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Original Article

Accuracy of global and/or regional anthropometric measurements of adiposity in screening sleep apnea: the ELSA-Brasil cohort

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ABSTRACT

Objective: Adiposity is a well-established risk factor for obstructive sleep apnea (OSA) but the existence of a preferable anthropometric measurement is not established or whether the combination of measurements may improve the accuracy to detect OSA. This study aimed to compare the accuracies of body mass index (BMI), several surrogate markers of body fat (in isolation or combined) and validated questionnaires for screening OSA.

Methods: A total of 2059 participants from the ELSA-Brasil study given anthropometric measurements using standard procedures and a home sleep study. OSA was defined by an apnea-hypopnea index ≥15 events/hour.

Results: The frequency of OSA was 32.3%. Compared with the non-OSA group, all anthropometric measurements were higher in the OSA group. Age and gender-adjusted BMI afforded the highest accuracy to detect OSA [AUC = 0.760 (0.739–0.781)], followed by waist [AUC = 0.753 (0.732–0.775)] and neck [AUC = 0.733 (0.711–0.755)] circumferences, waist-to-hip ratio [AUC = 0.722 (0.699–0.745)] and body shape index [AUC = 0.680 (0.656–0.704)]. The combination of two or more anthropometric measurements did not improve the accuracy of BMI in predicting OSA. The adjusted BMI had similar predictive performance to the NoSAS score [AUC = 0.748 (0.727–0.770)] but a better accuracy than the Berlin Questionnaire [AUC = 0.676 (0.653–0.699)].

Conclusions: Despite one's intuition, surrogate markers of regional adiposity are not better than BMI in screening OSA. Combining measurements of global and/or regional adiposity did not have additional value in detecting OSA. The merely fair accuracy range of BMI and sleep questionnaires underscore the need for additional tools to improve OSA underdiagnosis.

1. Introduction

Obstructive Sleep Apnea (OSA) is a clinical condition characterized by recurrent upper airway obstructions leading to sleep fragmentation and intermittent hypoxia during sleep [1]. Among the traditional risk factors for OSA, it is well established that being overweight or obese increases the risk of OSA [2–4]. Indeed, data from the Wisconsin Cohort showed that a 10% weight gain led to a six-fold increase in the risk of developing moderate-to-severe OSA [2]. Despite the clear association of OSA with excessive weight, considerable variability exists in the prevalence and severity of OSA in overweight or obese subjects [5]. In this scenario, it is conceivable that the heterogeneity of fat deposition may influence OSA severity. Fat deposition in the tissues surrounding the upper airway appears to result in a smaller lumen and increased collapsibility of the upper airway, predisposing to obstructive events [6–8]. On the other hand, visceral adiposity may also contribute to lung volume reductions and to reducing longitudinal tracheal traction forces, which predisposes a narrowing of the upper airway [9,10].

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Several studies examined the link between adiposity and OSA based on the analysis of individual anthropometric measures, including body mass index (BMI), waist circumference (WC), neck circumference (NC), and waist to hip ratio (WHR) [6–12]. However, the following gaps in this research area are notable: (1) the potential role of some anthropometric measurements have not been tested; (2) the utility in combining different anthropometric measurements to potentially increase the classification accuracy of OSA is not clear; (3) the direct comparison of selected anthropometric measurements and traditional sleep questionnaires for screening OSA requires further investigation. To explore these critical issues, we performed a cross-sectional analysis on a subset of the subjects from a large cohort of adults non-referred for sleep studies. Based on the relative importance of distinct compartments in predisposing to upper airway collapse during sleep, we speculated that the combination of anthropometric markers might add value to screening for OSA as compared to each parameter in isolation.

2. Methods

Participants in this study are a subset from the Brazilian Longitudinal Study of Adult Health (ELSA-Brasil), Sao Paulo site. The ELSA-Brasil cohort profile and routines were previously reported (please see Supplemental file) [13–15]. Briefly, ELSA-Brasil is a cohort study of 15,105 civil servants aged 35–74 years from five universities and one research institute located in different regions of Brasil. Exclusion criteria were current or recent (<4 months before the first interview) pregnancy, intention to quit working at the institutions in the near future, severe cognitive or communication impairment, and, if retired, residence outside of a study center's corresponding metropolitan area. In our study, we excluded only subjects who refused to participate. Further details of the cohort are described elsewhere [14,15]. Of 5061 participants from the São Paulo center, clinical (including anthropometric measurements) and sleep evaluations were performed in a blinded way as previously described [16].

2.1. Anthropometric measurements

Weight and height were measured following standard procedures in the ELSA-Brasil cohort [13]. Briefly, WC was measured using an inelastic tape of 150 cm (Mabis-Gulick, Waukegan, IL, USA) at the midpoint between the inferior edge of the costal border and the iliac crest, in the mid-axillary line, and hip circumference at the maximum posterior protrusion of the gluteus muscles. The intraclass correlation coefficient for repeat measurements was 0.98 (95% CI 0.85–1.0) [17]. We measured height to the nearest 0.1 cm (Seca-SE-216, Hamburg, Germany). We measured body weight with an electronic scale having a maximum capacity of 200 kg (Toledo, São Bernardo do Campo, Brazil). The BMI was calculated by dividing the participant's weight in kilograms by the square of their height in meters (kg/m^2). According to the World Health Organization, we defined a normal BMI ranging from 18.5 to 24.9 Kg/m^2. Overweight was defined by a BMI from 25.0 to 29.9 Kg/m^2 and obesity by a BMI over 30.0 Kg/m^2 [18]. We performed three measurements using WC data: WHR, waist-to-height ratio (WHtR), and body shape index (BSI). WHR was calculated as WC (cm) divided by the hip circumference (cm); WHtR was calculated using WC (cm) divided by height (cm). The BSI corresponds to a higher concentration of body volume centrally and expresses WC relative to height and weight (therefore, statistically independent of BMI) [19]. We chose this variable not only because it has never been tested in the OSA setting but because previous evidence pointed to BSI as an independent risk factor for premature mortality [20,21]. BSI was obtained using the following formula [19]:

$$BSI = \frac{WC}{BMI^{2/3}\, height^{\frac{1}{2}}}$$

2.2. Screening tools for OSA

All participants completed the Berlin questionnaire and the NoSAS score in a blinded fashion (a sleep medicine expert scored all sleep studies with no access to the questionnaires). Briefly, the Berlin questionnaire is a tool for screening OSA based on questions in the following categories: (1) snoring and cessation of breathing; (2) tiredness and fatigue after sleep; and (3) the presence of obesity or hypertension. A high risk of OSA was defined by $\geq$ two positive categories [22]. The NoSAS score is a simple and effective screening tool for OSA [23]. The NoSAS score, which ranges from 0 to 17 points, includes five questions, attributing four points for a neck circumference of >40 cm, three points for a BMI of 25 to <30 kg/m^2, five points for a BMI of $\geq$30 kg/m^2, two points for snoring, four points for being older than 55 years of age, and two points for male gender. A score above or equal to eight points identifies individuals at risk of OSA [23].

2.3. Overnight home sleep study

Sleep studies were performed using the Embletta Gold (Natus Medical Inc., Ontario, Canada), a standardized level-3 portable diagnostic device, as previously described [16,24]. The Embletta system has been validated for use against in-laboratory polysomnography [25,26] and possesses a sensitivity and specificity ranging from 92% to 97% and 64%–96%, respectively [27–29]. All studies were manually scored by an expert in sleep medicine, according to the American Academy of Sleep Medicine (AASM) 2012 criteria [30]. Apnea was defined as a $\geq$90% decrease in airflow from the baseline value for $\geq$10 s. Hypopnea was defined by a $\geq$ 30% drop of airflow lasting at least 10 s with a $\geq$3% SpO_2 drop. Apneas were further classified as obstructive or central, based on the presence or absence, respectively, of respiratory-related chest-wall movement. The sum of apnea and hypopneas per hour determined the apnea-hypopnea index (AHI). We excluded participants with predominantly (>50%) central sleep apnea. Considering growing evidence suggesting that mild OSA is not associated with increased cardiovascular risk [31], we used a more conservative AHI cut-off of $\geq$15 events/hour of sleep. A recent populational study used a similar definition [32].

Our study was conducted according to the guidelines laid down in the Declaration of Helsinki, and all procedures involving human subjects/patients were approved by the local ethical committee approved the study (1166/11). Written informed consent was obtained from all participants.

2.4. Statistical analysis

Statistical analysis was carried out using SPSS version 24.0 (SPSS Inc., Chicago, IL, USA). Continuous data were reported as the mean ± standard deviation (SD) or the median/interquartile range, according to the normal distribution status. For categorical data, frequencies and percentages were reported. Mean differences between the OSA and non-OSA groups were tested using analysis of covariance. Proportion differences were tested using the Chi-square test. Logistic regression analysis was used, considering OSA occurrence as the dependent variable. Considering that the fat accumulation in the abdominal and/or neck regions may be more closely related to OSA [6–9], four sets of regression models, with and without adjustment for gender and age, were evaluated for OSA with the following combinations of independent variables:

(1) each anthropometric variable alone; (2) BMI combined with other surrogate markers abdominal (WC, WHtR, WHR or BSI) or cervical fat (NC); (3) BMI combined with other surrogate markers abdominal (WC, WHtR, WHR or BSI) and cervical fat (NC); and (4) Combined surrogate markers cervical (NC) with other abdominal fat (WC, WHtR, WHR, and BSI).

The discrimination ability of fitted logistic models was assessed using the receiver operating characteristic (ROC) curve. The discrimination ability of the model was reported through the area under the estimated ROC curve with 95% confidence intervals (CI). The ROC analysis was also performed to determine the optimal sex-specific cut-off values for BMI, NC, WC, WHtR, WHR, and BSI, with optimal sensitivity and specificity for the identification of OSA. Also, we performed a sub-analysis, including anthropometric measurements in a dichotomized way. Finally, the best model for discriminating OSA was compared to two widely used sleep screening tools, namely the Berlin questionnaire and the NoSAS score. All tests were 2-sided, and a P-value of <0.05 was considered statistically significant.

3. Results

From the 5061 participants of ELSA-Brasil, Sao Paulo Center, a total of 2224 were consecutively invited to perform a sleep evaluation during a two-year recruitment period. After excluding refusals, technical issues in sleep monitoring, predominant central sleep apnea, previous OSA treatment and nocturnal/shift workers, a total of 2059 participants were included in the final analysis (see details on Figure S1, Supplemental file).

Table 1 reports the characteristics of the studied population. Overall, one-third of them had OSA. Compared to participants without OSA, those with OSA were predominantly men, older, less educated and had a higher frequency of being overweight, obese, and had co-morbidities including hypertension, diabetes, dyslipidemia, excessive drinking, and lower physical activity. We found that there was no difference in excessive daytime sleepiness between the groups.

In the Supplementary file, Table S1 reports the correlations between all anthropometric measurements. The BMI yielded a strong correlation with two surrogate markers of abdominal fat (WC and WHtR) but a moderate correlation with WHR and a weak correlation with BSI. The NC presented a strong correlation with WC and WHR and moderate correlation with BMI, WHtR, and BSI. The BSI showed a weak-to-moderate correlation with all indexes, except WHR.

The discrimination ability to detect OSA of each anthropometric variable as well as combinations of them are reported in Table 2. Overall, there was a fair accuracy of each variable in detecting OSA after adjustments for age and gender (except BSI, where a poor accuracy was found). The BSI had a significantly worse performance than the remaining variables. Also, BMI, WC, and WHtR had significantly higher AUC than the WHR measurement. We found that the combination of BMI with the other variables or the combination of variables other than BMI did not improve the accuracy compared to the best variables (BMI, NC, WC and WHtR in isolation) for identifying individuals with OSA.

Table S2 (Supplemental file) estimates optimal anthropometric cutoffs to diagnose OSA in our cohort. The analysis of the anthropometric variables categorized according to the selected cutoff points showed similar results to the analysis of the continuous variables (Table 3). Combining two or more anthropometric variables did not improve the accuracy of identifying OSA compared to the BMI, adjusted for gender and age (Table 3 and Fig. 1). Fig. 1 also shows that the AUC using BMI as a categorical variable was significantly lower than the continuous BMI. In addition, as shown in Table S3 of the Supplementary file, there were no significant differences among AUCs when we compared mild, moderate, and severe OSA.

Finally, we compared the performance of BMI adjusted for gender and age with traditional sleep questionnaires used in

Table 1
Characteristics of studied population stratified by the presence of obstructive sleep apnea (OSA).

Variable	All subjects N = 2059	No OSA n = 1394 (67.7%)	OSA n = 665 (32.3%)
Gender, male, n (%)	900 (43.7)	517 (37.1)	383 (57.6)**
Age (years)	49 ± 8	48 ± 8	51 ± 8**
Ethnicity − White, n (%)	1314 (63.8)	908 (65.1)	406 (61.1)
Overweight, n (%)	847 (41.1)	546 (39.2)	301 (45.3)**
Obesity, n (%)	468 (22.7)	217 (15.6)	251 (37.7)**
Anthropometry	847 (41.1)	546 (39.2)	301 (45.3)**
Body mass index, kg/m^2	27.0 ± 4.3	26.0 ± 3.9	29.1 ± 4.2**
Waist circumference, cm	89.1 ± 11.6	86.0 ± 10.6	95.5 ± 11.0**
Waist-to-height ratio	0.54 ± 0.07	0.52 ± 0.06	0.58 ± 0.07**
Waist-to-hip ratio	0.88 ± 0.08	0.86 ± 0.08	0.92 ± 0.08**
Body shape index, m$^{11/6}$ kg$^{-2/3}$	0.077 ± 0.004	0.077 ± 0.004	0.079 ± 0.004**
Neck circumference, cm	35.9 ± 3.5	35.1 ± 3.3	37.6 ± 3.4**
Education beyond high school, n (%)	967 (47.0)	680 (48.8)	287 (43.2)*
Hypertension, n (%)	536 (26.0)	281 (20.2)	255 (38.3)**
Diabetes mellitus, n (%)	300 (14.6)	147 (10.5)	153 (23.0)**
Dyslipidemia, n (%)	1106 (54.1)	702 (50.5)	404 (61.5)**
Current smoker, n (%)	629 (30.5)	399 (28.6)	230 (34.6)*
Excessive drinking, n (%)	108 (11.5)	61 (9.8)	47 (14.6)*
Insufficient physical activity, n (%)	944 (48.7)	607 (46.5)	337 (53.2)*
AHI, events/h	14.6 ± 15.0	6.6 ± 4.1	31.1 ± 15.9**
Lowest SpO_2, %	85.0 ± 6.1	87.4 ± 4.2	80.0 ± 6.4**
Time SpO_2 < 90, %	3.9 ± 10.7	1.2 ± 4.9	9.4 ± 16.1**
ESS score	9 (6–13)	9 (5–13)	9 (6–13)*
Excessive daytime sleepiness, n (%)	797 (38.7)	530 (38.0)	267 (40.2)
High risk for OSA, BQ, n (%)	831 (40.4)	440 (31.6)	391 (58.9)**
NoSAS score, n	5 (3–7)	5 (2–7)	7 (5–11)
High risk for OSA, NoSAS score, n (%)	505 (24.5)	226 (16.2)	279 (42.0)**

AHI: apnea-hypopnea index; BQ: Berlin Questionnaire; ESS: Epworth Sleepiness Scale. *$p < 0.05$ *vs.* no OSA. **$p < 0.001$ *vs.* no OSA.

Table 2
Area under the curve (AUC) using receiver operating characteristic (ROC) coefficients from multivariate logistic regression models for the associations of anthropometric measurements and Obstructive Sleep Apnea.

Model	Crude		Adjusted for age and gender		AUC[a]
	AUC	95% CI	AUC	95% CI	
Base model[b]	0.671	0.647–0.695			
Alone					
BMI	0.708	0.684–0731	0.760	0.739–0.781	*WHR < BMI, WC and WHtR*
NC	0.698	0.674–0.721	0.733	0.711–0.755	*BSI < All*
WC	0.735	0.712–0.757	0.753	0.732–0.775	
WHtR	0.723	0.700–0.746	0.758	0.737–0.780	
WHR	0.713	0.690–0.736	0.722	0.699–0.745	
BSI	0.635	0.610–0.661	0.680	0.656–0.704	
BMI and Surrogate Markers Abdominal or Cervical Fat					
BMI + NC	0.735	0.712–0.757	0.761	0.740–0.782	*NS*
BMI + WC	0.735	0.712–0.757	0.761	0.740–0.782	
BMI + WHtR	0.724	0.701–0.747	0.762	0.741–0.784	
BMI + WHR	0.746	0.724–0.768	0.763	0.742–0.784	
BMI + BSI	0.734	0.711–0.756	0.762	0.741–0.783	
BMI and Surrogate Markers Abdominal and Cervical Fat					
BMI + NC + WC	0.742	0.720–0.764	0.762	0.741–0.783	*NS*
BMI + NC + WHtR	0.748	0.726–0.770	0.764	0.743–0.785	
BMI + NC + WHR	0.749	0.727–0.771	0.764	0.743–0.785	
BMI + NC + BSI	0.746	0.724–0.768	0.763	0.742–0.784	
Combined Surrogate Markers Abdominal and Cervical Fat					
NC + WC	0.740	0.718–0.718	0.756	0.735–0.778	*NS*
NC + WHtR	0.748	0.726–0.770	0.762	0.741–0.783	
NC + WHR	0.722	0.699–0.745	0.744	0.723–0.766	
NC + BSI	0.707	0.683–0.730	0.735	0.713–0.756	

CI: confidence interval; BMI: body-mass index; BSI: body shape index; NC: neck circumference; WC: waist circumference; WHtR: waist-to-height ratio; WHR: waist-to-hip ratio; NS: Not significant.
[a] Multiple Comparison Method: Bonferroni-corrected P values.
[b] Base model includes age and gender.

Table 3
Coefficient of determination Nagelkerke R-squared (R^2) and area under the curve (AUC) using receiver operating characteristic (ROC) coefficients from multivariate logistic regression models for the associations of anthropometric parameters (categorized) and OSA.

Model	R^2	AUC (95% CI)	AUC[a]
Alone			
High BMI	0.171	0.721 (0.698–0.744)	*WC > WHR*
High NC	0.143	0.702 (0.679–0.726)	
High WC	0.166	0.715 (0.692–0.738)	*BSI < all (except WHR)*
High WHtR	0.157	0.709 (0.685–0.733)	
High WHR	0.116	0.677 (0.653–0.701)	
High BSI	0.093	0.666 (0.642–0.691)	
High BMI and Surrogate Markers Abdominal or Cervical Fat			
High BMI + NC	0.192	0.734 (0.712–0756)	*NS*
High BMI + WC	0.187	0.730 (0.707–0.752)	
High BMI + WHtR	0.182	0.727 (0.704–0.749)	
High BMI + WHR	0.188	0.729 (0.707–0.752)	
High BMI + BSI	0.199	0.738 (0.716–0.760)	
High BMI and Surrogate Markers Abdominal and Cervical Fat			
High BMI + NC + WC	0.200	0.738 (0.716–0760)	*NS*
High BMI + NC + WHtR	0.199	0.739 (0.717–0.761)	
High BMI + NC + WHR	0.203	0.739 (0.717–0.761)	
High BMI + NC + BSI	0.214	0.748 (0.726–0.770)	
Combined Surrogate Markers Abdominal and Cervical Fat			
High NC + WC	0.187	0.731 (0.709–0753)	*NS*
High NC + WHtR	0.188	0.733 (0.710–0.755)	
High NC + WHR	0.169	0.718 (0.695–0.740)	
High NC + BSI	0.171	0.721 (0.699–0.744)	

CI, confidence interval; ROC, receiver operating characteristic; BMI, body-mass index; BSI, body shape index; NC, Neck circumference; WC, waist circumference; WHtR, waist-to-height ratio; WHR, waist-to-hip ratio. R^2 = coefficient of determination Nagelkerke R-squared. NS: Not significant.
[a] Multiple Comparison Method: Bonferroni-corrected P values. All models were adjusted for age and gender.

clinical practice. We found that BMI adjusted for gender and age presented a similar performance to the NoSAS score but higher than the Berlin Questionnaire (Fig. 2). We found a similar AUC of the NoSAS score for mild, moderate, and severe OSA as compared to the original validation study [23] (Table S4, Supplemental file), suggesting the potential external validation of our findings.

4. Discussion

In this large cross-sectional study of Brazilian civil servants, we investigated the relative accuracy of multiple anthropometric variables as well as their combination in identifying individuals with significant OSA (moderate to severe forms). Despite the higher AUC values of BMI (adjusted for age and gender), our study revealed that all anthropometric measurements in isolation had a fair accuracy in identifying individuals with OSA as compared to the formal sleep study. The present study contributes to our understanding that the combination of BMI and surrogate markers of the neck and abdominal fat, as well as the combination of BMI with both, did not significantly add value for OSA diagnosis. However, because the AUC of adjusted BMI and the most-used sleep questionnaires were in the fair accuracy range, our results underscore the need for additional tools for screening OSA.

The utility of anthropometric measurements in OSA have been explored in the literature but usually in patients with high suspicions for OSA (referred for sleep studies) [6–12,33–36]. Moreover, previous descriptions of the pathophysiology of OSA underscores the importance of the distribution of both local (neck) and abdominal fat in contributing to the upper airway obstructions during sleep [1]. In this setting, no previous study explored the clinical applicability of using one or more surrogate markers of global vs. local fat or their combination in OSA screening. Anthropometric measurements are feasible and straightforward measures to estimate global (BMI), neck (NC) or abdominal (WC, WHtR, WHR, and BSI) fat and may help to improve OSA suspicions. Previous estimates suggest that approximately 75–80% of OSA cases may not be diagnosed even in patients with obesity [37,38].

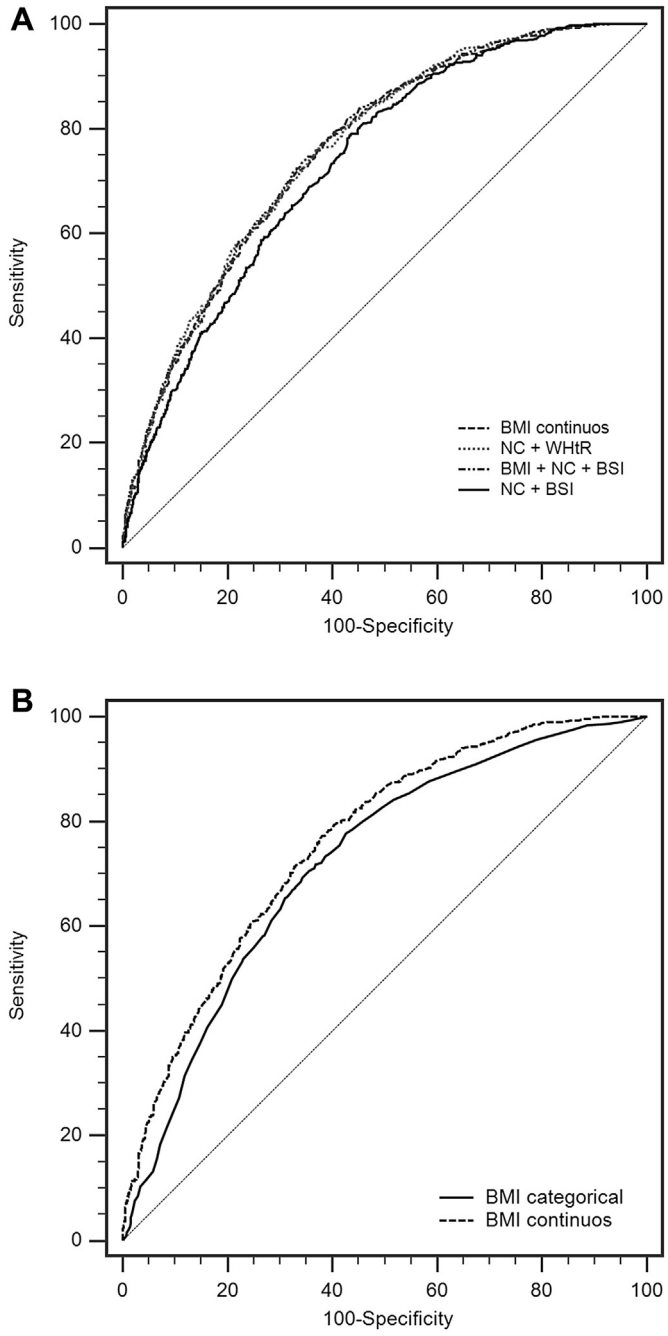

Fig. 1. Comparison of the area under the curve (AUC) of the four models with better accuracy (A) and comparison between models with body mass index (BMI) as a continuous and categorical variable (B). See Table 2 for abbreviations. All models were adjusted for age and gender. Continuous BMI had higher AUC than categorical BMI ($p = 0.014$).

OSA underdiagnosis has multiple causes including the lack of typical symptoms in a significant proportion of patients (in our study we did not observe significant differences in the subjective daytime sleepiness in participants with and without OSA) as well as the low availability and long waiting lists for polysomnography (considered the gold standard method for the OSA diagnosis). In the present study, we confirmed the individual contribution of BMI, NC, WC, WHtR, WHR, and BSI) for screening OSA, but all of them only presented fair accuracy. Contrary to our initial hypothesis, our data underscore the lack of utility in measuring multiple anthropometric parameters in order to improve the accuracy for detecting OSA.

An additional important finding of our study was the similar performance of BMI adjusted for gender and age with the most promising sleep questionnaire (namely NoSAS score) for screening OSA. It is essential to mention that both screening tools use adiposity parameters to score the risk of OSA. We consistently found a similar AUC of NoSAS as in the original investigation [23] for moderate-to-severe OSA. The stratification of OSA into mild, moderate, and severe forms showed results similar to those obtained by the HypnoLaus cohort (Fig. 2). Our study is also consistent with the previous report that the NoSAS score had a better performance than the Berlin Questionnaire [23]. These facts reinforce the potential applicability of our findings to other adult populations.

Despite the clear association of OSA with excessive weight, many individuals who are overweight or obese may not have OSA. It has been estimated that obesity accounts for only 40% of the genetic variation of OSA [39]. This fact underscores that the severity of OSA is predominantly mediated by other mechanisms, such as those related to craniofacial anatomy, regulation of upper-airway anatomy/collapsibility, upper-airway muscle responsiveness, chemoreflex control of ventilation and arousability from sleep, leptin resistance (that may impair the neuroanatomic interactions necessary for stable breathing), among others [40,41]. Despite the significant contributions of the functional measurements in the pathogenesis of OSA, these techniques (in contrast to the anthropometric measurements) are quite labor intensive, and not easily applied in the routine clinical practice. The development of automatic estimations of these parameters for phenotyping pharyngeal pathophysiology using sleep studies, seems potentially useful and may change some previous concepts concerning adiposity measurements in detecting OSA [41]. Alternatively, the development and validation of simple objective techniques for monitoring sleep at low cost (for example: using apps in mobile phones) may surpass the relatively low accuracy of the anthropometric measurements and sleep questionnaires for screening OSA.

Our cohort study has strengths and limitations to be addressed. We studied a large, non-referred population with low refusal and sleep-monitoring failure rates. We carefully excluded people undergoing specific OSA treatment that may have some impact, even modest [42], in the body weight measure. We used standardized anthropometric procedures without any foreknowledge of sleep data.

The following limitations should be acknowledged: First, our results may be cautiously extrapolated to the general population. However, we did have a significant proportion of participants from all economic strata (unskilled, technical, and faculties) with a broad socioeconomic gradient. Second, for feasibility purposes, we used portable sleep monitoring for one night in this large population. In comparison with full in-laboratory polysomnography, the level-3 portable diagnostic device is limited by an inability to examine sleep staging and the lack of an objective measurement of sleep duration. However, as previously described [25–27], this monitor was validated correctly. We cannot discard the possibility of night-to-night variability in OSA severity, but performing a study at home on a typical day may mitigate significant variation. Third, anthropometric measurements are unable to estimate the amount of visceral and subcutaneous fat. Previous evidence showed that among individuals with similar BMI, OSA is more likely to be present in those with higher visceral fat volume than subcutaneous fat volume, suggesting the potential importance of visceral fat in the pathogenesis of OSA [10,43]. However, objective measurements of fat (using computed tomography, magnetic resonance imaging, etc.) are labor intensive and present significant costs, limiting their utility in clinical practice.

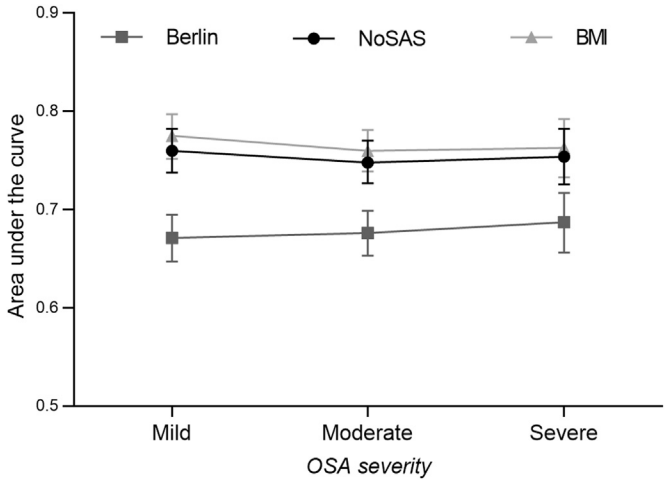

Fig. 2. Comparison of the performance of the Berlin Questionnaire, NoSAS score, and body mass index (BMI) adjusted for gender and age in the ELSA-Brasil cohort. There was only a significant difference in the AUC of the Berlin Questionnaire compared to the NoSAS score and the BMI ($p < 0.001$), regardless of the severity of OSA. Multiple Comparison Method: Bonferroni-corrected P values.

In conclusion, we observed that anthropometric measurements are individually useful but have limited ability to identify individuals with OSA. The combination of anthropometric variables (including regional fat) did not add value to the global adiposity parameter, BMI. BMI adjusted for gender and age yielded similar performance as the sleep questionnaires typically used in clinical practice.

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Conflict of interest

The authors declared no conflict of interest.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.sleep.2019.04.020.

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