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# Estimating the Prevalence of Sleep-Disordered Breathing Among Collegiate Football Players

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**BACKGROUND: Obstructive sleep apnea is a clinical disorder characterized by loud snoring, apneic episodes, and chronic sleep disruption. Collegiate football players exhibit several risk factors for OSA, including large neck circumference and high body mass index, although the prevalence of OSA in this cohort is unknown. METHODS: The STOP-BANG questionnaire was administered at random to members of a collegiate football team and used to stratify the players into high and low risk for sleep-disordered breathing (SDB). Those who completed the questionnaire were then evaluated for SDB during preseason camp using a single-channel (finger pulse oximetry) photoplethysmography-based device. SDB was defined as an apnea-hypopnea index of ≥5. RESULTS: Of 56 players who underwent overnight photoplethysmography monitoring, valid results were available for 51. Forty-eight percent of the players were high-risk (neck size = 44.6 ± 2.2 cm, body mass index = 33.0 ± 5.4) versus low-risk (neck size = 41.4 ± 2.8 cm, body mass index = 27.6 ± 3.6) (both *P* values <.001). An apnea-hypopnea index of ≥5 was found in 2 (8.3%, 95% CI 1.0–20.0%) high-risk and 2 (7.7, 95% CI 1.0–18.4%) low-risk players. Two offensive linemen, a linebacker, and a tight end accounted for the positive cases. CONCLUSIONS: Based on our sample, we estimate the prevalence of SDB among collegiate football players to be 8%, regardless of risk stratification. Given the strong link between SDB and cardiovascular disease, these data underscore the importance of screening and subsequent treatment of SDB in this highly conditioned yet potentially vulnerable group of athletes.** *Key words: photoplethysmography; athletes; sleep disorders; STOP-BANG; dual energy x-ray absorptiometry (DXA).* [Respir Care 0;0(0):1–•. © 0 Daedalus Enterprises]

## Introduction

Obstructive sleep apnea (OSA) is a clinical disorder characterized by loud snoring, apneic episodes (cessation of breathing) and chronic sleep disruption.[1] The estimated prevalence of OSA among United States adults is 4%,[2] and prospective studies have demonstrated that OSA has dire effects on cardiovascular health.[2,3] Although OSA affects primarily middle to older age adults, it can occur in younger individuals.[4] American football players represent a unique cohort of young adults who are well-conditioned athletes but have several risk factors for OSA, including

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male sex, high body mass index, and a large neck circumference. Indeed, others[5,6] have demonstrated that OSA, or sleep-disordered breathing (SDB), is present in approximately 14–19% of professional football players. This represents an approximately 5-fold higher prevalence compared with young community-based volunteers,[7] yet thousands of young men of similar size play football at the collegiate level and are likely to go undiagnosed. Since alertness, reaction time, and concentration are linked to proper sleep,[8] those with SDB may experience detriments in athletic as well as academic performance. Importantly, given the strong association between SDB and the development of hypertension,[2] these athletes may suffer from greater cardiovascular disease burden. Although treatment of SDB will undoubtedly minimize cardiovascular disease risk, the presence of this disorder among collegiate football players must first be recognized.

Accordingly, the purpose of the present investigation was to estimate the prevalence of SDB in collegiate football players. A secondary aim was to evaluate the relationship between markers of SDB and body composition parameters using state-of-the-art dual-energy x-ray absorptiometry imaging. We hypothesized that the prevalence of SDB among collegiate football players would be similar to that observed in professional players and that total body and regional body fat would be higher among those who exhibit SDB compared with those without the disorder.

**QUICK LOOK**

**Current knowledge**

Sleep-disordered breathing (SDB) is a common disorder associated with obesity that has dire consequences on cardiovascular health. The prevalence of SDB among professional football players is approximately 14–19%, which represents an approximately 5-fold higher prevalence compared with young, community-based volunteers. Thousands of young men of similar size play football at the collegiate level, yet the prevalence of SDB in this cohort has not been studied.

**What this paper contributes to our knowledge**

Using overnight photoplethysmography, we found the estimated prevalence of SDB in collegiate football players to be 8%, regardless of risk stratification. Thirty-five percent of the players reported a clinically important degree of daytime sleepiness. Greater total body and central fat, as assessed by dual-energy x-ray absorptiometry, was observed in those with greater oxygen desaturation index.

## Methods

### Study Participants

The study sample was randomly selected from the 100-man roster of the Towson University Division 1AA collegiate football team. Testing took place during a preseason mini-camp in August 2014. The Towson University Institutional Review Board approved the study protocol, and each participant provided written informed consent. All players were eligible to participate. Individuals were excluded if they had previously been diagnosed with a sleep disorder.

Participants completed a STOP-BANG questionnaire and Epworth Sleepiness Scale to assess risk for SDB and level of daytime sleepiness, respectively. Players were given a Morpheus Ox (WideMed Ltd., Herziliya, Israel) portable single-channel (pulse oximeter), photoplethysmography (PPG)-based sleep-monitoring device to be worn for one night. An athletic trainer provided instructions for home use. Players were encouraged not to alter sleep patterns. Within 3 weeks of sleep monitoring, body composition was assessed using dual-energy x-ray absorptiometry.

### STOP-BANG Questionnaire

The STOP-BANG questionnaire was utilized to provide risk stratification for SDB.[9] This questionnaire consists of 8 items that address the following risk factors: (1) snoring, (2) daily fatigue, (3) someone observing you to stop breathing during sleep, (4) high blood pressure, (5) high body mass index ($>35$ kg/m$^2$), (6) age $>50$ y, (7) large neck circumference ($>40$ cm), and (8) male sex. Neck circumference was measured around the thickest portion of the neck by an investigator. High risk for SDB was defined as $\geq 3$ affirmative answers to the 8 STOP-BANG items.

### Epworth Sleepiness Scale

The Epworth Sleepiness Scale (ESS) is a validated 8-item questionnaire that measures subjective sleepiness.[10] Participants were asked to rate how likely they are to fall asleep in the following situations: (1) sitting and reading, (2) watching television, (3) sitting inactive in a public place, (4) being a passenger in a car for at least 1 h, (5) lying down to rest in the afternoon, (6) sitting and talking to someone, (7) sitting quietly after lunch, and (8) driving a car stopped in traffic. Each question is scored from 0 to 3. ESS values range from 0 (unlikely to fall asleep in any situation) to 24 (high chance of falling asleep in all 8 situations). High risk for daytime sleepiness was defined as an ESS total score $>10$.

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### Morpheus Ox Sleep Monitoring Device

The theory behind the use of the Morpheus Ox device to obtain PPG-derived signals is described elsewhere.[11] Briefly, PPG uses a pulse oximeter to detect blood volume changes in the microvascular bed of the fingertip, and proprietary software[12,13] is used to analyze the PPG for baseline variations, envelope, and rate. These parameters are combined to generate a PPG-derived respiration waveform that is correlated with saturation reductions to detect a clinically relevant apnea-hypopnea index (AHI). In this study, we report a 4% (2007 American Academy of Sleep Medicine criteria) oxygen desaturation index (ODI4) and 3% ODI (ODI3) (2012 American Academy of Sleep Medicine criteria) for defining hypopneas. Furthermore, the PPG software platform facilitates the detection and measurement of respiratory events, sleep/wake epochs, and total sleep time.[12,13] Respiratory events occurring during sleep epochs are averaged over total sleep time to generate the AHI value. Data were excluded from analysis if the PPG estimated sleep time was <2 h.

### Body Composition

Height and weight were measured in metric units using a stadiometer and digital scale, respectively. Total body dual-energy x-ray absorptiometry scans were acquired in the frontal plane using the Lunar iDXA scanner (GE Healthcare, Madison, Wisconsin). Abdominal visceral fat was computed over the android region and reported in kg. The android region is roughly 10 cm in height, extending from the iliac crest toward the head, a height that is 20% of the distance from the iliac crest to the base of the mandible. Lunar iDXA scans were analyzed with the enCORE software, version 14.0 (GE Healthcare, Madison, Wisconsin).

### Statistical Analysis

All data are reported as mean SD. Independent-samples *t* tests were used to compare demographic and body composition parameters between players classified as high-risk versus low-risk based on responses to the STOP-BANG questionnaire. Confidence intervals are reported for SDB prevalence estimates. A $\phi$ coefficient of association was calculated to examine the differences between risk categories in the number of players with an ESS >10 and ODI ≥5 events/h. Independent-samples *t* tests were conducted to examine differences in body composition parameters between players who exhibited sleep parameters (AHI, ODI4, and ODI3) of ≥5 events/h versus those below this threshold. The limit for statistical significance was set at $P < .05$. Data analysis was conducted using Stata 10.1 (StataCorp, College Station, Texas).

## Results

Of 56 players who underwent overnight PPG monitoring, valid results were available for 51. The reason for insufficient data was that the Morpheus Ox device estimated sleep time of <2 h. The remaining sample consisted of 13 linebackers (25%), 10 offensive linemen (20%), 9 defensive linemen (18%), and 3 tight ends (6%). The remaining 31% of the cohort consisted of defensive backs, wide receivers, kickers, quarterbacks, and a long snapper. Seven linebackers, 6 offensive linemen, 8 defensive linemen, and one tight end were classified as high-risk for OSA, whereas only one wide receiver, a quarterback, and the long snapper were classified as high-risk based on the STOP-BANG questionnaire. Table 1 displays participant characteristics according to risk category. Fifty-six percent of the sample were African-American. The players classified as high-risk weighed more (115.5 ± 18.2 kg vs 95.6 ± 17.2 kg, $P < .001$) and had a larger neck circumference (44.6 ± 2.2 cm vs 41.4 ± 2.8 cm, $P < .001$) and higher body mass index (33.0 ± 5.4 kg/m$^2$ vs 27.6 ± 3.6 kg/m$^2$, $P < .001$) compared with the low-risk players.

Body composition parameters from the dual-energy x-ray absorptiometry scan are given in Table 2. Scheduling conflicts resulted in 4 players being unavailable for dual-energy x-ray absorptiometry testing. As expected, due to differences in weight, high-risk players had greater total lean mass (82.0 ± 7.9 kg vs 73.3 ± 8.4 kg, $P < .001$), total fat mass (26.5 ± 11.9 kg vs 20.1 ± 10.0 kg, $P = .043$) and trunk fat mass (12.6 ± 6.7 kg vs 9.1 ± 5.3 kg, $P = .047$). The large girth of several of the players required that we complete half-body scans, thereby precluding the software from estimating visceral fat in 6 players. No differences in visceral fat were observed between the high- and low-risk groups.

Regarding PPG-derived sleep parameters, total sleep time was low but did not differ between the high- and low-risk groups (4.2 ± 1.1 h vs 3.8 ± 0.9 h, $P = .17$). An AHI between 5 and 15 events/h was found in 2 (8.3%, 95% CI 1.0–20.0%) high-risk and 2 (7.7, 95%, CI 1.0–18.4%) low-risk players. Two offensive linemen (AHI = 7 and 11 events/h), a linebacker (AHI = 11 events/h), and a tight end (AHI = 9 events/h) accounted for the positive cases. No players were observed to have moderate to severe OSA, defined as having an AHI >15. Of the 4 positive cases, 3 players had an ESS score >10, a scoring threshold suggestive of a clinically important degree of daytime sleepiness. Thus, the estimated prevalence of an AHI between 5 and 15 events/h among those at high risk for daytime sleepiness was 15.7% (95% CI 1.0–33.1%). However, we found no significant difference between risk categories in the number of players who reported an ESS score >10 (high risk: 8 [44%] vs low risk: 10 [56%]; $\phi = 0.02$, $P = .90$). Similarly, we found no significant

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Table 1. Characteristics of Players per STOP-BANG-Derived Risk Category

| | High-Risk Players ($n$ = 25) | | | Low-Risk Players ($n$ = 26) | | | $P$* |
|---|---|---|---|---|---|---|---|
| | Mean ± SD | Range | Median | Mean ± SD | Range | Median | |
| Age, y | 19.8 ± 1.4 | 18–23 | 19.5 | 19.4 ± 1.4 | 17–22 | 19.0 | .38 |
| Height, cm | 187.5 ± 9.4 | 157–201 | 188.0 | 185.7 ± 6.6 | 170–198 | 185.4 | .44 |
| Weight, kg | 115.5 ± 18.2 | 79–143 | 111.6 | 95.6 ± 17.2 | 76–135 | 93.9 | <.001 |
| BMI, kg/m$^2$ | 33.0 ± 5.4 | 24.3–45.9 | 32.2 | 27.6 ± 3.6 | 22.7–34.4 | 26.6 | <.001 |
| Neck circumference, cm | 44.6 ± 2.2 | 40.0–49.0 | 45 | 41.4 ± 2.8 | 38.0–49.0 | 41.0 | <.001 |
| ESS total score | 9.3 ± 4.2 | 1–16 | 9 | 8.4 ± 3.7 | 0–16 | 9 | .40 |
| Ethnicity | $n$ (%) | | | $n$ (%) | | | |
| African-American | 16 (31) | | | 13 (25) | | | |
| White | 6 (12) | | | 13 (25) | | | |
| Mixed race | 1 (2.3) | | | 1 (2.3) | | | |
| Hispanic | 1 (2.3) | | | 0 (0) | | | |

* Equality of variances not assumed.
BMI = body mass index
ESS = Epworth Sleepiness Scale

Table 2. Body Composition per STOP-BANG-Derived Risk Category

| | High Risk ($n$ = 22) | | | Low Risk ($n$ = 25) | | | $P$* |
|---|---|---|---|---|---|---|---|
| | Mean ± SD | Range | Median | Mean ± SD | Range | Median | |
| Total mass, kg** | 112.9 ± 16.4 | 77.5–138.2 | 112.9 | 97.3 ± 16.8 | 76.5–130.6 | 95.1 | .002 |
| Total fat mass, kg | 26.5 ± 11.9 | 6.8–44.7 | 22.6 | 20.1 ± 10.0 | 7.6–43.7 | 15.4 | .043 |
| Total fat, % | 22.6 ± 7.9 | 8.8–34.3 | 21.1 | 19.8 ± 6.8 | 9.7–33.4 | 17.4 | .16 |
| Total lean mass, kg | 82.0 ± 7.9 | 66.5–103.5 | 82.1 | 73.3 ± 8.4 | 58.9–89.1 | 75.3 | <.001 |
| Trunk fat mass, kg | 12.6 ± 6.7 | 2.2–25.3 | 10.8 | 9.1 ± 5.3 | 2.3–19.5 | 6.6 | .047 |
| Trunk fat % of total fat mass | 45.5 ± 6.3 | 31.9–56.8 | 45.1 | 43.5 ± 6.5 | 27.0–56.7 | 43.9 | .23 |
| Abdominal visceral fat mass, kg† | 0.42 ± 0.4 | 0.04–1.1 | 0.25 | 0.30 ± 0.3 | 0.00–1.3 | 0.20 | .24 |
| Visceral fat % of total fat mass† | 1.7 ± 1.1 | 0.39–4.2 | 1.5 | 1.5 ± 1.2 | 0.01–4.8 | 1.2 | .47 |

* Equality of variances not assumed.
** Total mass = total fat mass + total lean mass + total bone mass (not displayed).
† Data unavailable for 4 high-risk and 2 low-risk players.

difference between risk categories in the number of players with an ODI4 or ODI3 of ≥5 events/h (high risk: 4 [67%] versus low risk: 2 [33%]; $\phi$ = 0.153, $P$ = .28 and high risk: 8 [53%] versus low risk: 7 [47%]; $\phi$ = 0.096, $P$ = .50, respectively).

Body composition parameters in the players who exhibited SDB were compared with those of players without SDB. Whereas players with SDB tended to have higher total fat mass (SDB: 31.8 ± 9.5 kg vs 21.2 ± 11.2 kg, $P$ = .12), trunk fat mass (SDB: 15.1 ± 5.0 kg vs non-SDB: 10.1 ± 6.2 kg, $P$ = .14), abdominal visceral fat mass (SDB: 0.73 ± 0.43 kg vs non-SDB: 0.31 ± 0.33 kg, $P$ = .24), and total body fat (SDB: 26.7 ± 4.9% vs non-SDB: 20.3 ± 7.5%, $P$ = .07), the data were not statistically significant. These analyses were repeated using ODI as a surrogate for AHI. Table 3 displays data according to an ODI4 threshold of 5. Of note, players with ODI4 ≥5 had greater total body mass ($P$ = .028), whereas differences in total fat mass ($P$ = .062), total body fat percentage ($P$ = .069), and trunk fat mass ($P$ = .08) approached statistical significance. Similar trends were observed when an ODI3 ≥5 events/h was used as a cut-point (data not shown).

## Discussion

Two previous studies have evaluated SDB among football players. George et al[14] stratified 302 players from the National Football League (NFL) into high and low risk for SDB using the Multivariable Apnea Prediction index. Fifty-two players (high-risk = 38; low-risk = 14) subse-

Table 3. Body Composition Parameters According to 4% Oxygen Desaturation Index

| | ODI4 <5 events/h ($n$ = 41) | | | ODI4 ≥5 events/h ($n$ = 6) | | | $P$* |
|---|---|---|---|---|---|---|---|
| | Mean ± SD | Range | Median | Mean ± SD | Range | Median | |
| Total mass, kg | 101.8 ± 18.3 | 76.5–138.2 | 102.2 | 117.6 ± 13.1 | 95.1–130.8 | 119.4 | .028 |
| Total fat mass, kg | 21.4 ± 11.0 | 6.8–44.7 | 19.4 | 31.5 ± 10.5 | 18.8–42.6 | 34.2 | .062 |
| Total fat, % | 20.0 ± 7.3 | 8.8–34.1 | 18.4 | 26.3 ± 6.7 | 16.8–34.3 | 28.1 | .069 |
| Total lean mass, kg | 76.3 ± 9.5 | 58.9–103.5 | 77.5 | 81.7 ± 6.5 | 72.5–89.5 | 82.5 | .11 |
| Trunk fat mass, kg | 9.9 ± 6.2 | 2.2–25.3 | 8.5 | 14.7 ± 5.2 | 8.4–19.8 | 15.8 | .08 |
| Trunk fat % of total fat mass | 43.9 ± 6.8 | 27.0–56.8 | 44.5 | 46.3 ± 2.6 | 43.1–49.2 | 46.1 | .12 |
| Abdominal visceral fat mass, kg† | 0.30 ± 0.33 | 0.00–1.3 | 0.19 | 0.65 ± 0.39 | 0.22–1.1 | 0.76 | .11 |
| Visceral fat % of total fat mass† | 1.5 ± 1.2 | 0.01–4.8 | 1.1 | 1.9 ± 0.63 | 1.2–2.8 | 2.0 | .25 |

* Equality of variances not assumed.
† Data unavailable for 5 players with 4% oxygen desaturation index <5 events/h and one player with 4% oxygen desaturation index ≥5 events/h.
ODI4 = 4% oxygen desaturation index

quently underwent overnight polysomnography. An AHI of ≥10 was found in 13 (34%) of the high-risk players, compared with just one (7%) in the low-risk group. In a follow-up analysis,[5] these investigators reported that 10 additional players had an AHI of between 5 and 9.9 events/h, thereby increasing the estimated prevalence rate of SDB in this athletic cohort to nearly 50%, if using a more traditional AHI threshold. More recently, Rice et al[6] evaluated SDB among 137 active veteran NFL players using a home-based, unattended, portable sleep monitor. The prevalence of at least mild SDB (respiratory disturbance index ≥5) was 19%. Although linemen weighed more and had a greater body fat percentage, waist circumference and neck circumference compared with non-linemen, there was no difference in SDB prevalence estimates between these groups. However, the average body mass index across both studies was >30, a noteworthy finding given that increased body weight is perhaps the strongest predictor of SDB.[15,16] Thus, despite presumably being highly conditioned, these young athletes are at significantly greater risk for SDB than community-dwelling men of similar age.[7]

The current study extends this line of inquiry in several meaningful ways. First, we have estimated the prevalence of SDB in a Division 1AA collegiate football team. Our observed prevalence of approximately 8% for those with at least mild SDB (AHI ≥ 5) is lower than what has been reported previously in the professional ranks, but we feel it is quite significant considering that sleep screenings are not likely to be part of most physical exams for collegiate athletics programs. By 2015, the number of United States colleges and universities offering football will have increased to 773 (http://www.footballfoundation.org. *Accessed August 20, 2015*). Assuming that most teams maintain rosters of about 100 athletes, this equates to roughly 6,000 players who are living and competing with SDB. The clinical and public health importance of this is underscored by the fact that other studies[17,18] have reported early signs of atherosclerosis (eg, endothelial dysfunction, increased carotid intima-media thickness, and arterial stiffening) among minimally symptomatic, middle-age OSA subjects, suggesting that if left untreated, collegiate football players with SDB are likely to suffer from a greater cardiovascular disease burden after their athletic careers have ended. Perhaps of more immediate concern is the adverse impact sleep apnea has on attention and cognitive function.[8] The potential implication is that players with SBD may experience detriments in academic as well as athletic performance.[19]

Second, emerging data suggest that proper sleep may play an important role in recovery from and possibly prevention of concussions.[20] That said, our finding of an average total sleep time of 4 h is alarming. Although the short sleep duration may be attributable to extraneous factors (eg, wearing the PPG device and anxiety associated with preseason camp [see below]), existing data suggest[21] that insufficient sleep and poor quality sleep in college students is more likely the rule, than the exception. Indeed, 35% of the players in this study reported a clinically important degree of daytime sleepiness.

Third, we found that the STOP-BANG questionnaire had low sensitivity to predict mild to severe SDB (50%). By comparison, the sensitivity to detect positive cases increased to 75% when using the Epworth Sleepiness Scale. Our decision to choose the STOP-BANG was based on ease of use and the simple scoring system. Thus, it is more likely to be incorporated into a sports medicine setting. However, it appears that it may lack sufficient sensitivity in this cohort to avoid missing a large number of SDB cases. The reasons for this may be related to the fact that some of the elements of the STOP-BANG (1) could not be observed (eg, player may lack a bed partner to observe whether he stops breathing), (2) were constants (eg, all players were men <50 y old), and (3) were also contained in body composition measures (eg, body mass index and

neck circumference). Clearly, our findings call into question the suitability for use of the STOP-BANG to identify positive cases in this demographic group.

Fourth, we employed state-of-the-art dual-energy x-ray absorptiometry imaging that allowed us to quantify total and regional fat with greater precision than previous reports.[5,6] Consequently, we found higher regional (ie, trunk fat mass and abdominal visceral fat mas) and total body fat mass in players with oxygen desaturations above a threshold of 5 events/h, albeit most of these difference did not reach statistical significance. However, we cannot discount the possibility that we made a type-2 error. Still, this observation is consistent with the notion that central adiposity increases OSA susceptibility by elevating mechanical loads on the upper airway. Also, adipose tissue is an abundant source of pro-inflammatory cytokines, such as TNF-$\alpha$, and IL-6. These are markedly elevated in central obesity[22,23] and their purported somnogenic activity[24] may lead to a depression in upper airway neuromuscular control.

The above findings should be viewed in the context of several limitations. The sample size was small, thereby limiting our power to detect (1) group differences in the number of high-risk versus low-risk players who have clinical signs of SDB (eg, ESS >10, AHI/ODI ≥5 events/h) or (2) differences in body composition parameters between those with and without SDB. Moreover, although ethnicity may affect body composition, the small sample precludes us from making meaningful comparisons between racial groups. Also, we used one night of PPG monitoring, and all tests were performed during preseason mini-camp. During this time, the players undergo intensive training, experience high levels of stress, and sleep in dormitory beds. These factors are likely to affect sleep habits and, in turn, ESS scores. Further, any direct comparisons of SDB prevalence rates between collegiate and professional football players must be interpreted with caution, since George et al[14] relied on overnight polysomnography and Romem et al[11] used a portable monitoring system with air flow measurements to diagnose OSA. Although prior investigations[11-13] have shown that PPG displays a sensitivity ranging from 80 to 98%, we acknowledge that these studies were conducted in subjects who were referred for overnight sleep testing because of clinically suspected SDB.

Also, Romem et al[11] failed to show a significant correlation between polysomnography-derived and PPG-based parameters for total sleep time, which they attributed, in part, to possible degradation of transmission signal from the PPG to the cellphone receiver at the bedside. Accordingly, studies designed to examine the validity and reliability of PPG in this younger, athletic cohort are warranted. Related to this point, because the PPG is a single-channel device, it does not meet American Academy of Sleep Medicine standards for home sleep studies. Therefore, it is not acceptable for insurance reimbursement or prescriptive treatment. Alternatively, it may serve as a good indictor for further sleep disorder diagnostic studies for players with an AHI or ODI of ≥5 events/h. Last, we did not assess physiological markers of cardiovascular disease burden, markers of athletic or academic performance, or cognitive function, so it is not possible to draw conclusions regarding the potential impact of SDB in these players. However, we feel that the above limitations are balanced by our unique sample and the clear advantage PPG monitoring has over laboratory sleep studies with regard to cost effectiveness, convenience, and noninvasiveness.

## Conclusions

In summary, we found the estimated prevalence of SDB (AHI ≥ 5) among a group of Division 1AA collegiate football players to be approximately 8%. Players stratified as high-risk had a similar prevalence rate compared with low-risk players. Thirty-five percent of the sample reported a clinically important degree of daytime sleepiness. Moreover, our data suggest that players with greater oxygen desaturations have high total body and regional fat. Hence, our data emphasize the need to screen for sleep disorders, educate these athletes about the importance of sleep, and help facilitate the translation of this knowledge into practice.

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