Nature and Science of Sleep

Elevate Series

Open Access Full Text Article

ORIGINAL RESEARCH

The Association of Body Mass Index and Adiposity-Estimating Equations with Measures of Obstructive Sleep Apnea Severity: A Cross-Sectional Study

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Background and Purpose: Obesity, a risk factor for obstructive sleep apnea (OSA), is usually estimated by body mass index (BMI). However, other adiposity-estimating equations may better capture variations in fat distribution. This study assessed the relationship between OSA severity and 15 adiposity-estimating equations, compared to BMI, with subgroup analyses by sex and age (<50 vs ≥50).

Patients and Methods: We conducted a cross-sectional cohort study using data from 5021 consecutive adults who underwent a Level 1 polysomnography (2015–2017) in a large academic sleep center in Ottawa, Canada. We assessed correlations between adiposity measures and the apnea–hypopnea index (AHI) and examined discriminative ability for moderate-to-severe (AHI ≥15/h) and severe OSA (AHI >30/h) using univariate logistic regressions.

Results: The mean age was 49.5 years, 46.6% were women; the mean BMI was 30.0 kg/m^2 and 12.7% had severe OSA. All adiposity equations showed negligible (Pearson r 0.0 to ±0.3) to low (Pearson r ± 0.30 to 0.50) statistically significant correlations with AHI, with many of the equations having a marginally stronger correlation coefficient than BMI, in total and subgroup analysis. Discriminative ability for severe OSA was generally low, with c-indices ranging from 0.52 to 0.67 in the overall sample. However, in females under 50, several equations (eg, Gallagher 2000, Deurenberg 1991 and 1998, ECORE BF) reached excellent discriminative ability (c-indices 0.81), including BMI (c-index 0.80). This pattern was not observed in other subgroups.

Conclusion: In this clinical cohort, BMI was associated poorly with AHI; however, the other equations did not outperform BMI. Moreover, BMI demonstrated poor discriminative ability for moderate/severe and severe OSA, with none of the other equations performing better in this context. Notable subgroup differences—particularly among younger females—suggest that tailoring screening strategies by age and sex may improve risk stratification and support refining obesity-based screening approaches.

Keywords: obstructive sleep apnea, body mass index, adiposity, equations, apnea–hypopnea index, discriminative ability, sex and age stratification

Introduction

Recurring obstructive episodes of the upper airway characterised by apneas and hypopneas describe obstructive sleep apnea (OSA).[1] The prevalence of OSA varies by age, sex, and country, with an estimated global prevalence for adults aged 30–69 at 936 million.[2] Early screening and diagnosis are important, given the association of untreated OSA with various adverse health outcomes, including reduced executive function[3] and increased motor vehicle accidents.[4] Recent studies highlight the need for improved screening tools to enhance OSA early detection, especially in primary care settings.[5,6]

Among the various risk factors for OSA considered in screening tools, obesity is one of the most important.[7] In 2016, the World Health Organization estimated that 650 million adults worldwide were obese.[8] Adiposity status is incorporated in several clinical screening tools for OSA, including the STOP-BANG questionnaire,[9] the Berlin questionnaire,[10] the NoSAS[11]

Received: 18 November 2024
Accepted: 17 April 2025
Published: 22 May 2025

and the BOAH scores,[12] which use BMI to measure obesity, given its simplistic formula.[8] However, all questionnaires rely on BMI, height, and weight, with no central obesity measures (eg, waist circumference) included.

Many studies estimate the relationship between obesity and OSA by utilizing BMI; some studies have found a positive correlation between BMI and AHI,[13] while others have found no relationship.[14] However, when compared to technologies including dual x-ray absorptiometry, a gold standard for body composition, BMI has been shown to misclassify adiposity status in a significant portion of individuals.[15,16] While other technologies are valuable tools in measuring body fat,[17] at a population level, they are not cost nor time effective options. Therefore, efforts have been made to develop novel equations using anthropometric data which are more strongly associated with true adiposity when compared to BMI.[18] A study by Krachler et al explored the relationship between multiple anthropometric-derived equations and various cardiovascular risk factors.[19] Two new equations, the Equation Córdoba for Estimation of Body Fat (ECORE-BF) and the Clínica Universidad de Navarra-Body Adiposity Estimator (CUN-BAE), have been developed to aid in estimating body fat.[20–22]

Little is known regarding these equations and their relationship with OSA prevalence and severity. Therefore, our research objective was to investigate correlations between different measures of obesity and markers of OSA presence and severity. We hypothesized stronger correlations between many of these novel adiposity equations and OSA presence/severity when compared to BMI. We also hypothesized that the relationship between different measures of obesity and OSA presence and severity varies by sex and age because of sex differences in fat distribution[7] and age-related changes in body composition.[23] In addition, postmenopausal changes and hormonal shifts in women may increase susceptibility to OSA. As such, stratified analysis by sex and age groups may improve prediction models and clinical risk stratification.

Materials and Methods

Study Design

We conducted a cross-sectional clinical cohort study using a database of consecutive adults (18 years and older) who underwent a diagnostic sleep study (Level 1 polysomnography [PSG]) between 2015 and 2017 in a large academic clinical center (Ottawa, Ontario, Canada). Ethics approval was completed through the Ottawa Health Science Network Research Ethics Board (OHSN-REB) (Protocol ID: 20210473-01H). Informed consent from the study participants for this specific study was not obtained. This study uses a database based on routinely collected de-identified clinical data registered and approved by the OHSN-REB. This study is a minimal-risk, meta-analysis, non-interventional, retrospective study that does not require enrollment or any sort of patient participation/contact and does not require any personally identifiable information. Patient confidentiality was upheld during the analysis and review of medical records, as only de-identified data was used in this study. The OHSN-REB abides by all applicable regulations and guidelines pertaining to human participant protection; these include, but are not limited to the Food and Drugs Act and applicable Regulations, the International Council on Harmonization Good Clinical Practice Guidelines, the Declaration of Helsinki: Ethical Principles for Medical Research Involving Human Subjects, the Tri-Council Policy Statement: Ethical Conduct for Research Involving Humans, and where applicable, US Federal Regulations (more details can be found at: https://www.ohri.ca/ohsn-reb/procedures.htm).

Data Source

The Ottawa Hospital Sleep database (NCT03834792) is a clinical database consisting of clinical and polysomnographic variables, including demographics, self-reported symptoms, medical history, and PSG indices. Each patient in the cohort underwent a full in-laboratory PSG recording that was scored by a sleep technologist and reviewed by a sleep physician. Height and weight were measured and recorded by a sleep technologist on the night of the sleep study. Details on the database are described elsewhere.[24]

PSG Indices Considered

Apnea index, apnea–hypopnea index (AHI), mean and minimum oxygen saturation in sleep, and percentage of sleep with an oxygen saturation of less than 88%. Where applicable, indices were quantified for position (ie, supine versus non-supine) and rapid eye movement (REM)/non-rapid eye movement (NREM) sleep. The sleep software used was Sandman 10.1, and event

scoring was in accordance with the AASM (American Academy of Sleep Medicine) criteria 2012.[25] The definition of hypopnea was as follows: ≥30% decrease in flow from baseline with an associated oxygen desaturation of ≥3% OR an associated arousal. AHI of 5–15, 15–30, and >30 were classified as mild, moderate, and severe OSA, respectively.

Adiposity-Estimating Equations

Fifteen equations, in addition to BMI, were selected based on data availability and a comprehensive literature review. The equations were as follows: the Rohrer index,[26] University of Navarra Clinic-Body Fat Estimator (CUN-BAE) index,[22] Equation Córdoba for Estimation of Body Fat (ECORE-BF),[20] Body surface index (BSI), Normalized weight-adjusted index (NWAI),[27] and equations by Larrson,[28] Heo,[29] Heitmann,[30] Visser,[31] Noppa,[32] Gallagher (1996[33] and 2000[34]) and Deurenberg (1989,[35] 1991,[36] and 1998). [37] All equations are described in Table 1.

Table 1 Adiposity Estimating Equations

Name of Equation/Anthropometric Measurement	**Equation/ Units**
Body Mass Index (BMI)	Weight (kg)/ [height (m)]^2
Rohrer index	Weight (kg)/ [height (m)]^3
Equation by Larsson et al	Female: BF%= [−24.18 + 1.181 x (weight (kg) / height (m))] / weight (kg) x 100%
	Male: BF%= [−30.84 + 1.120 x (weight (kg) /height (m))] / weight (kg) x 100%
Equation by Gallagher et al (2000)	BF%= 64.5–848 x (1/BMI) + 0.079 x age (yrs) −16.4 x sex* + 0.05 x sex x age (yrs) + 39.0 x sex x (1/BMI)
Equation by Deurenberg et al (1991)	BF%= 1.2 x BMI + 0.23 x age (yrs) −10.8 x sex* − 5.4
University of Navarra Clinic-Body Fat Estimator (CUN-BAE) index	BF%=−44.988+(0.503×age (yrs)) + (10.689×sex#)+(3.172×BMI) − (0.026×BMI^2) + (0.181×BMI×sex) − (0.02×BMI×age (yrs)) − (0.005×BMI^2×sex)+(0.00021×BMI^2×age (yrs))
Equation Córdoba for Estimation of Body Fat (ECORE-BF)	BF%=−97.102 + (0.123 x age (yrs)) + (11.900 x sex#) + (35.959 x (LnBMI))
Body Surface Index (BSI)	BSI = Weight (kg) / √ BSA
	BSA: weight (kg)$^{0.425}$ x height (cm)$^{0.725}$ x 0.007184
Normalized weight-adjusted index (NWAI)	[(weight (kg)/10) – (10 x height (m)) + 10]
Equation by Deurenberg et al (1998)	BF% = 1.294 x BMI + 0.20 x age (yrs) – 11.4 x sex* – 8.0
Equation by Heo et al (2013)	Female: BF%= [−17.6 + 1.72 x (weight (kg)/height (m)^2)]/ weight (kg) x 100%
	Male: BF%= [−24.0 + 1.77 x (weight (kg)/height (m)^2)]/ weight (kg) x 100%
Equation by Heitmann et al (1990)	Female: BF% = [0.988 x BMI + 0.344 x weight (kg) + 0.094 x age (yrs) – 30.180]/weight (kg) x 100%
	Male: BF% = [0.988 x BMI + 0.242 x weight (kg) + 0.094 x age (yrs) – 30.180]/weight (kg) x 100%
Equation by Gallagher et al (1996)	BF% = 1.46 x BMI + 0.12 x age (yrs) – 11.61 x sex* – 10.02
Equation by Visser et al (1994) (uses Siri's equation for BF%)	BD = 0.0226 x sex* – 0.0022 x BMI + 1.0605
	BF% = (4.95/BD – 4.50) x 100%
Equation by Noppa et al (1979)	BF% = [1.52 x BMI – 17.8]/weight (kg) x 100%
Equation by Deurenberg et al (1989)	Female: BF% = 0.672 x BMI + 26.6
	Male: BF% = 0.835 x BMI + 10.2

Notes: *Sex = 1 for males, 0 for females. #Sex = 0 for male, 1 for female.
Abbreviations: BD, body density; BF%, body fat percentage; BMI, body mass index; BSA, body surface area; BSI, body surface index.

https://doi.org/10.2147/NSS.S504426

Analyses: Descriptive statistics were used to characterize the cohort. We used Pearson correlations with 95% confidence intervals to assess the relationship between measures of OSA severity and adiposity estimating equations/anthropometric data listed above. A Pearson correlation coefficient of ±0.90 to 1.00 was interpreted as a very high correlation, ± 0.70 to 0.90 as a high correlation, ± 0.50 to 0.70 as a moderate correlation, ± 0.30 to 0.50 as low correlation and 0.0 to ±0.30 as a negligible correlation.[38,39] Logistic regressions were used to assess the relationship and quantify the discriminative ability of various adiposity equations and the presence of moderate/severe (AHI>15) or severe OSA (AHI>30). The estimates, odds ratios (OR) and 95% confidence intervals (CIs), were standardized to report the effect per interquartile range (IQR), comparing the 75th to 25th percentile. The discriminative ability was assessed using the c-statistic.[40] The c-statistic value ranges from 0.5 to 1 with a value of 0.7–0.8, 0.8–0.9, and >0.9 interpreted as acceptable, excellent, and outstanding discrimination, respectively.[41]

Secondary analysis: To understand the effect of sex and age on the relationship between measures of OSA and obesity, given the importance of these variables on body composition,[42] we repeated all analyses described above stratified by sex and age (<50 vs ≥50 as based on the median age distribution in the cohort). The age of 50 years is also often considered a clinically important cutoff due to several physiological (eg, close to the average age of menopause [43]), epidemiological factors (eg, increased prevalence of moderate to severe OSA [44] and associated increased cardiovascular risk).[45]

The number of missing data for AHI, used to define OSA severity, ranged from 128 (2.5% for BMI) to 131 (2.6% for Heo and Heitmann equations). Since complete case analysis is considered reasonable and unlikely to introduce significant bias when missing data is below 5%,[46] we proceeded with complete case analysis.

Results

Descriptive Data

The descriptive data in the total cohort and by subgroups are shown in Table 2 and Supplementary Table 1. Of 5021 adult individuals included in the current analysis, 2679 (53.4%) were male, and 2569 (51.2%) were 50 years and older. The mean age was 49.5 years (SD of 14.9), while the mean BMI and AHI were 30.0 kg/m^2 (SD of 6.7) and 13.8/hr (SD of 15.79), respectively. The percentage of individuals with an AHI of <5, 5–15, 15–30, and >30 were 36.0% (1807), 31.5% (1581), 19.9% (997), and 12.7% (636), respectively.

Correlative Analysis

Table 3 shows Pearson correlations between adiposity estimating equations and indices derived from the PSG in the total cohort. All adiposity equations showed a statistically significant positive association with AHI ranging from 0.05 (Deurenberg 1989) to 0.31 (BSI). Most correlations were negligible (0.0 to ±0.3); however, BSI and NWAI were consistent with low correlations (± 0.30 to 0.50). Notably, BMI had a correlation coefficient of 0.29. Moreover, all equations were negatively associated with mean oxygen saturation in sleep. Correlation coefficients ranged from –0.11 (Deurenberg 1989) to –0.30 (Deurenberg 1991), thus negligible, but approaching low correlations. BMI had a correlation coefficient of −0.27. Similar findings, approaching low, but statistically significant, correlations were evident for other respiratory-related PSG variables (Table 3).

Logistic Regression and Receiver Operating Characteristic (ROC) Curves

Figures 1–3 and Supplementary Table 2 present ROC curve analyses and ORs with 95% CIs and c-statistics from logistic regressions for the total cohort. The odds of being diagnosed with severe OSA associated with an IQR increase in obesity measures ranged from 1.12 to 1.97, with the highest odds associated with an increase in BSI (Figure 3 and Supplementary Table 2). The c-indices ranged from 0.52 to 0.67, demonstrating a less than acceptable discriminative ability, with BSI, NWAI, and BMI showing the greatest discriminative ability (Figure 1 and Supplementary Table 2). Similar trends were evident regarding moderate/severe OSA (Figure 2 and Supplementary Table 2).

Correlative Analysis Stratified by Sex, Age, and Sex/Age Subgroups

When the cohort was stratified based on sex, age, and both age and sex, similar results were found (Supplementary Tables 3–10). While some adiposity equations had mildly stronger correlation coefficients than BMI, others had minimally weaker correlation coefficients with AHI. Regardless, all associations with AHI were generally negligible

Table 2 Anthropometric and Polysomnographic Variables for the Study Population: Total and by Sex and Age Subgroups

Variable	Total				Male				Female				Age <50				Age > 50			
	N	Mean		SD	N	Mean		SD	N	Mean		SD	N	Mean		SD	N	Mean		SD
Age (years)	5021	49.5	±	14.9	2679	49.5	±	14.9	2341	49.5	±	14.8	2452	37.0	±	8.3	2569	61.4	±	8.8
Weight (kg)	4892	87.4	±	21.1	2612	92.5	±	19.4	2279	81.6	±	21.4	2403	89.3	±	22.9	2489	85.7	±	19.0
Height (cm)	4894	170.5	±	10.1	2612	176.9	±	7.6	2281	163.3	±	7.4	2404	171.8	±	10.0	2490	169.3	±	10.1
BMI (kg/m2)	4893	30.0	±	6.7	2612	29.5	±	5.7	2280	30.6	±	7.7	2403	30.2	±	7.2	2490	29.9	±	6.3
ESS	4983	8.4	±	4.7	2659	8.2	±	4.6	2323	8.7	±	4.9	2435	8.9	±	4.7	2548	7.9	±	4.7
PSG indices																				
Apnea Index (events/hr)	5021	3.1	±	7.5	2679	4.1	±	8.9	2341	2.0	±	5.2	2452	2.3	±	6.8	2569	3.9	±	8.0
AHI (events/hr)	5021	13.8	±	15.8	2679	16.5	±	16.9	2341	10.8	±	13.8	2452	10.9	±	14.6	2569	16.7	±	16.3
Min. wake SaO2 (%)	5018	75.7	±	29.2	2677	75.3	±	29.2	2340	76.2	±	29.2	2452	77.7	±	28.8	2566	73.9	±	29.5
Sleep with SaO2 < 88% (%)	5021	1.5	±	7.8	2679	1.5	±	7.5	2341	1.5	±	8.2	2452	0.9	±	6.3	2569	2.2	±	9.0
Mean Sleep SaO2 (%)	5020	94.8	±	2.0	2678	94.7	±	1.9	2341	95.0	±	2.1	2451	95.5	±	1.7	2569	94.1	±	2.1
Min Sleep SaO2 (%)	5019	83.3	±	17.6	2677	82.4	±	18.2	2341	84.2	±	16.8	2451	84.3	±	18.6	2568	82.3	±	16.4
Adiposity-Estimating Equations																				
Rohrer index	4891	17.7	±	4.3	2611	16.7	±	3.3	2279	18.8	±	4.9	2403	17.6	±	4.5	2488	17.8	±	4.1
Larrson	4890	34.4	±	9.2	2611	28.7	±	6.2	2279	40.9	±	7.6	2403	34.3	±	9.3	2487	34.6	±	9.1
Gallagher 2000	4892	32.2	±	9.0	2612	26.1	±	5.4	2280	39.0	±	7.1	2403	30.7	±	9.3	2489	33.6	±	8.4
Deurenberg 1991	4892	36.2	±	10.6	2612	30.6	±	7.5	2280	42.7	±	9.8	2403	33.5	±	10.8	2489	38.9	±	9.6
CUN BAE	4892	35.9	±	9.6	2612	30.2	±	6.9	2280	42.4	±	7.9	2403	34.5	±	10.2	2489	37.2	±	8.7

(*Continued*)

Table 2 (Continued).

Variable	Total				Male				Female				Age <50				Age > 50			
	N	Mean		SD	N	Mean		SD	N	Mean		SD	N	Mean		SD	N	Mean		SD
ECORE BF	4892	36.0	±	10.1	2612	30.1	±	6.8	2280	42.8	±	8.9	2403	34.5	±	10.4	2489	37.5	±	9.6
BSI	4891	61.6	±	11.1	2611	63.6	±	10.0	2279	59.2	±	11.9	2403	62.4	±	12.1	2488	60.8	±	10.1
NWAI	4891	1.7	±	1.9	2611	1.6	±	1.8	2279	1.8	±	2.1	2403	1.7	±	2.1	2488	1.6	±	1.8
Deurenberg 1998	4892	34.7	±	11.1	2612	28.7	±	7.8	2280	41.5	±	10.4	2403	32.3	±	11.5	2489	37.0	±	10.3
Heo	4890	35.5	±	8.8	2611	29.9	±	5.7	2279	42.0	±	7.1	2403	34.9	±	8.6	2487	36.2	±	8.9
Heitmann	4890	32.5	±	8.9	2611	27.3	±	5.5	2279	38.4	±	8.3	2403	30.8	±	9.1	2487	34.1	±	8.4
Gallagher 1996	4892	33.6	±	11.9	2612	27.4	±	8.4	2280	40.6	±	11.4	2403	32.2	±	12.5	2489	34.9	±	11.2
Visser	4892	42.0	±	9.6	2612	36.3	±	6.1	2280	48.6	±	8.7	2403	42.1	±	10.0	2489	41.9	±	9.2
Noppa	4891	31.3	±	6.3	2611	28.8	±	4.5	2279	34.2	±	6.7	2403	30.8	±	6.2	2488	31.8	±	6.3
Deurenberg 1989	4892	40.6	±	7.9	2612	34.9	±	4.8	2280	47.2	±	5.2	2403	40.6	±	8.1	2489	40.6	±	7.7

Note: Data presented as means and standard deviations.
Abbreviations: AHI, apnea-hypopnea index; BMI, body mass index; ESS, Epworth Sleepiness Scale; PSG, polysomnography; SaO2, oxygen saturation.

https://doi.org/10.2147/NSS.S504426

Table 3 Pearson Correlations Between Sleep-Related Breathing Polysomnography Parameters and Adiposity Estimating Equations for the Total Cohort

Variables	BMI	Rohrer index	Larrson	Gallagher 2000
Apnea Index	0.07 (0.04–0.10)	0.05 (0.03–0.08)	−0.03 (−0.06–0.00)	−0.02 (−0.05–0.01)
AHI	0.29 (0.26–0.31)	0.25 (0.22–0.27)	0.10 (0.07–0.13)	0.11 (0.08–0.14)
Supine AHI	0.22 (0.19–0.25)	0.17 (0.14–0.20)	0.03 (0.00–0.06)	0.04 (0.01–0.07)
Non-Supine AHI	0.33 (0.30–0.36)	0.30 (0.27–0.32)	0.16 (0.13–0.19)	0.16 (0.13–0.19)
REM AHI	0.39 (0.36–0.41)	0.37 (0.35–0.40)	0.26 (0.23–0.29)	0.27 (0.24–0.30)
Non-REM AHI	0.25 (0.23–0.28)	0.21 (0.18–0.24)	0.06 (0.04–0.09)	0.07 (0.04–0.10)
Mean wake SaO2	−0.15 (−0.18–0.13)	−0.14 (−0.17–0.11)	−0.07 (−0.10–0.04)	−0.10 (−0.13–0.07)
% sleep with SaO2 <88%	0.08 (0.05–0.11)	0.08 (0.06–0.11)	0.06 (0.03–0.09)	0.07 (0.04–0.10)
Mean sleep SaO2	−0.27 (−0.30–0.25)	−0.25 (−0.28–0.23)	−0.16 (−0.19–0.14)	−0.20 (−0.23–0.17)
Mean REM SaO2	−0.29 (−0.31–0.26)	−0.27 (−0.30–0.25)	−0.18 (−0.20–0.15)	−0.21 (−0.24–0.18)
Mean non-REM SaO2	−0.26 (−0.29–0.24)	−0.24 (−0.27–0.22)	−0.15 (−0.18–0.13)	−0.19 (−0.22–0.16)
Min Sleep SaO2	−0.09 (−0.12–0.06)	−0.08 (−0.11–0.05)	−0.04 (−0.07–0.01)	−0.04 (−0.07–0.01)
Variables	**Deurenberg 1991**	**CUN BAE**	**ECORE BF**	**BSI**
Apnea Index	0.03 (0.00–0.06)	0.01 (−0.02–0.03)	0.00 (−0.02–0.03)	0.09 (0.06–0.12)
AHI	0.20 (0.18–0.23)	0.16 (0.13–0.18)	0.16 (0.13–0.19)	0.31 (0.28–0.33)
Supine AHI	0.13 (0.10–0.16)	0.09 (0.06–0.12)	0.08 (0.05–0.11)	0.27 (0.24–0.30)
Non-Supine AHI	0.25 (0.23–0.28)	0.21 (0.18–0.24)	0.21 (0.19–0.24)	0.33 (0.31–0.36)
REM AHI	0.35 (0.33–0.38)	0.31 (0.28–0.34)	0.32 (0.29–0.34)	0.36 (0.33–0.38)
Non-REM AHI	0.16 (0.14–0.19)	0.12 (0.09–0.15)	0.12 (0.09–0.15)	0.28 (0.26–0.31)
Mean wake SaO2	−0.17 (−0.20–0.14)	−0.12 (−0.15–0.09)	−0.13 (−0.15–0.10)	−0.16 (−0.18–0.13)
% sleep with SaO2 <88%	0.09 (0.07–0.12)	0.07 (0.05–0.10)	0.08 (0.05–0.11)	0.06 (0.04–0.09)
Mean sleep SaO2	−0.30 (−0.32–0.27)	−0.23 (−0.26–0.20)	−0.24 (−0.27–0.21)	−0.27 (−0.29–0.24)
Mean REM SaO2	−0.31 (−0.34–0.28)	−0.24 (−0.27–0.21)	−0.25 (−0.28–0.22)	−0.27 (−0.29–0.24)
Mean non-REM SaO2	−0.28 (−0.31–0.26)	−0.22 (−0.25–0.19)	−0.23 (−0.25–0.20)	−0.26 (−0.29–0.24)
Min Sleep SaO2	−0.06 (−0.09–0.03)	−0.05 (−0.08–0.02)	−0.05 (−0.08–0.02)	−0.10 (−0.13–0.07)
Variables	**NWAI**	**Deurenberg 1998**	**Heo**	**Heitmann**
Apnea Index	0.08 (0.05–0.11)	0.03 (0.00–0.05)	−0.02 (−0.05–0.00)	0.00 (−0.02–0.03)
AHI	0.30 (0.27–0.32)	0.20 (0.17–0.22)	0.08 (0.05–0.10)	0.15 (0.12–0.18)
Supine AHI	0.23 (0.20–0.26)	0.12 (0.09–0.15)	0.00 (−0.03–0.03)	0.08 (0.05–0.11)
Non-Supine AHI	0.34 (0.31–0.36)	0.25 (0.22–0.27)	0.13 (0.10–0.16)	0.20 (0.17–0.23)
REM AHI	0.39 (0.36–0.41)	0.35 (0.32–0.37)	0.23 (0.20–0.26)	0.31 (0.28–0.34)
Non-REM AHI	0.26 (0.24–0.29)	0.16 (0.13–0.18)	0.04 (0.01–0.07)	0.11 (0.08–0.14)

(*Continued*)

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Table 3 (Continued).

Mean wake SaO2	−0.15 (−0.18–0.13)	−0.15 (−0.18–0.13)	−0.06 (−0.09–0.04)	−0.13 (−0.16–0.11)
% sleep with SaO2 <88%	0.08 (0.05–0.11)	0.09 (0.06–0.12)	0.06 (0.04–0.09)	0.07 (0.05–0.10)
Mean sleep SaO2	−0.27 (−0.30–0.25)	−0.28 (−0.30–0.25)	−0.14 (−0.17–0.11)	−0.24 (−0.27–0.22)
Mean REM SaO2	−0.29 (−0.31–0.26)	−0.29 (−0.32–0.27)	−0.16 (−0.19–0.13)	−0.25 (−0.28–0.22)
Mean non-REM SaO2	−0.26 (−0.29–0.24)	−0.27 (−0.29–0.24)	−0.13 (−0.16–0.10)	−0.23 (−0.26–0.21)
Min Sleep SaO2	−0.09 (−0.12–0.06)	−0.06 (−0.09–0.03)	−0.02 (−0.05–0.00)	−0.05 (−0.08–0.02)
Variables	**Gallagher 1996**	**Visser**	**Noppa**	**Deurenberg 1989**
Apnea Index	0.01 (−0.01–0.04)	−0.03 (−0.05–0.00)	0.03 (0.00–0.05)	−0.05 (−0.08–0.03)
AHI	0.18 (0.16–0.21)	0.11 (0.09–0.14)	0.17 (0.14–0.19)	0.05 (0.02–0.08)
Supine AHI	0.10 (0.07–0.13)	0.03 (0.00–0.06)	0.10 (0.07–0.13)	−0.03 (−0.06–0.00)
Non-Supine AHI	0.24 (0.21–0.27)	0.18 (0.15–0.21)	0.21 (0.18–0.24)	0.12 (0.09–0.15)
REM AHI	0.34 (0.31–0.36)	0.27 (0.25–0.30)	0.30 (0.28–0.33)	0.21 (0.18–0.24)
Non-REM AHI	0.14 (0.12–0.17)	0.08 (0.05–0.11)	0.13 (0.10–0.16)	0.02 (−0.01–0.05)
Mean wake SaO2	−0.13 (−0.15–0.10)	−0.06 (−0.09–0.03)	−0.12 (−0.15–0.09)	−0.03 (−0.05–0.00)
% sleep with SaO2 <88%	0.08 (0.05–0.11)	0.06 (0.03–0.09)	0.08 (0.05–0.10)	0.05 (0.03–0.08)
Mean sleep SaO2	−0.25 (−0.27–0.22)	−0.16 (−0.19–0.13)	−0.21 (−0.23–0.18)	−0.11 (−0.14–0.09)
Mean REM SaO2	−0.26 (−0.29–0.24)	−0.19 (−0.21–0.16)	−0.22 (−0.25–0.19)	−0.14 (−0.17–0.11)
Mean non-REM SaO2	−0.23 (−0.26–0.21)	−0.15 (−0.18–0.12)	−0.20 (−0.22–0.17)	−0.10 (−0.13–0.07)
Min Sleep SaO2	−0.06 (−0.09–0.03)	−0.04 (−0.07–0.01)	−0.05 (−0.08–0.02)	−0.02 (−0.05–0.00)

Notes: Estimates are presented as correlation coefficients (r) and 95% confidence intervals.
Abbreviations: AHI, apnea-hypopnea index; SaO2, oxygen saturation.

to low, with the highest correlation coefficient noted for REM AHI of 0.53 for Deurenberg 1991 and 1998 equation in female younger than 50 years old.

Logistic Regression and ROC Curves Stratified Based on Sex, Age, and Sex/Age Subgroups

When the cohort was stratified based on sex, age, and both age and sex, similar results were found (Figure 4, Supplementary Tables 11 to 18 and Supplementary Figures 1–8). While some adiposity equations had mildly higher discriminatory ability for severe OSA compared to BMI, others had a minimally weaker discriminatory ability. Regardless, most equations' discriminatory ability ranged from less than acceptable to acceptable. However, in females younger than 50, c-indices ranged from 0.73 to 0.81, demonstrating acceptable to excellent discriminative ability, with Gallagher 2000, Deurenberg 1991, ECORE BF, Deunrenberg 1998, Heitmann, and Gallagher 1996 all showing the greatest discriminative ability (c-indices of 0.81). However, other equations, including BMI, had c-indices of 0.80, also corresponding with excellent discriminative ability (Supplementary Table 17 and Supplementary Figure 7).

Discussion

To our knowledge, this is the first study to comprehensively assess the relationship between the severity of OSA and a particular set of BMI, height, and weight-based adiposity-estimating equations, including the Rohrer index, CUN-BAE

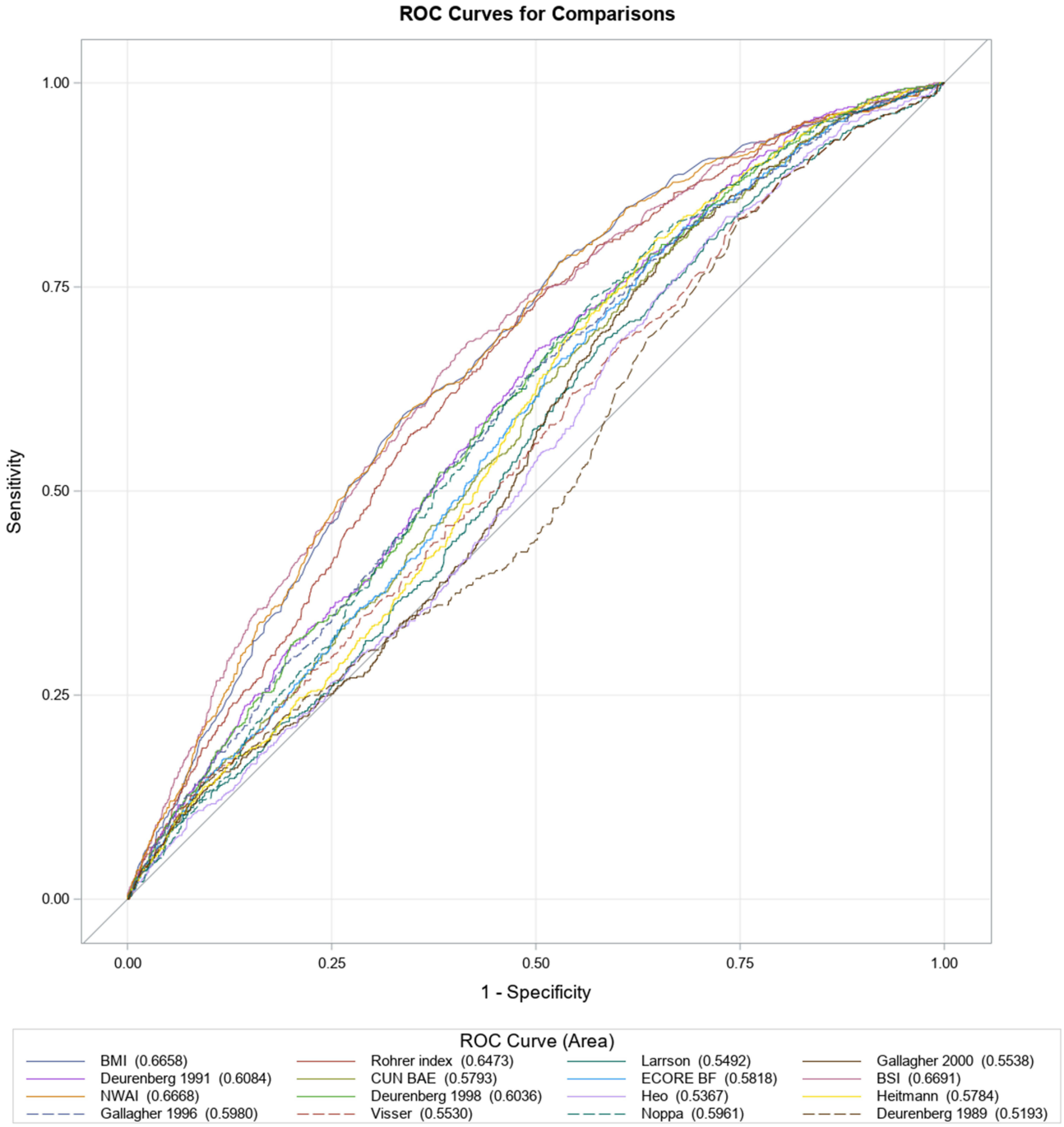

Figure 1 Receiver operating characteristic (ROC) curve analyses of BMI and 15 adiposity-estimating equations in discrimination of individuals with severe obstructive sleep apnea (OSA) versus not in the total study population.

index, ECORE-BF, BSI, NWAI, and equations by Larrson, Heo, Heitmann, Visser, Noppa, Gallagher (1996 and 2000) and Deurenberg (1989, 1991, and 1998), compared with BMI. We did not demonstrate stronger correlations between novel adiposity equations and OSA presence/severity compared to BMI, as hypothesized, regardless of stratification. In the total study population, BMI was poorly correlated with AHI, and the other equations did not outperform BMI. Similarly, BMI demonstrates poor discriminative ability for moderate/severe and severe OSA; however, none of the other equations performed better in this context. This study further supports the argument that measures of obesity alone should

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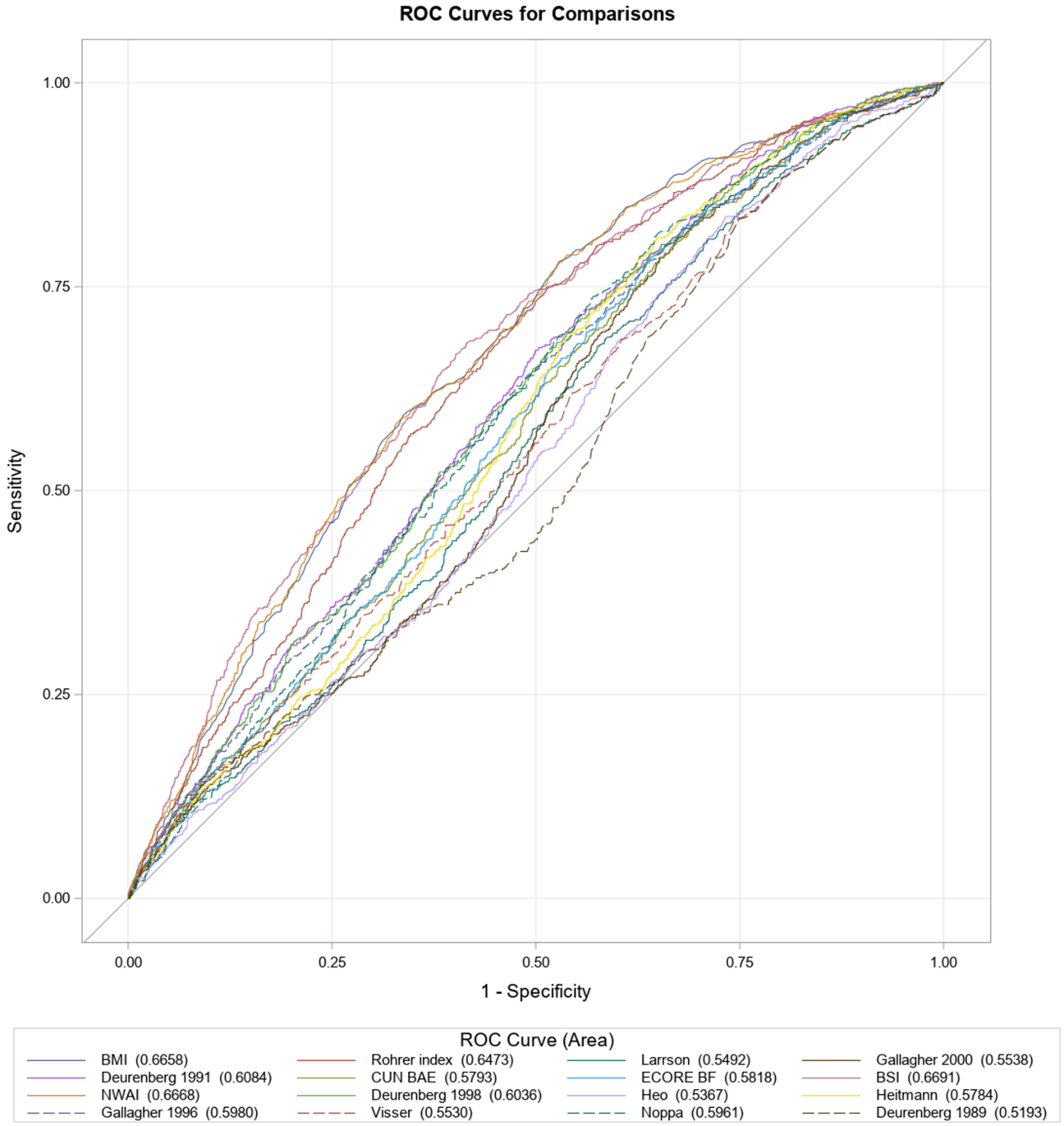

Figure 2 Receiver operating characteristic (ROC) curve analyses of BMI and 15 adiposity-estimating equations in discrimination of individuals with moderate to severe obstructive sleep apnea (OSA) versus not in the total study population.

not be used to discriminate between severe or moderate/severe OSA. Critically, no adiposity-estimating equations outperformed BMI.

In the literature, BMI has taken precedence for estimating obesity due to its ease of calculation. BMI is strongly associated with body fat,[47] but can misclassify obesity in a proportion of individuals.[15,16,48] Studies have demonstrated a relationship between increasing BMI and OSA[13,49] and a similar correlation (r=0.38) between AHI and BMI,[13] compared to the current study. Conversely, other studies found no relationship between AHI and BMI, but did note a negative relationship (r=−0.42) between minimum oxygen saturation and BMI,[14] which was stronger than in the current

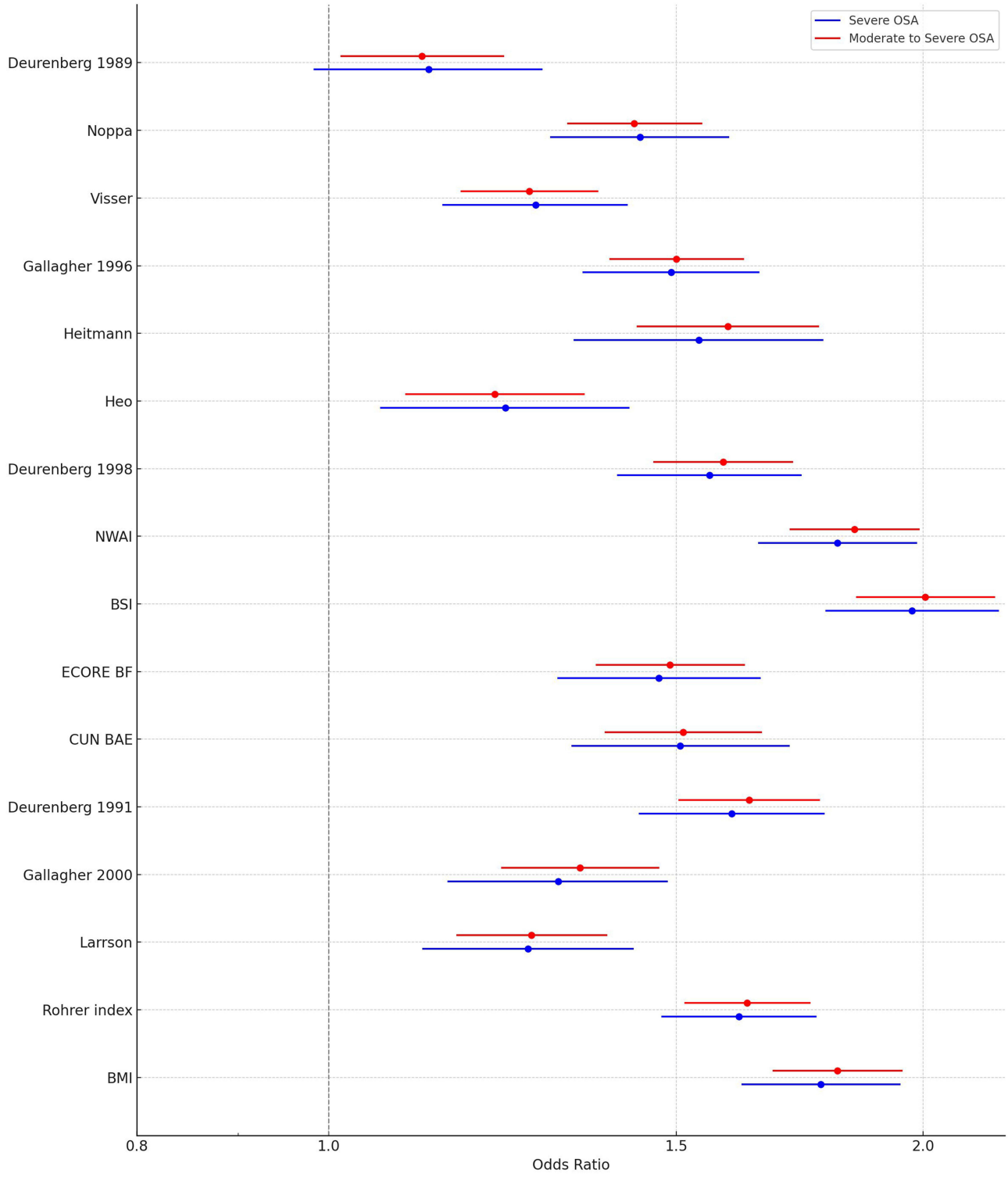

Figure 3 Forest plot of odds ratio (OR) and 95% confidence interval (CI) of the association between BMI and 15 adiposity-estimating equations and severe or moderate to severe obstructive sleep apnea (OSA) for the total study population.

study. Notably, for most of the used equations, cut-offs do not exist to qualify someone as underweight, normal weight, overweight, or obese as they do for BMI,[8] and thus, estimates per IQR were used in the present study. Further studies are warranted to assess this complex relationship. Importantly, little data exists regarding OSA and obesity defined by the adiposity equations investigated in this study. At the time of the current manuscript preparation, only one study was

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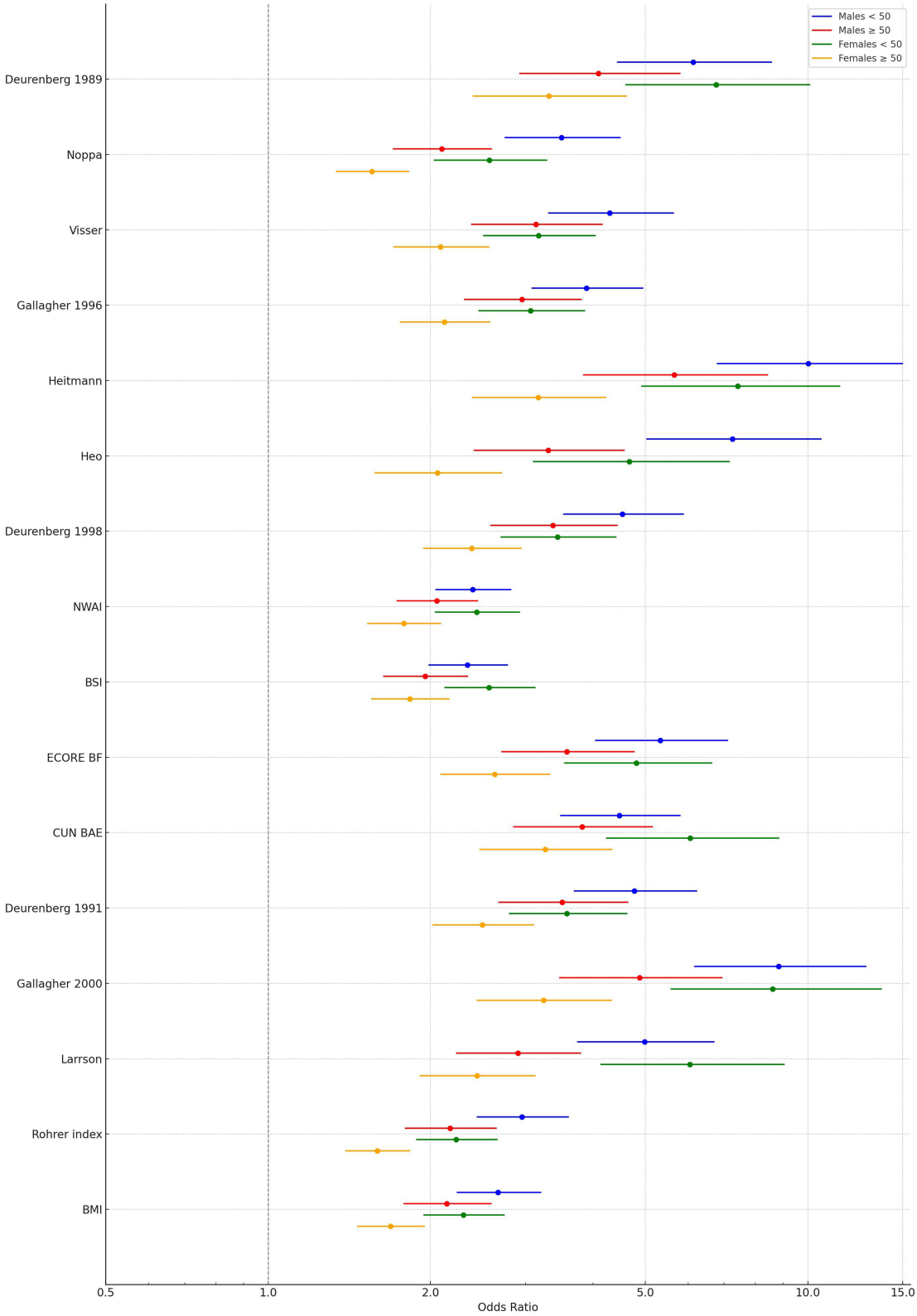

Figure 4 Forest plot of odds ratio (OR) and 95% confidence interval (CI) of the association between BMI and 15 adiposity-estimating equations and moderate to severe obstructive sleep apnea (OSA) by sex and age subgroups.

available; this study showed an increased risk of self-reported sleep-disordered breathing with increasing Rohrer index in primary school students (OR of 1.82; 95% CI: 1.46–2.27; p<0.001).[50] However, compared to our study, no objective measures of OSA were used, nor was a comparison to BMI made.

In the current study for females younger than 50, many obesity estimating equations, including BMI, showed an excellent discriminative ability for severe OSA. The differences observed when stratified by sex and age could be explained by variations in body fat percentages and distribution in males vs females,[51,52] as women, on average, have more body fat than men[53] and, as body fat can increase with age.[54] However, equations incorporating sex-specific formulas may have performed poorly as they technically should not be used to assess discriminative ability in the total population, given different distributions for males and females. This assumption was confirmed by the fact that when stratified by age and sex, the discriminative ability for equations was very close (ie, smaller variation than in the total cohort). Furthermore, the relationship between BMI and AHI, as a predictor of OSA, varies between men and women. When assessing the impact of BMI on AHI, mediating factors—characteristics that help explain the relationship between two variables—have been shown to vary by sex. Upper airway mechanics have been additionally shown to be differentially controlled by sex, age, and BMI, and partly mediate the relationship between these risk factors and OSA.[55] For example, Nokes et al in the study involved 2146 individuals (43% women), showing that BMI was significantly associated with AHI across both sexes and, at the same time, demonstrated the different AHI-BMI mediating factors between men and women.[56] Specifically, while in men, the correlation between BMI and AHI was influenced by reductions in upper airway stiffness, circulatory delay, and an increased arousal threshold, in women, only upper airway stiffness and circulatory delay significantly mediate the BMI-AHI relationship.[56] Taking together, these findings suggest the potential value of developing or adapting screening tools that account for sex- and age-related differences in obesity measures and OSA risk. For example, incorporating age- and sex-specific thresholds or weighting different anthropometric measures differently in prediction models could improve early identification of individuals at risk for OSA, particularly in underserved or underdiagnosed populations such as younger women. This approach may be especially relevant in specific settings, such as population-level screening, where access to full diagnostic testing is limited. However, given the exploratory nature of these stratified analyses and the potential for type I error due to multiple comparisons, these findings should be considered hypothesis-generating and require validation in external cohorts.

Regarding other metabolic conditions, Kracher et al showed BMI and adiposity estimated by averaging values obtained by Larsson, Gallagher, and Deurenberg (1991 and 1998), had higher discriminatory power when compared to DEXA for cardiometabolic risk markers (1.9% for BMI, 3.7% for adiposity-estimating equations p<0.01).[19] Both the Deurenberg 1991 equation and CUN-BAE have also been shown to aid in identifying metabolic syndrome,[57] which has been linked to OSA.[58] For women, the Deurenberg 1991 equation and CUN-BAE had similar areas under the curve (AUC >0.90 and >0.89, respectively) for predicting metabolic syndrome (depending on the metabolic syndrome model).[57] For men, both the Deurenberg and CUN-BAE had AUC values of >0.84 (depending on the metabolic syndrome model).[57] In our current paper, it is unclear in the correlative analysis why the Deurenberg 1991 equation seemed to consistently have the highest r-value once the data was stratified; further investigations are required. Notably, the Deurenberg 1991 and other equations take into consideration age, sex, and BMI, which may aid in the performance.

Another obesity measure that could be considered while investigating the relationship between the severity of OSA and obesity is the Body Roundness Index (BRI). While newer, BRI specifically aims to augment the shortcomings of BMI by measuring visceral fat via waist circumference in addition to height and weight, as visceral fat is more closely linked to health risks when compared to overall body weight.[59] Zhang et al explore further the relationship between BRI and all-cause mortality in their study of US adults using data from the National Health and Nutrition Examination Survey from 1999 to 2018 (N=32,995 and a median follow-up period of about 10 years).[60] This study demonstrated an increasing trend of BRI during a nearly 20-year period, especially in women and elderly individuals, and a U-shaped association between BRI and all-cause mortality,[60] suggesting that BRI as a noninvasive screening tool could be incorporated into public health practice, pending validation in future independent studies.

Our study is affected by several limitations. First, single-center cross-sectional study design and using clinical- (vs community) based data may affect the generalizability of our findings and may introduce selection bias. However, it is generalizable to the Canadian population given the current cohort's median BMI of 28.9 kg/m2 was similar to the average BMI found in the Canadian Primary Care Sentinel Surveillance Network[61] of 28.5 kg/m2 in January 2011 and 29.1 kg/m2 in

December 2016. Regarding the percentage of severe OSA (12.7% in the current study), various Canadian studies differ regarding the percentage of individuals referred for a sleep study and ultimately diagnosed with severe OSA. For example, two studies, one from Saskatchewan and the other from British Columbia, reported severe OSA incidence of 5.2%[62] and 26%,[63] respectively. Notably, the Sleep Heart Health Study (5800 participants) had 21% of the population with moderate-severe sleep apnea.[64] We also cannot exclude the possibility of measurement bias related to the BMI measurements (clinically routinely collected data) and variability in AHI due to night-to-night fluctuations. Thus, our results are needed to be externally validated, including on community-based cohorts. Another limitation was that only 15 obesity estimating equations were used in the analysis. Many more do exist in the literature,[65] but we were limited by the variables accessible in the dataset – we did not have access to some variables required for other calculated equations, such as neck, waist, and hip circumference. Of note, both an increased waist circumference and waist-to-hip ratio have been associated with OSA severity and prevalence.[66] Therefore, future studies should incorporate other adiposity estimating equations and imaging techniques,[67] specifically focusing on fat distribution, to assess associations with OSA. To support the importance of fat distribution, the study that utilized abdominal magnetic resonance imaging before and after a weight loss intervention demonstrated that improved AHI with weight loss was primarily mediated by tongue fat reductions.[68]

Furthermore, while it was not in our study's focus, we could not exclude a modifiable effect of OSA endotypic traits on the relationship between adiposity estimating equations and OSA severity.[69] For example, obese individuals often exhibit high upper airway collapsibility and poor upper airway compensation due to excess fat deposition around the airway and reduced lung volumes, while in non-obese individuals, OSA is more often driven by high loop gain, low arousal thresholds, or neuromuscular dysfunction rather than airway anatomy.[1] OSA endotypic traits could also explain 30% of the relative sex differences in non-REM AHI.[70] While exploring the intersection between age and sex, for men, age has been shown to correlate with increased collapsibility, increased loop gain, and decreased arousal threshold, whereas, in women, OSA endotypic traits were not associated with age, except for an increase in loop gain with advancing age.[71]

Overall, this is the first study to assess the relationship between measures of OSA severity and 15 BMI, height, and weight-based adiposity equations compared to BMI. While some equations had slightly stronger correlations with AHI, most had negligible or low correlations with AHI, regardless of stratification. While the discriminative ability for OSA for most equations was less than acceptable, in females younger than 50, many equations (including BMI) had excellent discriminative ability. These results further support the limitation of both the BMI, as one of the most commonly used measurements in clinics and research, and other evaluated BMI-based measures of adiposity in screening for OSA within clinical guidelines. However, if confirmed in future studies, in females under 50, BMI could serve as a simple screening tool for identifying those at risk for OSA. Future studies are required to both externally validate our findings and to further explore the relationship between other adiposity-estimating equations and OSA. Utilizing multi-parameter screening by combining BMI with measures of fat distribution, such as neck, waist, and hip circumference, demographic characteristics, relevant symptoms and the presence of relevant comorbidities, such as cardiovascular, may provide better discriminative value than using measures of adiposity alone. Advanced statistical methods, including machine learning,[72] would also help to improve the discriminative power and personalized screening approach for OSA.

Data Sharing Statement

The dataset from this study is available under REB approval upon reasonable request from the corresponding author.

Acknowledgments

We acknowledge the Ottawa Methods Center at the Ottawa Hospital Research Institute for aiding with data analysis. The authors also wish to thank the research assistants at the Ottawa Hospital Research Institute, Moussa Meteb, Aseel Ahmad, and Randa Ahmad, for their role in databases preparation as well as Mouaz Saymeh for his role in the manuscript preparation.

Author Contributions

DW, GC, SDA, ZWG, JF, and TK were involved in the study conceptualization and methodology. MS, GC, SDA, ZWG, JF, ES, and BY were involved in validation, visualization, and writing (review and editing). DW and TK were also

involved in investigation, formal analysis, validation, and writing (original draft). TK was additionally involved in project administration and supervision (of DW) and is a custodian of the TOH Sleep Database (data curation). ES was additionally involved in formal analysis. TK, ES, and DW had full access to all the data in the study and take responsibility for the integrity of the data and the accuracy of the data analysis. All co-authors (DW, MS, GC, SDA, ZWG, JF, ES, BY, TK) agreed on the journal to which the article will be submitted, reviewed, and agreed on all versions of the article before submission, during revision, the final version accepted for publication, and any significant changes introduced at the proofing stage, and take responsibility and be accountable for the contents of the article.

Disclosures/Disclosure of Interest

The TOH Sleep Database was created with the support from the Ottawa Hospital Sleep Walk and the University of Ottawa Department of Medicine (DOM) Developmental Research Grant (Ottawa, Ontario, Canada). The analyses, conclusions, opinions, and statements expressed herein are solely those of the authors and do not reflect those of the funding or data sources; no endorsement is intended or should be inferred.

The authors have no conflicts of interest to declare. TK was supported by the PSI Foundation when the project was conducted. Outside of the submitted work, TK projects are supported by the CIHR and CHEST foundations.

The abstract of this paper was presented at the 2022 American Thoracic Society International Conference as a poster presentation with interim findings. The poster's abstract was published in the American Journal of Respiratory and Critical Care Medicine: https://doi.org/10.1164/ajrccm-conference.2022.205.1_MeetingAbstracts.A2709. Only the abstract was presented at the conference with preliminary results: correlation coefficients only for the total sample without stratifications. Since the abstract was presented, we have performed several additional analyses, including logistic regressions, presenting c-indices, visualizing the discriminative ability and subgroup analyses stratifying by age and sex; this is why additional co-investigators were involved in bringing expertise in the analytic approaches and interpretations of the obtained results.

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