1802	7 (0.7)	6 (0.8)	1 (0.3)	0.339	
WTAR score	1798	32.12 ± 7.29	32.18 ± 7.07	31.98 ± 7.76	0.590	
Position	1699					
Offensive lineman		287 (16.9)	201 (16.9)	86 (16.8)		
Quarterback/Wide receiver		393 (23.1)	269 (22.7)	124 (24.2)		
Defensive lineman		284 (16.7)	176 (14.8)	108 (21.1)		
Linebacker		267 (15.7)	199 (16.8)	68 (13.3)		
Defensive back		301 (17.7)	220 (18.5)	81 (15.8)		
Running back		167 (9.8)	121 (10.2)	46 (9.0)		

ADHD, attention-deficit/hyperactivity disorder; AFE, estimated age of first exposure; IEP, individualized education plan; LD, learning disorder; SES, socioeconomic status; SRC, sports-related concussion; WTAR, Wechsler Test of Adult Reading; Y/N, yes/no.

^aInformation presented as mean ± SD or as number (percentage).

^bP value for tests of AFE group differences across demographic and history variables. Chi-square tests were performed for categorical and independent-samples *t* tests were performed for continuous variables. Boldfaced *P* value indicates potential covariates significant at the <0.10 level.

^cCohen *d* effect size interpretation: small = 0.2, medium = 0.5, large = 0.8.

^dPhi effect size interpretation: [*df* = 1] small = 0.07, medium = 0.21, large = 0.35.

^ePercentage of those who answered this question.

Table 2. General linear models examining AFE groups across clinical outcome variables

	Total Sample	AFE < 12	AFE ≥ 12	F	P ^a	η _p ^{2b}
SCAT3 symptom score (n = 1779) ^c	3.51 (5.77)	3.42 (5.68)	3.70 (5.98)	0.88	0.349	0.0
SAC total score (n = 1771) ^c	26.27 (2.16)	26.03 (2.17)	26.20 (2.15)	0.78	0.377	0.0
BESS total score (n = 1772) ^c	12.13 (4.82)	12.11 (4.71)	12.15 (5.07)	0.02	0.885	0.0
ImPACT composite scores						
Verbal memory (n = 1532) ^c	83.54 (10.70)	83.66 (10.64)	83.26 (10.82)	0.45	0.5	0.0
Visual memory (n = 1532) ^c	76.42 (13.01)	77.01 (13.04)	75.12 (12.87)	6.98	0.008	0.005
Visual motor speed (n = 1532) ^c	38.68 (6.52)	38.83 (6.47)	38.38 (6.64)	1.54	0.211	0.001
Reaction time (n = 1532) ^c	0.58 (0.08)	0.58 (0.08)	0.59 (0.09)	0.51	0.51	0.0
BSI-18 somatization (raw; n = 1779) ^c	0.91 (1.66)	0.86 (1.52)	1.04 (1.94)	4.40	0.036	0.002
BSI-18 depression (raw; n = 1779) ^c	1.15 (2.21)	1.11 (2.14)	1.24 (2.07)	1.24	0.266	0.001
BSI-18 anxiety (raw; n = 1779) ^c	1.19 (1.95)	1.16 (2.0)	1.26 (1.85)	0.99	0.319	0.001
TMT A (sec; n = 1046) ^d	22.37 (7.07)	22.24 (6.91)	22.66 (7.41)	0.81	0.369	0.001
TMT B (sec; n = 1046) ^d	57.00 (19.5)	56.35 (19.37)	58.46 (19.73)	2.611	0.106	0.002
King Devick (n = 526) ^d	46.84 (8.55)	46.68 (8.58)	47.19 (8.52)	0.40	0.526	0.001
DIS-11 (n = 1078) ^d	5.58 (5.16)	5.60 (5.45)	5.53 (4.44)	0.05	0.82	0.0
BSSS (n = 1078) ^d	26.87 (6.21)	26.93 (6.08)	26.73 (6.49)	0.23	0.63	0.0
WAIS-IV symbol search (raw; n = 1038) ^d	36.39 (6.38)	36.29 (6.25)	36.61 (6.68)	0.53	0.47	0.001
WAIS-IV coding (raw; n = 1038) ^d	69.32 (11.60)	68.95 (11.29)	70.13 (12.23)	2.31	0.129	0.002
ACT scores (n = 1326) ^c	22.64 (3.52)	22.74 (3.45)	22.64 (3.522)	2.23	0.135	0.002

ACT, American College Testing; BESS, Balance Error Scoring System; BSI-18, Brief Symptom Inventory; BSSS, Brief Sensation Seeking Scale; DIS-11, Disinhibition-11; ImPACT, Immediate and Post-Concussion Assessment and Cognitive Testing; SAC, Standardized Assessment of Concussion; SCAT3, Sport Concussion Assessment Tool third edition; TMT A/B, Trail Making Test; WAIS, Wechsler Adult Intelligence Scale–Fourth Edition.

^aBoldfaced P value indicates a significant relationship between AFE and clinical outcome at the <0.05 level.

^bPartial eta-square (η_p²) effect size interpretation: small = 0.01, medium = 0.06, large = 0.14.

^cMeasures included as part of Projects Head to Head I and II.

^dMeasures added as part of Project Head to Head II.

variables were generally not normally distributed, the estimated parameters and corresponding 95% CI estimates are generally robust to violations of this assumption within larger sample sizes.^{16,48} Additionally, sensitivity analyses were performed to assess the effect of different distribution models (eg, negative binomial regression) on the associations between predictors and outcomes. These analyses suggested that the nature of associations was not meaningfully changed with different model distributions, and as such, GLMs were maintained for all analyses in order to be able to compare effect sizes across outcomes.

RESULTS

Demographic, medical/treatment history, and sport information (eg, SRC history and football position) on the sample is provided in Table 1. The mean sample age was 17.99 years (SD = 1.83) and the average AFE was 10.11 years (SD = 2.96), with roughly 70% of the sample reporting AFE before age 12 years. The AFE (<12 and ≥12) groups significantly differed across a number of these variables, including age, race, weight, a diagnosis of ADHD, household socioeconomic status (SES), number of diagnosed sports-related concussions (SRCs), number of

Table 3. Binary logistic regression of potential covariates and AFE group^a

	B	SE	Wald	<i>P</i> ^b	Exp (B)	95% CI Lower Limit	95% CI Upper Limit
Age	0.211	0.033	40.46	<0.001	1.235	1.157	1.317
Weight	0.001	0.001	0.17	0.678	1.001	0.998	1.003
Household SES	−0.006	0.005	1.31	0.253	0.994	0.984	1.004
Number of diagnosed SRC	−0.180	0.072	6.25	0.012	0.835	0.725	0.962
Number of undiagnosed SRC	−0.159	0.090	3.13	0.077	0.853	0.715	1.017
Typical hours of sleep	−0.035	0.058	0.37	0.543	0.966	0.862	1.081
ADHD+	0.488	0.180	7.35	0.007	1.629	1.145	2.319
Race	0.399	0.098	16.56	<0.001	1.490	1.230	1.806

ADHD, attention-deficit/hyperactivity disorder; AFE, age of first exposure; SES, socioeconomic status; SRC, sports-related concussion.

^aModel: $\chi^2(8) = 86.81$, $P < 0.001$, Nagelkerke $R^2 = 0.07$.

^bBoldfaced *P* value indicates covariates that remained significant in the multivariable regression at the <0.05 level.

undiagnosed SRCs, and typical hours of sleep per night. A sensitivity analyses was performed in order to examine the relationship between these variables and AFE as a continuous variable. Results showed that many of the variables maintained their significant associations with AFE, with the exception of weight and household SES. Regardless, all significant variables were included in a multivariable binary logistic regression with AFE group as the dependent variable. Results indicated that 4 variables independently predicted whether participants had begun football participation <12 or ≥ 12 years of age, $\chi^2(8) = 86.81$, $P < 0.001$: age, history of SRC, diagnosis of ADHD, and race (Table 3).

Single-predictor GLM models predicting clinical outcomes from the AFE group revealed significant effects for 2 outcome measures. Specifically, those who began playing football before the age of 12 years exhibited significantly higher visual memory scores, $F = 6.98$, $P < 0.01$, $\eta_p^2 = 0.01$ (Table 2). Additionally, AFE <12 was associated with significantly lower somatization symptom endorsement, $F = 4.4$, $P = 0.04$, $\eta_p^2 = 0.002$. GLMs containing the 4 identified covariates along with AFE were performed for each clinical outcome. Once covariates were included in the GLMs, AFE <12 remained significantly associated with lower levels of somatization $F = 11.39$, $P = 0.01$, $\eta_p^2 = 0.01$, while the effect of AFE on visual memory was no longer significant ($P = 0.11$), nor was any other outcome predicted newly by AFE (Table 4).

DISCUSSION

In a large sample of over 1800 active high school and collegiate football players, the current study failed to find any meaningful associations between AFE to football before the age of 12 years

and several measures of behavioral, cognitive, emotional/psychological, and physical functioning. Ultimately, when using psychometrically validated measures, there were no adverse outcomes during adolescence/young adulthood associated with football participation before the age of 12 years. A single significant relationship was identified, in which football participation before the age of 12 years was associated with lower severity of somatic symptoms.

The current findings that AFE is not associated with worse behavioral, cognitive, psychological, or physical functioning extends upon previous studies, which failed to find any associations between similar measures of functioning during adolescence/early adulthood and contact sport participation. Specifically, Katz et al²⁶ observed better ImPACT verbal and visual memory scores, SCAT symptom endorsement (ie, lower symptom endorsement), and BSI-18 scores (ie, lower scores) among 15,681 contact sport athletes on baseline testing; but worse ImPACT reaction time and SAC total scores. Furthermore, Houck et al²² demonstrated that amount of participation, or years exposed to sport, was not associated with any metrics of cognitive functioning on ImPACT in 87 collegiate football players. Given that statistical tests will very likely demonstrate significant differences with a large enough sample size,⁵³ the lack of association between AFE and clinical outcomes in the current sample is particularly revealing. The current sample contained more than adequate power to detect even rather small effects, suggesting that there is close to or essentially no effect whatsoever.⁵³

Unlike the present study, which investigated active high school and collegiate athletes, prior studies reporting significant relationships between earlier exposure (AFE <12) to football and eventual poorer cognitive and emotional outcomes have

Table 4. General linear models including AFE groups and covariates across clinical variables

	AFE		Age		Race		ADHD+		Prior SRC	
	P^a	η_p^{2b}	P	η_p^2	P	η_p^2	P	η_p^2	P	η_p^2
BSI-18										
Somatization	0.001	0.006	0.084	0.003	< 0.001	0.031	0.153	0.001	0.002	0.007
Depression	0.225	0.001	0.259	0.002	0.186	0.001	0.758	0.000	0.013	0.005
Anxiety	0.166	0.001	0.016	0.003	0.906	0.000	< 0.001	0.008	0.013	0.005
ImPACT composite score										
Verbal memory	0.733	0.000	0.001	0.010	0.098	0.002	0.019	0.004	0.299	0.002
Visual memory	0.109	0.002	< 0.001	0.016	0.218	0.001	< 0.001	0.008	0.206	0.002
Visual motor speed	0.176	0.001	< 0.001	0.039	< 0.001	0.025	0.174	0.001	0.110	0.003
Reaction Time	0.681	0.000	< 0.001	0.036	< 0.001	0.012	0.132	0.001	0.543	0.010
Trail Making Test										
A	0.146	0.002	< 0.001	0.019	< 0.001	0.016	0.566	0.000	0.074	0.005
B	0.107	0.003	0.003	0.009	< 0.001	0.054	0.047	0.004	0.832	0.000
WAIS-IV										
Symbol search	0.788	0.000	< 0.001	0.019	< 0.001	0.015	0.022	0.005	0.653	0.001
Coding	0.494	0.000	< 0.001	0.043	0.006	0.010	< 0.001	0.012	0.375	0.002
SCAT3 symptom severity score	0.113	0.001	0.117	0.002	< 0.001	0.013	< 0.001	0.010	0.001	0.007
SAC total score	0.955	0.000	< 0.001	0.029	0.163	0.001	< 0.001	0.015	0.014	0.005
BESS total score	0.692	0.000	0.093	0.003	0.002	0.006	0.074	0.002	0.901	0.000
King Device	0.272	0.002	0.351	0.002	0.414	0.003	0.225	0.003	0.177	0.007
ACT score	0.909	0.000	0.182	0.001	< 0.001	0.098	< 0.001	0.014	0.028	0.005
DIS-11	0.814	0.000	0.001	0.011	0.201	0.003	0.001	0.010	0.006	0.010
BSSS	0.607	0.000	0.247	0.0001	< 0.001	0.021	0.290	0.001	0.934	0.000

ACT, American College Testing; ADHD, attention-deficit/hyperactivity disorder; AFE, age of first exposure; BESS, Balance Error Scoring System; BSI-18, Brief Symptom Inventory; BSSS, Brief Sensation Seeking Scale; DIS-11, Disinhibition-11; ImPACT, Immediate and Post-Concussion Assessment and Cognitive Testing; SAC, Standardized Assessment of Concussion; SCAT3, Sport Concussion Assessment Tool third edition; SRC, sports-related concussion; WAIS, Wechsler Adult Intelligence Scale.

^aBoldfaced P value indicates a significant relationship between AFE or covariate and clinical outcome at the <0.05 level.

^bPartial eta-square (η_p^2) effect size interpretation: small = 0.01, medium = 0.06, large = 0.14.

focused on older adults.^{1,2,46,50} At this point, the gap between a lack of adverse outcomes earlier in development and adverse sequelae reported among those later in life is unclear. Efforts have been employed to better understand the potential underlying process in vivo during the earlier stages of development. For example, using diffusion tensor imaging, multiple studies have observed a significant association between a quantified, objective measure of head impact exposure and changes in fractional anisotropy across multiple white matter tracts, with medium to large effect sizes,^{5,13,28} suggesting a possible dose-dependent response relationship. These studies may provide important insights into the relationship between exposure and adverse outcomes later in life, as the football players included within the above-cited studies did not sustain a concussion over the course of the measured season.

Furthermore, the microstructural changes associated with cumulative impact exposure over the course of a single high school football season observed by Davenport et al¹⁵ is particularly important, as the football players had multiple years of prior football experience and exhibited changes in white matter tracts, even in the absence of clinical changes over the course of the season (ie, no significant decline or change in cognitive functioning on ImPACT). This may be especially relevant to the current study, where, although associations between AFE and several outcomes were not observed, AFE could still possibly be correlated with underlying microstructural changes earlier in development. However, Davenport et al¹⁵ advise that the pathological implications of these metrics are not well understood and that interpretations should be made with caution.

The current study also emphasized the importance of controlling for preexisting demographic and medical variables when attempting to examine the effect of AFE to football. AFE was more strongly and consistently associated with demographics and medical history than clinical outcome measures. This suggests that there is a need to account for confounding variables such as demographics when looking at clinical outcomes between AFE groups. This is especially true, as the covariates identified in the current investigation were significantly associated with several clinical outcomes. This is consistent with several prior studies, which observed similar effects of preexisting or medical histories and performance/scores on measures of behavioral, cognitive, psychological, physical, and general symptoms measures. Specifically, ADHD,^{3,8,9,19,59} race,^{22,56} age,^{7,11,42} and self-reported concussion history^{12,42} have been associated with several clinical outcome variables noted above. The importance of controlling for preexisting conditions becomes even more crucial when attempting to investigate the long-term effects of AFE, as medical histories become ever more complex in older age.

In addition to clinical outcomes, previous studies have examined the relationship between AFE and football before the age of 12 years and neuroimaging metrics.^{46,51} For example, using diffusion tensor imaging, Stamm et al⁵¹ observed a significant relationship between AFE <12 and football and altered white matter microstructure in the corpus callosum

within 40 former professional football players. Additionally, younger AFE (continuous variable) was mildly associated with subcortical structural changes; specifically, right thalamic volumes.⁴⁶ Future research examining the effect of AFE on brain structure and function using advanced neuroimaging metrics in the current cohort of high school and collegiate athletes is currently in process. Future work should look to identify the mechanistic relationship between AFE and brain function. In interpreting longitudinal neuroimaging studies, it is important to consider the influence of not only exposure but also normal development, exercise, and so on to allow one to understand to what degree changes represent problematic/abnormal phenomena that may have negative clinical implications. Additionally, future work should look to further investigate any mechanistic relationship between earlier AFE and lower levels of somatic symptom endorsement. One possibility is the previously identified protective effect of sport participation on psychological dysfunction/symptomology⁴ and physical well-being (ie, lower rates of obesity) in young adulthood.⁵⁵

This study has important limitations worth noting. A limitation exists within the self-report nature of some data. First, as with most other studies within this subject area, AFE was reliant on athletes' self-report of years of participation and exposure history. Additionally, it is worth noting that reliability of self-reported concussion history can be variable. In a study of athletes ranging from age 13 to age 18 years, approximately 20% exhibited inconsistency in reported concussion history over a 2-year period.⁵⁸ Additionally, other sport participation throughout development was not accounted for in the current investigation. While it is possible for a compounding effect of football, plus additional contact sport participation, to influence clinical outcome measures of the study, the null effect of greater exposure to football (ipso facto, football at an earlier age) observed in the current study decreased this possibility. Furthermore, a high correlation of AFE and overall exposure in the current sample ($r = 0.80$) does make it difficult to differentiate the influence of each factor. However, given the null results, the need to deconstruct observed effects is not critical (ie, if an AFE effect was observed, it would be difficult to attribute results directly to AFE or total exposure). Finally, the results from the current study do not necessarily negate previous findings suggesting that adverse long-term outcomes may be associated with earlier AFE in a select subset of former athletes. Rather, the current study suggests that changes are not evident in adolescence and early adulthood.

As highlighted above, in a prior study 52% of pediatricians reported that they would not allow their child to play full contact sports.¹⁷ With the well-documented benefits of youth sport participation,^{4,55} it is essential that clinical education provided, public policy, and organizational guidelines comprehensively account for factors that may influence adverse outcomes often associated with contact sport and football participation. Currently active athletes who participated in football before the age of 12 years do not exhibit worse behavioral, cognitive, psychological, or physical (oculomotor

and postural stability) functioning during high school and collegiate age years. Furthermore, athletes who participated in football before the age of 12 years are less likely to endorse symptoms of somatization. These findings suggest that previously recorded long-term adverse outcomes associated with earlier AFE later in life are not apparent earlier in development. Further research is needed to better understand the potential underlying mechanisms and processes that account for the differential effects of AFE through development.

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