RESEARCH ARTICLE

Sleep Apnea Screening and Neuropsychiatric Symptoms in Former Professional American-Style Football Players

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Neurology® 2026;106:e214922. doi:10.1212/WNL.0000000000214922

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Abstract

Background and Objectives

Former professional American-style football (ASF) players may be at increased risk of sleep apnea (SA) compared with the general population. However, SA may be underdiagnosed or undertreated in former ASF players and be associated with cardiovascular, cognitive, and psychological health. The aim of this study was to characterize former professional ASF players self-reporting SA diagnoses, as well as undiagnosed players who screened positively on a validated SA instrument.

Methods

A cross-sectional survey was administered as part of the Football Players Health Study at Harvard University between 2017 and 2020. Former ASF players who signed a professional contract after 1960 were eligible to enroll. Data included demographics and football-related exposures (e.g., position and career duration), self-reported SA diagnosis, and STOP-BANG screening scores. Head injury exposure was measured using the number of concussion symptoms accrued during football practice or play. Outcomes included self-reported cognitive functioning, depression, anxiety, and pain. Multivariable logistic and linear regression was used to identify associations between sleep outcomes and (1) demographic and football characteristics and (2) recent mood, cognitive, and pain symptoms, respectively.

Results

Among 1,951 participants, 31.8% self-reported SA diagnoses (n = 621), which were associated with older age, Black race, lineman status, and higher numbers of football-accrued concussion symptoms. Only 39.8% (n = 247) of the SA group reported using their positive airway pressure therapy 4+ times per week. Of those without a current diagnosis of SA (n = 1,330), 74.6% (n = 992) scored in the intermediate or high range on the STOP-BANG. In multivariable models adjusted for demographic and football-related exposures, SA diagnosis and intermediate/high STOP-BANG scores were associated with more pain, anxiety, depression, and cognitive symptoms. Data showed that participants with diagnosed but untreated SA reported the highest symptom burden (all $p < 0.001$).

Discussion

This study identified that many former professional ASF players may be at risk of not being appropriately screened or treated for SA. These results estimate that the actual proportion of former professional ASF players with SA may be as high as 69%. Mood, pain, and cognitive symptoms may be exacerbated in those with treated, undertreated, or suspected SA.

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Glossary

AHI = apnea hypopnea index; **ASF** = American-style football; **BMI** = body mass index; **BPI** = Brief Pain Inventory; **CPAP** = continuous positive airway pressure; **CSS** = concussion signs and symptoms; **GAD-7** = Generalized Anxiety Disorder; **FPHS** = Football Players Health Study at Harvard University; **Neuro-QoL** = Quality of Life in Neurologic Disorders; **NFL** = National Football League; **OR** = odds ratio; **OSA** = obstructive sleep apnea; **PHQ-9** = 9-item Patient Health Questionnaire; **SA** = sleep apnea.

Introduction

Obstructive sleep apnea (OSA) is a breathing disorder characterized by repetitive obstructive apneas (i.e., airflow cessation) or hypopneas (i.e., airflow reduction) characterized by upper airway collapse. This reduction in airflow can acutely cause hypoventilation, hypoxemia, and arousals from sleep.[1] Many patients with undiagnosed sleep apnea (SA) report clinical symptoms such as daytime sleepiness, fatigue, difficulty with focus,[2] falling asleep in monotonous situations (e.g., watching television and sitting quietly),[3] and morning headaches.[4] Physical symptomatology in individuals with undiagnosed OSA includes snoring, choking, or gasping while sleeping.[5] There is significant evidence that untreated OSA can increase the risk of serious long-term adverse health outcomes, such as all-cause mortality,[6] cardiovascular events (e.g., myocardial infarction and stroke),[6,7] and low testosterone.[8] In addition, untreated OSA may also be associated with depression,[6,7] suicide,[9] diabetes,[6,7] and neurocognitive dysfunction,[10] and likely has a bidirectional relationship with neurodegenerative diseases,[11-14] although the directionality and strength of these associations are less well established.

OSA is traditionally diagnosed using polysomnography, which generates an apnea-hypopnea index (AHI), such that AHI scores greater than 4 are considered to have mild (5–14), moderate (15–29), or severe (30+) OSA. Previous research has suggested that approximately 80% of persons with OSA are undiagnosed.[15,16] Given the difficulties with administering polysomnography, screening tools are used to identify risk of OSA. One of the most commonly used tools to identify persons at risk of OSA is the STOP-BANG,[17,18] which uses 8 yes/no questions to predict OSA. In a systematic review and meta-analysis of the STOP-BANG questionnaire, the psychometrics of screening positively for having an objective diagnosis of SA were as follows: sensitivity = 0.73, specificity = 0.66, positive predictive value = 0.74, and negative predictive value = 0.64.[19]

Former professional football players may be at risk of OSA, given their unique physical characteristics that are known risk factors of OSA, such as male biological sex, larger neck circumference, and elevated body mass index (BMI).[20] Previous studies examining SA risk that use self-reported screening tools suggest that former professional football players may be at higher risk compared with the general population.[21,22] Using objective measures, the prevalence of sleep-disordered breathing in active National Football League (NFL) players (n = 137; mean age = 27 ± 3) was 19% using a single-channel screening tool,[23] whereas the prevalence of sleep-disordered breathing in former NFL players (n = 257; mean age = 53.9 ± 1.0) was 52.7% using a self-applied limited-channel portable sleep device.[24] The prevalence of objectively identified SA in former professional football players is likely higher than in men in the general population. A population-based review of 11 epidemiologic studies estimated that 22% of men have SA,[25] and a recent study found that former NFL players had a higher rate of self-reported SA than asymptomatic male controls (36.7% vs 16.7%).[26] Previous research from the Football Players Health Study at Harvard University (FPHS; n = 4,189) found that only 22.4% of former professional football players self-reported being diagnosed with SA by a health care provider.[27] This estimate is substantially lower than research suggesting that 52.7% of former NFL players were diagnosed with SA using portable sleep tests.[24]

The potential discrepancy or underdiagnosis of SA in this population spurred the current investigation in hopes of better understanding sleep-related symptomatology to guide optimal screening and treatment. In a subset of the FPHS cohort of former professional football players who completed the STOP-BANG screening tool, this study aimed to (1) compare those who reported being diagnosed with SA with those without a diagnosis of SA on a variety of health outcome variables, (2) understand how those with and without diagnosed SA scored on the STOP-BANG, (3) quantify associations with established SA risk factors and football-related exposures, and (4) assess whether those who screened positively for SA but do not have a current SA diagnosis report adverse health outcomes including depression, anxiety, self-reported cognitive symptoms, and pain.

Methods

Study Participants

The FPHS[28] recruited former professional American-style football (ASF) players who signed a contract with any professional football league after 1960[29] to join a longitudinal epidemiologic cohort. Starting in January 2015, residential and electronic mails were used to invite 15,011 potential participants, among whom 4,180 (27.2%) enrolled and completed an initial self-reported survey (eFigure 1). On the initial survey, participants responded to questions regarding

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demographic factors, football-related experiences and exposures, and current health status. From this initial cohort, 47.4% (n = 1,980) completed a second questionnaire at least 2 years after enrollment that included additional football exposures and other measures of current health status. Data used in this study were collected between 2019 and 2022. SA diagnosis status was disclosed by 1,951 participants, of whom 1,850 completed the STOP-BANG (eFigure 1).

Standard Protocol Approvals, Registrations, and Patient Consents

This study was approved by the Institutional Review Board of the Harvard T.H. Chan School of Public Health (Protocol # IRB18-1365), and participants provided informed consent before enrollment.

Demographic and Football Measures

Age at time of follow-up was determined using birth date and SA survey completion date. As previously described,[30] race was categorized into Black, White, Native Hawaiian/Pacific Islander/Asian/American Indian/Alaskan Native, and missing. The initial survey collected data on self-reported number of seasons of professional football, age at first exposure to organized football, team position, and previous use of performance-enhancing drugs. The number of seasons, which represents career duration, was categorized into 1–6, 7–12, and 13+ professional seasons; these groups were created based on previous analyses of this data set, suggesting a nonlinear relationship between career duration and outcomes.[31] Position was dichotomized into linemen (offensive line, defensive line, and linebacker) or nonlinemen (all other positions). Cumulative exposure to head injury during football was assessed using the concussion signs and symptom (CSS) score, whereby participants were asked, "While playing or practicing football, did you experience a blow to the head, neck, or upper body followed by any of the following: headaches, nausea, dizziness, loss of consciousness, memory problems, disorientation, confusion, seizure, visual problems, or feeling unsteady on your feet?" For each of these 10 signs/symptoms, participants chose "none," "once," "2–5 times," "6–10 times," or "11 times." These were coded and summed as previously described[30,32-34] to create a concussion symptom score and categorized into quartiles.

SA and Continuous Positive Airway Pressure Therapy Outcomes

Self-reported diagnoses of SA were based on a yes/no response to the following prompt: "Has a health care provider ever told you that you have …sleep apnea?" The STOP-BANG questionnaire was included in the follow-up questionnaire and is an 8-item screening tool for OSA (including male biological sex, age, hypertension status, BMI, fatigue, and observed behavior during sleep [e.g., snoring, gasping, and choking; eTable 1]).[17] Participants were assigned 1 point for each item: 1) snoring loudly; 2) feeling tired or fatigued during the day; 3) anyone observed breath difficulties or choking/gasping during sleep; 4) having or being treated for high blood pressure; 5) body mass index over 35 kg/m^2; 6) age older than 50; 7) neck size greater than 16 inches; and 8) male biological sex. The original development study reports that those with STOP-BANG scores of either 3 or 4 are at intermediate risk and scores at or above 5 are at high risk of SA.[17] For this study, participants were classified as "not at risk" if they had a low STOP-BANG score (i.e., 0–2), and "at risk" if they received a score of 3 or higher.

Participants were also asked, "Do you currently use a continuous positive airway pressure (CPAP) device for sleep apnea?" Those who reported yes were further asked, "About how many days per week do you use your CPAP device?" Responses were dichotomized into those who use a continuous positive airway pressure (CPAP) fewer than half the week (0–3 days per week; untreated) and more than half the week (4 or more days per week; treated) consistent with a previous study.[35] Self-reported SA, STOP-BANG score, and CPAP use were used to create a composite variable. First, we identified participants who reported a SA diagnosis. These participants were divided into "diagnosed and treated" (self-reported SA diagnosis and regular use of a CPAP) and "diagnosed and untreated" (participants reporting SA diagnoses but no or infrequent CPAP use). Among the remaining undiagnosed participants, we categorized those with STOP-BANG scores >3 as "undiagnosed and at-risk" and "none" for those without an SA diagnosis and with STOP-BANG scores below 3.

Comorbidities

Cognitive symptoms were assessed using the Quality of Life in Neurological Disorders (Neuro-QoL) Applied Cognition-General Concerns scale, which queries 8 cognitive symptoms over the previous week.[36] The scores of each item were summed and converted to a T-score based on scores from the general population, with higher scores indicating better perceived cognitive functioning. For the purposes of this study, we inverted this scale by multiplying all T-scores by −1, such that higher scores indicate worse perceived cognitive functioning. Anxiety symptoms from the previous 2 weeks were assessed using the Generalized Anxiety Disorder (GAD-7) questionnaire.[37] Depressive symptoms from the previous 2 weeks were assessed using the 9-item Patient Health Questionnaire (PHQ-9).[38] Participants rated their pain using an item from the Brief Pain Inventory (BPI),[39] which asks, "Please rate your pain by marking the oval with the number that best describes your pain on the average," and offers values from 0 (no pain) to 10 (worst pain you can imagine).

Current BMI—as well as BMI during professional play—was calculated using self-reported weight (lbs.) and height (inches) as (lbs. × 0.45/inches × 0.025) on the follow-up survey. Stroke, heart attack, Alzheimer disease, and vascular and other dementia were based yes/no responses to "Has a health care provider ever told you that you have had any of the following diagnoses or health outcomes?" Participants were also asked "Has a medical provider ever recommended

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or prescribed medication for [condition]" and "Are you currently taking medication [for that condition]" for the following conditions: attention-deficit/hyperactivity disorder (ADHD), diabetes, headaches, high cholesterol, heart failure, heart rhythm problems (atrial fibrillation, supraventricular tachycardia, or other), and low testosterone. Participants also reported whether they had undergone the following cardiac procedures: (1) heart bypass, (2) angioplasty, or (3) stent replacement. Participants who self-reported a heart attack, heart failure, heart rhythm problems, or any of the cardiac procedures were classified as having a "heart condition."

Statistical Analyses

Bivariable associations between self-reported SA diagnoses and demographic, football-related, sleep-related, and health conditions were determined using Kruskal-Wallis rank sum tests for continuous variables and chi-square tests for categorical variables. Kruskal-Wallis tests generated η^2 effect sizes, which were interpreted as follows: <0.01 = negligible; 0.01–0.05 = small; 0.06–0.13 = medium; and ≥0.14 = large.[40] Chi-square tests generated Cramer *V* effect sizes, which were interpreted as (based on 1 degree of freedom): 0.00–0.09 = negligible; 0.10–0.29 = small; 0.30–0.49 = medium; and ≥0.50 = large.[41]

Two separate multivariable logistic regressions were used to examine the associations between demographic and football-related exposures and (1) self-reported SA and (2) intermediate or high risk of SA based on the STOP-BANG. These exploratory models included age, race, BMI during professional play, age at first football exposure, lineman status, concussion symptom score, debut year, career duration, and use of performance-enhancing drugs. Age, BMI during professional play, age at first football exposure, concussion symptom score, and debut year (first calendar year of professional play) were treated as continuous variables. Race, lineman status, career duration category, and use of performance-enhancing drugs were categorical variables.

In addition, we implemented 4 separate linear models to examine the relationship between the composite SA risk variable (i.e., "none"; "at-risk and undiagnosed"; "diagnosed and treated; and diagnosed and untreated") and 4 continuous health outcomes: (1) cognitive symptoms (Neuro-QoL score); (2) chronic pain severity (BPI score); (3) symptoms of anxiety (GAD-7 score); and (4) symptoms of depression (PHQ-9 score). These models were adjusted for the same variables as the aforementioned logistic regressions.

The threshold for statistical significance (α) was set at 0.05, and all analyses were conducted using R Language for Statistical Computing.[42] A complete-case approach was used because of the low proportion of missing data on the main outcome variable (incomplete STOP-BANG = 5.1%)[43,44] and the fact that the items on the STOP-BANG do not measure a unidimensional latent construct.[45] Owing to this study's large sample size, this study was highly powered to detect statistically significant differences between groups. However, not all statistically significant differences may be clinically meaningful. We, therefore, used both statistical significance and effect size estimates to interpret findings.

Data Availability

Owing to the high-profile nature of the participants in this study, data are not available at this time.

Results

Bivariable Associations Between Participant Characteristics and Self-Reported SA Status

Of the 1,951 participants who responded to the SA items in the follow-up survey, the average age was 57.5 years (SD = 13.9). A total of 621 participants (31.8%) self-reported a diagnosis of SA, and those who reported an SA diagnosis were slightly older than those who did not report an SA diagnosis (Table 1). Among those with SA diagnoses, a greater proportion self-identified as Black ($p < 0.001$), had a higher BMI ($p < 0.001$), started football at an older age ($p = 0.028$), had higher CSS scores ($p < 0.001$), endorsed the linemen field position ($p < 0.001$), reported an earlier professional debut year ($p = 0.003$), and used performance-enhancing drugs at some point in their career ($p = 0.007$). The magnitude of these differences ranged from negligible to small (Table 1).

We compared STOP-BANG scores and CPAP usage between participants who reported SA and those who did not (excluding those who did not provide complete STOP-BANG data, n = 101, 5.1%; Table 2). Those who self-reported an SA diagnosis had higher scores on the STOP-BANG questionnaire ($p < 0.001$, $\eta^2 = 0.28$, large effect; Table 2). Individual items on the STOP-BANG questionnaire were more likely to be positively endorsed by the SA group compared with those who did not report an SA diagnosis. For those who reported a diagnosis of SA, 341 (54.9%) reported never using a CPAP; 33 (5.4%) reported using CPAP 1–3 days per week, and 247 (39.8%) reported using their CPAP 4 or more times per week. A total of 992 participants who did not report an SA diagnosis screened positive for SA on the STOP-BANG. Specifically among this group, 732 participants (57.8%) were in the intermediate STOP-BANG range (STOP-BANG score = 3–4) and 260 (20.5%) were in the high STOP-BANG range (STOP-BANG score = 5–8).

Regarding health outcomes and current symptomatology, those who reported having a diagnosis of SA had higher symptom burdens on measures assessing recent anxiety, depression, and pain intensity (*p* values <0.001, small effect sizes; Table 3). They also reported worse perceived cognitive functioning on the Neuro-QoL ($p < 0.001$, small effect size) within the previous week. A higher proportion of the SA group reported having a variety of health conditions (Table 3), such as headaches (16.5% vs 6.5%, $p < 0.001$, Cramer $V = 0.16$), low testosterone (27.6% vs 10.7%, $p <$

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Table 1 Demographic and Football-Related Exposures for All Former Players and Stratified by Self-Reported Sleep Apnea Diagnosis

Characteristic	Total (N = 1,951)	No self-reported sleep apnea (N = 1,330)	Self-reported sleep apnea (N = 621)	*p* Value	Effect size
Age, y, mean (SD)	57.49 (13.87)	56.36 (14.43)	59.91 (12.24)	<0.001	η^2 = 0.01 small
Race, n (%)				<0.001	Cramer *V* = 0.12
Black	587 (30.1)	361 (27.1)	226 (36.4)		
Native Hawaiian/Pacific Islander/Asian/ American Indian/Alaskan Native	57 (2.9)	30 (2.3)	27 (4.3)		
White	1,284 (65.8)	921 (69.2)	363 (58.5)		
Missing	23 (1.2)	18 (1.4)	5 (0.8)		
Body mass index during professional play, mean (SD)	30.39 (4.06)	30.16 (4.01)	30.89 (4.10)	<0.001	η^2 = 0.03 small
Age at first football exposure, y, mean (SD)	11.76 (3.07)	11.66 (3.01)	11.99 (3.17)	0.028	η^2 = 0.00 small
N-Miss	24	15	9		
Career duration (seasons), n (%)				0.442	Cramer *V* = 0.03
1–6 seasons	1,077 (55.2)	747 (56.2)	330 (53.1)		
7–12 seasons	710 (36.4)	475 (35.7)	235 (37.8)		
13+ seasons	168 (8.5)	112 (8.2)	56 (9.0)		
Concussion signs and symptoms score, range	28.76 (25.68)	26.57 (23.94)	33.45 (28.52)	<0.001	Cramer *V* = 0.10
Quartile 1, 0–11	491 (25.3)	369 (27.7)	122 (20.0)	<0.001	
Quartile 2, 11–23	544 (28.0)	382 (28.6)	162 (26.6)		
Quartile 3, 23–44	496 (25.5)	329 (24.7)	167 (27.4)		
Quartile 4, 44–130	413 (21.2)	254 (19.0)	159 (26.1)		
N-Miss	36	25	11		
Linemen position, n (%)	683 (35.0)	433 (32.6)	250 (40.3)	<0.001	Cramer *V* = 0.08
First calendar year of professional play, mean	1980	1984	1971	0.003	η^2 = 0.03 small
N-Miss	4	3	1		
Previous use of performance-enhancing drugs, n (%)	304 (15.6)	187 (14.1)	117 (18.8)	0.007	Cramer *V* = 0.06

0.001, Cramer *V* = 0.21), and pain medication use (26.0% vs 14.9%) when compared with those who did not report an SA diagnosis.

Multivariable Associations Between Participant Characteristics and Self-Reported SA Outcomes

Two multivariable logistic regressions were used to explore associations between demographic and football-related exposures and (1) self-reported SA diagnoses and (2) intermediate/high risk based on the STOP-BANG. Identifying as a player of non-White race and having a higher career BMI were associated with both outcomes (Table 4 provides odds ratios [ORs], CIs, and *p* values). Older age was associated with self-reporting an OSA diagnosis (OR 1.04, 95% CI 1.03–1.05, $p < 0.001$). Higher reported head injury exposure (numbers of reported concussion signs and symptoms) was associated with higher odds of self-reporting an OSA diagnosis (e.g., quartile 4, OR 1.99, 95% CI 1.47–2.71, $p < 0.001$). Associations between head injury exposure and intermediate or high STOP-BANG scores were similarly positive (e.g., quartile 4, OR 1.97, 95% CI 1.30–3.00, $p < 0.001$; Table 4). The magnitude of these associations did not substantially differ in bivariable associations between the SA outcomes and each of the demographic and football-related variables (eTable 2).

Associations Between the SA Risk Variable and Current Neuropsychiatric Symptoms

To determine whether evidence of OSA (either self-reported diagnoses or intermediate/high STOP-BANG scores) and OSA treatment status were associated with current health outcomes, we used a composite sleep measure (in Methods) as an independent variable in 4 separate regression models that predicted recent anxiety symptoms (in the previous 2 weeks), self-reported cognitive symptoms (in the previous

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Table 2 STOP-BANG Questionnaire Responses for All Players and Stratified by Sleep Apnea Diagnosis

Characteristic	Total (N = 1,951)	No self-reported sleep apnea (N = 1,330)	Self-reported sleep apnea (N = 621)	*p* Value	Effect size
CPAP use, n (%)				<0.001	Cramer *V* = 0.62
None	1,671 (85.7)	1,330 (100.0)	341 (54.9)		
1–3 d/wk	33 (1.7)	NA	33 (5.3)		
4+ d/wk	247 (12.7)	NA	247 (39.8)		
STOP-BANG score, mean (SD)	4.08 (1.54)	3.53 (1.25)	5.28 (1.45)	<0.001	η^2 = 0.28 large
N-Miss	101	63	38		
STOP-BANG score, n (%)				<0.001	Cramer *V* = 0.54
1	27 (1.5)	27 (2.1)	0 (0.0)		
2	267 (14.4)	248 (19.6)	19 (3.3)		
3	435 (23.5)	384 (30.3)	51 (8.7)		
4	451 (24.4)	348 (27.5)	103 (17.7)		
5	317 (17.1)	174 (13.7)	143 (24.5)		
6	208 (11.2)	68 (5.4)	140 (24.0)		
7	113 (6.1)	15 (1.2)	98 (16.8)		
8	32 (1.7)	3 (0.2)	29 (5.0)		
N-Miss	101	63	38		
STOP-BANG categories, n (%)				<0.001	Cramer *V* = 0.49
Low (0–2)	294 (15.9)	275 (21.7)	19 (3.3)		
Intermediate (3–4)	886 (47.9)	732 (57.8)	154 (26.4)		
High (5–8)	670 (36.2)	260 (20.5)	410 (70.3)		
N-Miss	101	63	38		
STOP-BANG reported snoring, n (%)	827 (42.9)	427 (32.5)	400 (65.3)	<0.001	Cramer *V* = 0.31
N-Miss	23	15	8		
STOP-BANG reported fatigue, n (%)	725 (37.4)	392 (29.6)	333 (54.3)	<0.001	Cramer *V* = 0.24
N-Miss	13	5	8		
STOP-BANG reported observed disordered breathing, n (%)	532 (27.7)	141 (10.7)	391 (64.5)	<0.001	Cramer *V* = 0.56
N-Miss	27	12	15		
STOP-BANG high blood pressure, n (%)	661 (34.4)	374 (28.5)	287 (47.0)	<0.001	Cramer *V* = 0.18
N-Miss	29	18	11		
STOP-BANG neck circumference >16 in, n (%)	1,489 (76.3)	978 (73.5)	511 (82.3)	<0.001	Cramer *V* = 0.10
STOP-BANG BMI over 35, n (%)	347 (17.9)	147 (11.1)	200 (32.5)	<0.001	Cramer *V* = 0.26
N-Miss	14	8	6		
STOP-BANG age over 50, n (%)	1,356 (69.5)	870 (65.4)	486 (78.3)	<0.001	Cramer *V* = 0.12

Abbreviations: BMI = body mass index; CPAP = continuous positive airway pressure; N = sample size; N-miss = number of participants without data for that specific analysis; NA = not applicable.

week), depression symptoms (in the previous 2 weeks), and chronic pain. Models were further adjusted for age, race, linemen status, CSS score, career duration, career BMI, and use of performance-enhancing drugs. Compared with those with no OSA diagnosis or risk, participants with any OSA risk (at-risk and diagnosed, diagnosed and treated, diagnosed and

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Table 3 Health Outcomes for All Players and Stratified by Sleep Apnea Diagnosis

	Total (N = 1,951)	No self-reported sleep apnea (N = 1,330)	Self-reported sleep apnea (N = 621)	*p* Value	Effect size
PHQ-9 score, mean (SD)	4.87 (5.68)	4.10 (5.16)	6.52 (6.37)	<0.001	η^2 = 0.05 small
Cognitive symptoms (Neuro-QoL), mean (SD)	42.66 (9.59)	43.87 (9.36)	40.09 (9.58)	<0.001	η^2 = 0.04 small
N-Miss	18	15	3		
GAD-7, mean (SD)	3.69 (4.77)	3.20 (4.39)	4.73 (5.35)	<0.001	η^2 = 0.03 small
N-Miss	13	5	8		
Pain intensity, mean (SD)	4.15 (2.06)	3.90 (2.01)	4.67 (2.05)	<0.001	η^2 = 0.03 small
N-Miss	112	93	19		
Current Rx pain medication, n (%)	359 (18.4)	198 (14.9)	161 (26.0)	<0.001	Cramer *V* = 0.13
N-Miss	2	1	1		
Headaches, n (%)	184 (9.7)	85 (6.5)	99 (16.5)	<0.001	Cramer *V* = 0.15
N-Miss	49	27	22		
Current BMI, mean (SD)	30.85 (4.91)	29.85 (4.14)	33.01 (5.69)	<0.001	η^2 = 0.12 moderate
N-Miss	14	8	6		
ADHD, n (%)	139 (7.4)	71 (5.5)	68 (11.4)	<0.001	Cramer *V* = 0.10
N-Miss	62	38	24		
Diabetes, n (%)	209 (11.3)	101 (8.0)	108 (18.4)	<0.001	Cramer *V* = 0.15
N-Miss	108	75	33		
High cholesterol, n (%)	649 (34.6)	386 (30.0)	263 (44.6)	<0.001	Cramer *V* = 0.13
N-Miss	74	43	31		
Stroke, n (%)	55 (2.9)	27 (2.1)	28 (4.6)	0.002	Cramer *V* = 0.07
N-Miss	53	38	15		
Heart condition, n (%)	325 (17.7)	165 (13.1)	160 (27.7)	<0.001	Cramer *V* = 0.16
N-Miss	117	74	43		
Vascular dementia, n (%)	37 (1.9)	14 (1.1)	23 (3.8)	<0.001	Cramer *V* = 0.09
N-Miss	21	10	11		
Alzheimer disease, n (%)	31 (1.6)	13 (1.0)	18 (2.9)	0.001	Cramer *V* = 0.09
N-Miss	17	8	9		
Other dementia, n (%)	106 (5.5)	50 (3.8)	56 (9.2)	<0.001	Cramer *V* = 0.10
N-Miss	24	12	12		
CTE, n (%)	90 (4.7)	44 (3.3)	46 (7.5)	<0.001	Cramer *V* = 0.09
N-Miss	20	9	11		
Low testosterone, n (%)	309 (16.1)	141 (10.7)	168 (27.6)	<0.001	Cramer *V* = 0.21
N-Miss	26	14	12		

Abbreviations: BMI = body mass index; CTE = Chronic Traumatic Encephalopathy; GAD-7 = Generalized Anxiety Disorder; Neuro-QoL = Quality of Life in Neurological Disorders; PHQ-9 = 9-item Patient Health Questionnaire.

untreated) reported significantly more pain (β range = 0.46–0.91), anxiety (β range = 1.38–2.52), depression (β range = 1.93–3.51), and cognitive symptoms (β range = 2.26–4.93; Figure 1; eTable 3: models 5–8). Those with reported SA diagnoses who did not use a CPAP reported the most anxiety, depression, pain, and cognitive symptoms

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Table 4 Two Exploratory Multivariable Logistic Regressions Examining Associations With (1) Self-Reported Sleep Apnea Diagnosis (Model 1) and (2) Being at Risk of Sleep Apnea Using the STOP-BANG (Model 2)

	Model 1: DV: Self-reported sleep apnea diagnosis	Model 2: DV: STOP-BANG ≥3
	OR (95% CI), *p* value	OR (95% CI), *p* value
Age	1.04 (1.03–1.05), <0.001	1.02 (0.95–1.03), 0.63
Race (reference = White)		
Black	2.23 (1.77–2.81), <0.001	2.34 (1.70–3.24), <0.001
Native Hawaiian/Pacific Islander/Asian/American Indian/Alaskan Native	2.44 (1.36–4.36), 0.002	3.56 (1.35–12.34), 0.02
Missing	0.52 (0.15–1.46), 0.25	0.43 (0.16–1.36), 0.13
Age at first football exposure	1.02 (0.99–1.06), 0.28	0.97 (0.93–1.02), 0.21
Concussion signs and symptoms score		
Quartile 2	1.33 (1.0–1.78), 0.05	1.21 (0.85–1.72), 0.29
Quartile 3	1.60 (1.19–2.14), 0.002	1.56 (1.08–2.27), 0.02
Quartile 4	1.99 (1.47–2.71), <0.001	1.97 (1.30–3.00), 0.001
Lineman position (reference = nonlineman)	0.94 (0.68–1.28), 0.68	0.81 (0.51–1.29), 0.38
Performance-enhancing drug history	1.30 (0.99–1.70), 0.06	0.90 (0.62–1.33), 0.59
Career duration (reference = 1–6 seasons)		
7–12 seasons	0.89 (0.72–1.11), 0.31	0.86 (0.64–1.15), 0.30
13+ seasons	1.02 (0.70–1.48), 0.90	0.98 (0.59–1.70), 0.94
First calendar year of professional play	1.00 (1.0–1.00), 0.34	0.96 (0.89–0.99), 0.19
Career body mass index	1.09 (1.05–1.14), <0.001	1.13 (1.07–1.19), <0.001

Abbreviations: DV = dependent variable; OR = odds ratio.
Each of the 2 models included all variables simultaneously, including for age, age at first exposure to football, race, concussion symptom score quartile, lineman status, career duration, debut year, body mass index during professional play, and previous use of performance-enhancing drugs. Concussion signs and symptoms quartile 1, nonlineman, no reported use of performance-enhancing drugs, career duration <7 seasons, White race, and absence of risk nor self-reporting sleep apnea served as reference values in each model.

(Figure 1; eTable 3). Unadjusted, bivariable logistic regressions yielded similar relationships between the SA groups and outcome variables at slightly different magnitudes (eTable 3: models 1–4). Sensitivity analyses further subdivided the "undiagnosed and at-risk" group into 2 nonreference groups based on their STOP-BANG scores (i.e., low = 0–2 [reference]; intermediate = 3–4; high = 5–8), leading to a total of 5 groups. Similar to Figure 1, these multivariable analyses show that both the intermediate-risk and high-risk undiagnosed groups also had an elevated risk of pain, depression, anxiety, and cognitive symptoms compared with those with low STOP-BANG scores (eFigure 2).

Discussion

In a cohort of 1,951 former professional football players, approximately one-third reported that they had been diagnosed with SA, and those who reported this diagnosis had a variety of differences regarding demographic (e.g., older age and Black race) and health factors that set them apart from those who did not endorse SA. Furthermore, this study suggested that those who were diagnosed with SA may not be adequately treated, such that only 40% of participants who reported an SA diagnosis reported regular CPAP usage while the remaining 60% reported only using their CPAP 0–3 times per week. Of interest, this undertreated SA was associated with worse clinical symptomatology, such that those with SA who did not regularly use a CPAP had worse scores on measures of depression, anxiety, pain, and cognitive dysfunction—even when statistically adjusting for a variety of other variables. Furthermore, results indicate that many former professional football players may have undiagnosed SA based on the STOP-BANG results but had not been evaluated or diagnosed at the time of this survey.

The proportion of former professional football players who reported having SA in this study (31.8%) is generally consistent with other studies. For instance, in a study of 120 former professional football players, 36.7% self-reported being diagnosed with SA, compared with 30% of former college football players and 16.7% of controls,[26] although another more recent survey of 1,973 reported a SA prevalence of 22.6%.[46] In another study,[26] age and Black race were

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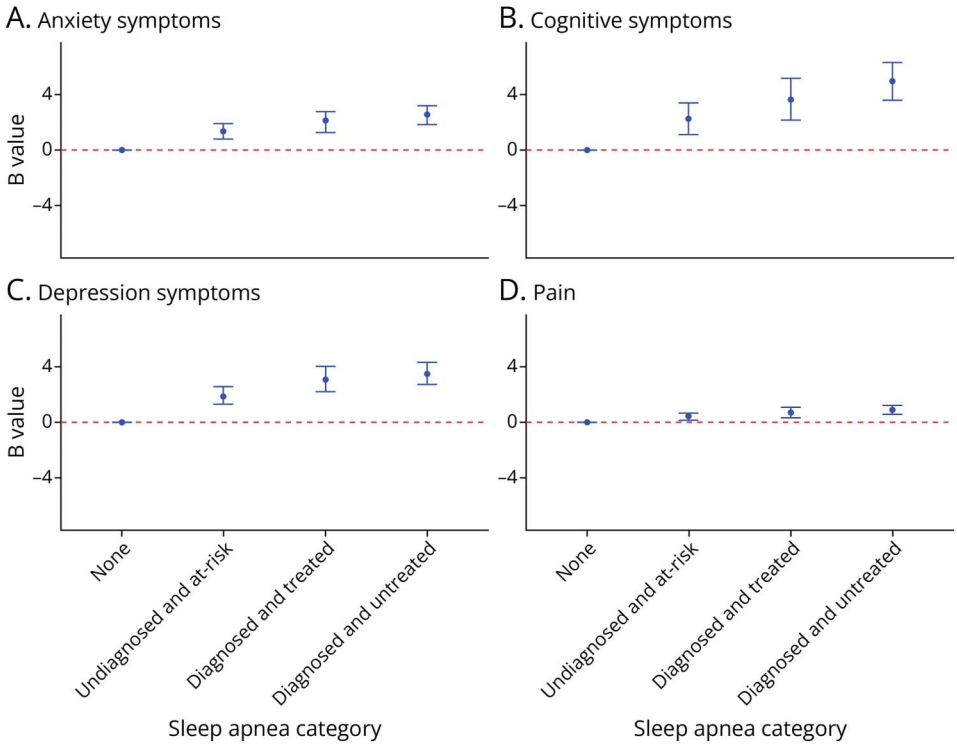
Figure 1 Multivariable Linear Models Examining the Relationship Between the Composite Sleep Apnea Risk Variable and Health Outcomes



β estimates with 95% CIs for 4 health outcomes by sleep apnea status. (A) Anxiety symptoms, (B) cognitive symptoms, (C) depression symptoms, and (D) pain. All models adjusted for age, race, linemen status, concussion signs and symptoms score, career duration, body mass index during active play, and use of performance-enhancing drugs. The red line denotes null value (0.0).

associated with specific SA symptoms and those with SA had worse cognitive test scores/higher depressive symptoms, but many of these relationships became null when CPAP treatment was added to the model. In this study, multivariable models identified several predictors of reporting an SA diagnosis such as age, race, higher football career BMI, and past use of performance-enhancing drugs. In addition, we found a dose-response relationship between past signs and symptoms of concussions and SA risk, such that the risk of reporting a SA diagnosis increased for each CSS score quartile. These findings align with other studies of TBI and sleep disorders among patient,[47] ASF,[46] and military[48] populations, supporting the hypothesis that concussion history may increase the likelihood of SA. The significant association between SA and Black race seen here and in a previous study[26] may reflect the disproportionate exposure to more dangerous football positions for Black players,[49] or other social determinants of health that are external to football play.

While 31.8% of this sample reported having already received a SA diagnosis, a significant portion of this sample may still be at risk of SA but have not yet received a diagnosis. Nearly 80% of the group without a current SA diagnosis scored in the intermediate (n = 732, 57.8%) or high STOP-BANG range (n = 260, 20.5%). While not all participants with at-risk STOP-BANG scores will have SA, nearly three-quarters of the general population who has a STOP-BANG score ≥3 will actually have a SA diagnosis, given its 0.74 positive predictive value (i.e., the ratio of [true positives] ÷ [true positives + false positives]).[19] If the psychometrics of this measure are the same for former professional football players, we estimate that 734 of the 992 participants with a STOP-BANG score ≥3 actually have SA. Because 621 participants self-reported being diagnosed with SA and we estimate that 734 participants without a current SA diagnosis may have SA based on their STOP-BANG score, we estimate that the true prevalence of SA in this sample is 69% ([621 + 734] ÷ 1,951). Specifically, we anticipate that over half of former professional football players who are not diagnosed with SA actually have this condition {[(732 + 260) × (0.74)]/1,330 = 55.5%}. It may be that those with the most severe/obvious cases of SA (i.e., have high scores on the STOP-BANG) were screened and diagnosed, but those in the intermediate-risk group may not be appropriately screened for this condition. Appropriately diagnosing and treating SA is especially important in this cohort, given that SA is associated with all-cause mortality,[6] cardiovascular events,[6,7] low testosterone,[8] depression,[6,7] diabetes,[6,7] neurodegenerative disease,[11,13,14] and neurocognitive dysfunction,[10]—and SA is a modifiable risk factor whose downstream effects may be accidently misattributed to other etiologies. Prospective screening programs and adequate treatment of former professional football players may be considered to ensure that this condition is comprehensively addressed by health professional.

There are several important limitations of this study. First, the diagnosis of SA is self-reported, and medical records were not

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available to review or cross-validate this diagnosis. Second, there are inherent characteristics about former professional football players that likely predispose them to having higher scores on the STOP-BANG (e.g., being male, high BMI, and large neck circumference). However, we have no evidence to suggest that the STOP-BANG would overestimate SA risk among ASF players simply because those risk factors are more common in this group. In fact, participants who met intermediate STOP-BANG criteria for SA showed significantly elevated risk of adverse outcomes, similar to those at high risk. There are few, if any, studies that examine the psychometric characteristics between STOP-BANG scores and OSA diagnosis in former professional football players. We have no reason to suspect that STOP-BANG scores of 3 or greater would overestimate risk in ASF players when compared with general populations, though this is a possibility. Future studies should investigate whether the STOP-BANG functions differently in this unique population. However, given that former professional football players likely possess many health factors known in the general population to increase the risk of SA, we believe that former professional football players who possess such risk factors be thoroughly screened. Third, while this is a large sample, those who chose to participate in this study may not represent the entire population of former professional football players. However, previous analyses suggest that age, BMI, position, and duration of play are consistent between former professional football players who participated in the FPHS survey and those who did not.[28] However, this study cannot account for those who may have passed away from conditions related to SA (e.g., cardiometabolic disease), because participation in this study conditions on surviving into postcareer years. Fourth, we did not analyze the potential role of other sleep disorders, alcohol use, cannabis use, or other current lifestyle factors in these analyses but acknowledge that these factors may affect current health status and/or interact with SA. Fifth, we acknowledge that not all individuals who have been diagnosed with SA may be recommended for CPAP treatment. Questionnaire data did not allow us to delineate between those who reported being diagnosed with SA but were nonadherent with CPAP recommendations and those who were not recommended to use CPAP. Sixth, these results cannot be generalized to other populations, like football players of other skill levels or other professional sports. Last, this survey inquired about "sleep apnea" and did not specify "obstructive sleep apnea" vs "central sleep apnea." While over 90% of diagnosed SA cases are due to OSA,[50] there is the potential for some study participants to have central SA when they responded to the question "Has a health care provider ever told you that you have …sleep apnea?"

This study elucidates the demographic, clinical, and football-related characteristics of self-reported SA in a cohort of former professional football players. It also highlights that those with SA may not be appropriately treating this condition, and that many participants without a current SA diagnosis may—in fact—have this condition. Last, analyses showed differences on clinical outcomes (e.g., psychological symptoms, cognitive symptoms, and pain) based on SA status and CPAP compliance. These findings suggest that additional awareness about SA, screening programs, and consistent follow-ups may be instrumental in optimizing the long-term health of former professional football players.

Author Contributions

D.P. Terry: drafting/revision of the manuscript for content, including medical writing for content; study concept or design; analysis or interpretation of data. R. Grashow: drafting/revision of the manuscript for content, including medical writing for content; major role in the acquisition of data; study concept or design; analysis or interpretation of data. C. Creager: drafting/revision of the manuscript for content, including medical writing for content. S. Bertisch: drafting/revision of the manuscript for content, including medical writing for content. S. Redline: drafting/revision of the manuscript for content, including medical writing for content. J. Kim: drafting/revision of the manuscript for content, including medical writing for content. M.G. Weisskopf: drafting/revision of the manuscript for content, including medical writing for content; study concept or design. A. Baggish: drafting/revision of the manuscript for content, including medical writing for content. R.D. Zafonte: drafting/revision of the manuscript for content, including medical writing for content; major role in the acquisition of data.

Study Funding

The Football Players Health Study is supported by the NFL Players Association. The NFL Players Association did not contribute to the design and conduct of the study; collection, management, analysis, and interpretation of the data; preparation, review, or approval of the manuscript; and decision to submit the manuscript for publication. This work received support from Harvard Catalyst | The Harvard Clinical and Translational Science Center (National Center for Advancing Translational Sciences, NIH Award UL1 TR002541) and financial contributions from Harvard University and its affiliated academic health care centers. The content is solely the responsibility of the authors and does not necessarily represent the official views of Harvard Catalyst, Harvard University and its affiliated academic health care centers, or the NIH.

Disclosure

D.P. Terry serves as a consultant for the National Football League (NFL; Senior Director of Research) and a scientific advisor for HitIQ; previously consulted for REACT Neuro Inc.; receives research funding from Football Research Inc. and Amgen Inc.; and has a consulting practice in forensic neuropsychology, including expert testimony. R. Grashow received grant funding from the NFL Players Association. C. Creager reports no disclosures relevant to the manuscript. S. Bertisch receives funding from the NIH, PCORI, and the American Academy of Sleep Medicine Foundation; and has consulted for Idorsia and Apnimed. S. Redline receives funding from the NIH; has consulted for Eli Lilly Inc.; is an unpaid consultant for Apnimed Inc.; is an unpaid board

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member for the National Sleep Foundation and Alliance of Sleep Apnea Partners; and is Editor-In-Chief for the journal *Sleep Health*. J. Kim receives grant funding from the National Institute of Health/National Heart, Lung, and Blood Institute, and the National Football Players Association; and receives compensation for his role as team cardiologist for the Atlanta Falcons, Atlanta Hawks, Atlanta United, and Atlanta Braves. M.G. Weisskopf reported receiving grants from the National Football League Players Association (NFLPA) and the NIH during the conduct of the study. A. Baggish has received funding from the National Institute of Health/National Heart, Lung, and Blood Institute; the National Football Players Association; and the American Heart Association; and receives compensation for his role as team cardiologist from the US Olympic Committee/US Olympic Training Centers, US Soccer, and the National Women's Soccer League. R.D. Zafonte reported receiving royalties from Springer/Demos publishing for serving as coeditor of the text Brain Injury Medicine and serving on the scientific advisory board of Myomo Inc. and NanoDx; he previously evaluated patients in the Massachusetts General Hospital Brain and Body-TRUST Program, which is funded by the NFL Players Association; and reports receiving grants from the NIH. Go to Neurology.org/N for full disclosures.

Publication History

Received by *Neurology*® July 29, 2025. Accepted in final form February 11, 2026. Submitted and externally peer reviewed. The handling editor was Associate Editor Emily Johnson, MD, MPH.

References

1. Al Lawati NM, Patel SR, Ayas NT. Epidemiology, risk factors, and consequences of obstructive sleep apnea and short sleep duration. *Prog Cardiovasc Dis*. 2009;51(4):285-293. doi:10.1016/j.pcad.2008.08.001
2. Chervin RD. Sleepiness, fatigue, tiredness, and lack of energy in obstructive sleep apnea. *Chest*. 2000;118(2):372-379. doi:10.1378/chest.118.2.372
3. Johns MW. A new method for measuring daytime sleepiness: the Epworth sleepiness scale. *Sleep*. 1991;14(6):540-545. doi:10.1093/sleep/14.6.540
4. Russell MB, Kristiansen HA, Kværner KJ. Headache in sleep apnea syndrome: epidemiology and pathophysiology. *Cephalalgia*. 2014;34(10):752-755. doi:10.1177/0333102414538551
5. Myers KA, Mrkobrada M, Simel DL. Does this patient have obstructive sleep apnea? The Rational Clinical Examination systematic review. *JAMA*. 2013;310(7):731-741. doi:10.1001/jama.2013.276185
6. Kendzerska T, Mollayeva T, Gershon AS, Leung RS, Hawker G, Tomlinson G. Untreated obstructive sleep apnea and the risk for serious long-term adverse outcomes: a systematic review. *Sleep Med Rev*. 2014;18(1):49-59. doi:10.1016/j.smrv.2013.01.003
7. Knauert M, Naik S, Gillespie MB, Kryger M. Clinical consequences and economic costs of untreated obstructive sleep apnea syndrome. *World J Otorhinolaryngol Head Neck Surg*. 2015;1:17-27. doi:10.1016/j.wjorl.2015.08.001
8. Wang H, Lu J, Xu L, et al. Obstructive sleep apnea and serum total testosterone: a system review and meta-analysis. *Sleep Breath*. 2023;27(3):789-797. doi:10.1007/s11325-022-02655-6
9. Brüdern J, Hallensleben N, Höller I, et al. Sleep disturbances predict active suicidal ideation the next day: an ecological momentary assessment study. *BMC Psychiatry*. 2022;22(1):65. doi:10.1186/s12888-022-03716-6
10. Seda G, Han TS. Effect of obstructive sleep apnea on neurocognitive performance. *Sleep Med Clin*. 2020;15(1):77-85. doi:10.1016/j.jsmc.2019.10.001
11. Wang C, Holtzman DM. Bidirectional relationship between sleep and Alzheimer's disease: role of amyloid, tau, and other factors. *Neuropsychopharmacology*. 2020;45(1):104-120. doi:10.1038/s41386-019-0478-5
12. Ercolano E, Bencivenga L, Palaia ME, et al. Intricate relationship between obstructive sleep apnea and dementia in older adults. *GeroScience*. 2024;46(1):99-111. doi:10.1007/s11357-023-00958-4
13. Andrade AG, Bubu OM, Varga AW, Osorio RS. The relationship between obstructive sleep apnea and Alzheimer's disease. *J Alzheimers Dis*. 2018;64(s1):S255-S270. doi:10.3233/JAD-179936
14. Sun A-P, Liu N, Zhang Y-S, Zhao H-Y, Liu X-L. The relationship between obstructive sleep apnea and Parkinson's disease: a systematic review and meta-analysis. *Neurol Sci*. 2020;41(5):1153-1162. doi:10.1007/s10072-019-04211-9
15. Young T, Evans L, Finn L, Palta M. Estimation of the clinically diagnosed proportion of sleep apnea syndrome in middle-aged men and women. *Sleep*. 1997;20(9):705-706. doi:10.1093/sleep/20.9.705
16. Finkel KJ, Searleman AC, Tymkew H, et al. Prevalence of undiagnosed obstructive sleep apnea among adult surgical patients in an academic medical center. *Sleep Med*. 2009;10(7):753-758. doi:10.1016/j.sleep.2008.08.007
17. Chung F, Abdullah HR, Liao P. STOP-Bang questionnaire: a practical approach to screen for obstructive sleep apnea. *Chest*. 2016;149(3):631-638. doi:10.1378/chest.15-0903
18. Nagappa M, Liao P, Wong J, et al. Validation of the STOP-Bang questionnaire as a screening tool for obstructive sleep apnea among different populations: a systematic review and meta-analysis. *PLoS One*. 2015;10(12):e0143697. doi:10.1371/journal.pone.0143697
19. Chen L, Pivetta B, Nagappa M, et al. Validation of the STOP-Bang questionnaire for screening of obstructive sleep apnea in the general population and commercial drivers: a systematic review and meta-analysis. *Sleep Breath*. 2021;25(4):1741-1751. doi:10.1007/s11325-021-02299-y
20. George CFP, Kab V, Kab P, Villa JJ, Levy AM. Sleep and breathing in professional football players. *Sleep Med*. 2003;4:317-325. doi:10.1016/s1389-9457(03)00113-8
21. George CFP, Kab V, Levy AM. Increased prevalence of sleep-disordered breathing among professional football players. *N Engl J Med*. 2003;348(4):367-368. doi:10.1056/NEJM200301233480422
22. Luyster FS, Dunn RE, Lauderdale DS, et al. Sleep-apnea risk and subclinical atherosclerosis in early-middle-aged retired National Football League players. *Nat Sci Sleep*. 2017;9:31-38. doi:10.2147/NSS.S125228
23. Rice TB, Dunn RE, Lincoln AE, et al. Sleep-disordered breathing in the National Football League. *Sleep*. 2010;33(6):819-824. doi:10.1093/sleep/33.6.819
24. Albuquerque FN, Kuniyoshi FHS, Calvin AD, et al. Sleep-disordered breathing, hypertension, and obesity in retired National Football League players. *J Am Coll Cardiol*. 2010;56(17):1432-1433. doi:10.1016/j.jacc.2010.03.099
25. Franklin KA, Lindberg E. Obstructive sleep apnea is a common disorder in the population-a review on the epidemiology of sleep apnea. *J Thorac Dis*. 2015;7(8):1311-1322. doi:10.3978/j.issn.2072-1439.2015.06.11
26. Banks SJ, Yhang E, Tripodis Y, et al. Clinical outcomes and tau pathology in retired football players: associations with diagnosed and witnessed sleep apnea. *Neurol Clin Pract*. 2024;14(2):e200263. doi:10.1212/CPJ.0000000000200263
27. Terry DP, Grashow R, Iverson GL, et al. Age of first exposure does not relate to postcareer health in former professional American-style football players. *Sports Med*. 2024;54(10):2675-2684. doi:10.1007/s40279-024-02062-9
28. Zafonte R, Pascual-Leone A, Baggish A, et al. The Football Players' Health Study at Harvard University: design and objectives. *Am J Ind Med*. 2019;62(8):643-654. doi:10.1002/ajim.22991
29. Harrison EA. The first concussion crisis: head injury and evidence in early American football. *Am J Public Health*. 2014;104(5):822-833. doi:10.2105/AJPH.2013.301840
30. Grashow R, Weisskopf MG, Miller KK, et al. Association of concussion symptoms with testosterone levels and erectile dysfunction in former professional US-style football players. *JAMA Neurol*. 2019;76(12):1428-1438. doi:10.1001/jamaneurol.2019.2664
31. Grashow R, Tan CO, Thomas JD, et al. Career duration and later-life health conditions among former professional American-style football players. *Occup Environ Med*. 2024;81(10):498-506. doi:10.1136/oemed-2024-109571
32. Roberts AL, Pascual-Leone A, Speizer FE, et al. Exposure to American football and neuropsychiatric health in former national football league players: findings from the football players health study. *Am J Sports Med*. 2019;47(12):2871-2880. doi:10.1177/0363546519868989
33. Roberts AL, Zafonte RD, Speizer FE, et al. Modifiable risk factors for poor cognitive function in former American-style football players: findings from the Harvard Football Players Health Study. *J Neurotrauma*. 2021;38(2):189-195. doi:10.1089/neu.2020.7070
34. Grashow R, Tan CO, Izzy S, et al. Association between concussion burden during professional American-style football and postcareer hypertension. *Circulation*. 2023;147(14):1112-1114. doi:10.1161/CIRCULATIONAHA.122.063767
35. Peker Y, Hedner J, Johansson A, Bende M. Reduced hospitalization with cardiovascular and pulmonary disease in obstructive sleep apnea patients on nasal CPAP treatment. *Sleep*. 1997;20(8):645-653. doi:10.1093/sleep/20.8.645
36. Cella D, Lai J-S, Nowinski CJ, et al. Neuro-QOL: brief measures of health-related quality of life for clinical research in neurology. *Neurology*. 2012;78(23):1860-1867. doi:10.1212/WNL.0b013e318258f744
37. Spitzer RL, Kroenke K, Williams JBW, Löwe B. A brief measure for assessing generalized anxiety disorder: the GAD-7. *Arch Intern Med*. 2006;166(10):1092-1097. doi:10.1001/archinte.166.10.1092
38. Bauer AM, Chan Y-F, Huang H, Vannoy S, Unützer J. Characteristics, management, and depression outcomes of primary care patients who endorse thoughts of death or suicide on the PHQ-9. *J Gen Intern Med*. 2013;28(3):363-369. doi:10.1007/s11606-012-2194-2
39. Cleeland CS, Ryan KM. Pain assessment: global use of the Brief Pain Inventory. *Ann Acad Med Singap*. 1994;23(2):129-138.
40. Fritz CO, Morris PE, Richler JJ. Effect size estimates: current use, calculations, and interpretation. *J Exp Psychol Gen*. 2012;141(1):2-18. doi:10.1037/a0024338
41. Cohen J. A power primer. *Psychol Bull*. 1992;112(1):155-159. doi:10.1037//0033-2909.112.1.155
42. R Core Team. *R: A Language and Environment for Statistical Computing*. R Foundation for Statistical Computing [online serial]; R-project.org/ (2021, December 12, 2024).

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43. Eekhout I, de Vet HCW, Twisk JWR, Brand JPL, de Boer MR, Heymans MW. Missing data in a multi-item instrument were best handled by multiple imputation at the item score level. *J Clin Epidemiol*. 2014;67(3):335-342. doi: 10.1016/j.jclinepi.2013.09.009
44. Schafer JL, Graham JW. Missing data: our view of the state of the art. *Psychol Methods*. 2002;7(2):147-177.
45. Peterson ED, Delong ER, Masoudi FA, et al. ACCF/AHA 2010 position statement on composite measures for healthcare performance assessment: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Performance Measures (Writing Committee to develop a position statement on composite measures). *Circulation*. 2010;121(15):1780-1791. doi: 10.1161/CIR.0b013e3181d2ab98
46. Brett BL, Sullivan ME, Aggarwal NT, et al. Higher cardiovascular risk factor burden associated with cumulative concussion history and age-related differences. *J Head Trauma Rehabil*. 2025. Published December 22, 2025. doi: 10.1097/HTR.0000000000001140.
47. Izzy S, Chen PM, Tahir Z, et al. Association of traumatic brain injury with the risk of developing chronic cardiovascular, endocrine, neurological, and psychiatric disorders. *JAMA Netw Open*. 2022;5(4):e229478. doi:10.1001/jamanetworkopen.2022.9478
48. Leng Y, Byers AL, Barnes DE, Peltz CB, Li Y, Yaffe K. Traumatic brain injury and incidence risk of sleep disorders in nearly 200,000 US veterans. *Neurology*. 2021; 96(13):e1792-e1799. doi:10.1212/WNL.0000000000011656
49. Marquez-Velarde G, Grashow R, Glass C, et al. The paradox of integration: racial composition of NFL positions from 1960 to 2020. *Sociol Race Ethn*. 2023;9(4): 451-469. doi:10.1177/23326492231182597
50. Donovan LM, Kapur VK. Prevalence and characteristics of central compared to obstructive sleep apnea: analyses from the sleep heart health study cohort. *Sleep*. 2016; 39(7):1353-1359. doi:10.5665/sleep.5962

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