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ORIGINAL RESEARCH

Association Between Metabolic Score for Insulin Resistance (METS-IR) and Risk of Obstructive Sleep Apnea: Analysis of NHANES Database and a Chinese Cohort

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Purpose: Insulin resistance (IR) plays a significant role in the development of obstructive sleep apnea (OSA). The metabolic score for insulin resistance (METS-IR) is a novel method for assessing IR. This study aims to explore the relationship between METS-IR and the risk of OSA.

Patients and Methods: This cross-sectional study included a total of 8297 subjects from NHANES (National Health and Nutrition Examination Survey) database, as well as 581 patients who underwent sleep monitoring in Renmin Hospital of Wuhan University. Logistic regression, subgroup analysis, and receiver operating characteristic (ROC) curve analysis were employed for evaluation.

Results: In the American population, a significant positive association was found between METS-IR and increased risk of OSA. For each unit increase in METS-IR, the risk of OSA increased by 4.4% (OR= 1.044; 95% CI: 1.037–1.059; P <0.001). A similar relationship was observed in the Chinese population. Multivariate Logistic regression model showed that for each unit increase in METS-IR, the prevalence of OSA increased by 6.7% (OR= 1.067; 95% CI: 1.035–1.103; P <0.001), and apnea-hypopnea index (AHI) increased by 0.732 (β= 0.732; 95% CI: 0.573–0.732; P <0.001). Gender subgroup analysis further showed that the association between METS-IR and OSA was particularly significant in male participants (OR= 1.111; 95% CI: 1.065–1.163; P <0.001). In the ROC analysis, the area under the curve (AUC) value of METS-IR for predicting OSA was 0.777, but it is not statistically significantly different from triglyceride glucose (TyG) (AUC = 0.749; P = 0.054), body mass index (BMI) (AUC = 0.769; P = 0.269), and triglyceride glucose-body mass index (TyG-BMI) (AUC = 0.777; P = 0.996).

Conclusion: METS-IR is significantly associated with the risk of OSA and may serve as an effective predictive marker for identifying OSA.

Keywords: obstructive sleep apnea, metabolic score for insulin resistance, Insulin resistance, cross-sectional study

Introduction

Obstructive sleep apnea (OSA) is a prevalent but often overlooked chronic condition. Its pathogenesis involves recurrent upper airway collapse during sleep, leading to apnea and intermittent hypoxia.[1] This cyclical process of hypoxia and reoxygenation activates various cellular and molecular pathways, including oxidative stress, inflammation, and sympathetic nervous system activation. These mechanisms create a vicious cycle that elevates the risk of cardiovascular and metabolic diseases associated with OSA.[2,3]

Metabolic syndrome is defined as a convergent state of a group of metabolic abnormalities, including central obesity, insulin resistance (IR), hypertension, and dyslipidemia.[4] Obesity is one of the most important risk factors for OSA and can directly promote the occurrence of OSA by increasing pharyngeal fat deposition and airway resistance.[5] Due to abnormal insulin and leptin levels caused by sleep fragmentation and circadian rhythm disturbance, patients with OSA

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may develop more appetite and further aggravate obesity.[6] Body mass index (BMI) is commonly used as a measure of obesity in an individual, and studies have shown that the risk of severe OSA increases two to four times for each unit increase in BMI.[7] Abnormal lipid and glucose metabolism are the two main components of metabolic disorders, and they are often closely intertwined with the clinical outcomes associated with OSA. Studies have shown that serum high-density lipoprotein cholesterol (HDL-C) is negatively correlated with the risk of OSA, while elevated serum triglyceride (TG) and fasting blood glucose (FBG) may increase the risk of OSA.[8] Intermittent hypoxia may also induce dyslipidemia through mechanisms such as increased lipolysis, up-regulation of hepatic triglyceride lipid biosynthesis, and inhibition of lipoprotein clearance.[9] It has been reported that up to 64.3% of OSA patients have insulin resistance.[10] On one hand, OSA induces oxidative stress and inflammation through intermittent hypoxia, which damages the insulin signaling pathway and induces IR.[11] Conversely, IR may aggravate the pathological process of OSA by promoting obesity, systemic inflammation and metabolic disorders.[6,12]

In recent years, triglyceride glucose (TyG) index, as a surrogate index for evaluating IR, has been shown to have a strong correlation with OSA.[13] However, TyG mainly depends on fasting plasma glucose and triglyceride levels, which may not fully reflect obesity and the multidimensional characteristics of insulin resistance. Obesity, the most common manifestation of metabolic syndrome, holds significant importance in clinical diagnosis.[14] It serves as a crucial link between OSA and metabolic syndrome.[15] Both BMI and TyG serve as potential predictors of OSA, yet neither is without limitations.[16] Therefore, it is of great clinical significance to explore a more comprehensive and sensitive surrogate indicator of insulin resistance for the risk assessment and early screening of OSA. Metabolic score for insulin resistance (METS-IR), a novel assessment tool for insulin resistance that combines FBG, TG, BMI, and HDL-C,[17] has been shown to have good application value in predicting cardiovascular disease, hypertension, and other metabolic disorders.[18,19] This study aimed to evaluate the association between METS-IR and OSA using data from both American and Chinese populations. It further compared METS-IR with traditional predictors, such as BMI and TyG, to explore the potential value of METS-IR in OSA risk prediction.

Materials and Methods

Study Population

This study first identified the relationship between METS-IR and high risk of OSA using data from the NHANES American population. To further validate this association, we examined it in the Chinese cohort, where OSA was confirmed through sleep monitoring. This objective assessment provided a more reliable evaluation of the association between METS-IR and OSA. We utilized cross-sectional data from the NHANES (National Health and Nutrition Examination Survey) cycles 2005–2006, 2007–2008, 2015–2016, and 2017–2018, encompassing a total of 39722 American population. Participants were excluded if they met any of the following criteria: (1) missing sleep-related questionnaire data; (2) lack of information on METS-IR index factors (including fasting blood glucose, high-density lipoprotein, triglycerides, and body mass index); (3) age <20 years; and (4) missing data on other covariates such as alcohol consumption (Figure 1). Ultimately, the study included 8297 participants. Additionally, In the model using METS-IR to predict OSA, the expected model performance is 0.7. The prevalence of OSA among hospitalized patients is 80%, with a shrinkage coefficient of 0.9. Based on these parameters, the estimated minimum sample size is 246, including 197 OSA events. Finally, the study incorporated data from 581 consecutively hospitalized patients who underwent sleep monitoring at the Department of Respiratory Medicine, Renmin Hospital of Wuhan University, China, from January to June 2024. These patients are hospitalized in respiratory, cardiology and endocrinology departments for conditions such as obstructive pulmonary disease (COPD), asthma, pneumonia, hypertension, coronary heart disease (CHD) and diabetes mellitus. During the stable phase of their condition, patients underwent sleep monitoring due to complaints of snoring, daytime sleepiness, or unexplained hypertension. Inclusion criteria were as follows: availability of complete sleep monitoring data, comprehensive clinical information, and biochemical marker data. Exclusion criteria included: age <18 years, receiving treatment for OSA (continuous positive airway pressure, surgery), or using anxiolytics, antidepressants, hypnotics, or antipsychotics. The results of sleep monitoring and important clinical information were collected from the electronic medical records, including details of the patient's

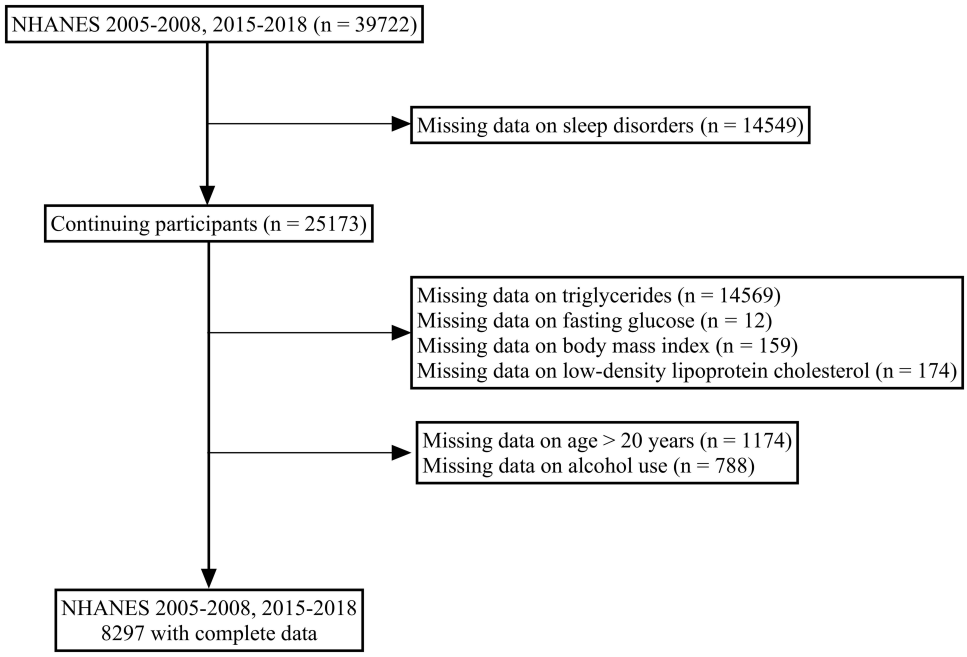

Figure 1 Flowchart of the screening process for the American population.

basic information (such as sex, age, height, weight, smoking, and alcohol consumption), medical history such as hypertension, diabetes mellitus, COPD, CHD, chronic kidney disease (CKD), and medication use (such as antihypertensive and lipid-lowering medications). The study was approved by the Ethics Committee of Renmin Hospital of Wuhan University, and informed consent was waived due to its retrospective nature.

Assessment of OSA in the NHANES

In this study, high risk of OSA in the American population was identified based on self-reported responses to specific questions related to sleep disorders in the NHANES survey.[20] Participants were classified as having a high risk of OSA if they answered "yes" to any of the following three binary questions: (1) Despite obtaining at least 7 hours of sleep on workdays, do you still experience excessive daytime sleepiness 16 to 30 times per month? (2) Do you experience breathing difficulties, snoring, or gasping for air at least three nights per week? (3) Do you snore at least three nights per week? This definition was first introduced in the NHANES 2005–2008 dataset and has since been validated and widely applied in epidemiological studies to explore the association between OSA and health outcomes.[21]

Assessment of METS-IR, TyG, TyG-BMI and Hyperlipidemia

METS-IR was calculated as ln[(2 × FBG (mg/dL) + TG (mg/dL)] × BMI (kg/m²))/(ln[HDL-C (mg/dL)]). Additionally, the TyG index was calculated as ln [TG (mg/dL) × FBG (mg/dL) /2]. BMI was calculated as weight (kg)/height (m²). TyG-BMI (triglyceride glucose-body mass index) was calculated as the TyG index × BMI. Hyperlipidemia was defined according to any of the following criteria: (1) TG ≥ 150 mg/dL, (2) total cholesterol (TC) ≥ 200 mg/dL, (3) low-density lipoprotein cholesterol (LDL-C) ≥ 130 mg/dL, or (4) HDL < 40 mg/dL for men or < 50 mg/dL for women.[22]

Assessment of Sleep Parameters and OSA in Chinese Population

Each patient underwent sleep assessment with a Zhaoguan ultra-wideband biological-radar sleep-screening (UWB, Hangzhou Megasens Technology Co., LTD., China). The noncontact monitor consisted of a radar transmitter (ZG-S01D) for tracking breathing and body movement and a finger plethysmograph (ZG-P11F) for continuous oxygen saturation recording. Our team has conducted multiple studies using this device.[23–25] To minimize interference with sleep quality, patients were instructed to avoid alcohol, caffeine, and sedatives for at least 24 hours before monitoring. On the

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night of monitoring, the operator positioned the radar transmitter approximately 1 meter away from the bedside and 0.5 meters above the mattress, ensuring it was parallel to the patient's chest and directed toward the torso. The monitoring device operated continuously throughout the overnight sleep period, with each patient was monitored for at least 7 hours. The next day, after monitoring ended, the operator downloaded the recorded data to a computer for processing, graphical visualization, and generation of a final report.[26,27] The report contains detailed sleep indicators, including total sleep time, sleep efficiency, sleep stages, lowest pulse oximetry (min SpO2), mean SpO2, percentage of sleep time with SpO2 <90% (T90), oxygen desaturation index (ODI; number of times per hour of sleep that the blood oxygen level drops by ≥4% from baseline), and apnea-hypopnea index (AHI). The severity of sleep apnea was measured using the AHI and was categorized as mild (AHI 5–14.9 events/h), moderate (AHI 15–29.9 events/h), or severe (AHI≥30 events/h) according to conventional cutoffs.

NHANES Covariates

To understand the effect of confounding factors in the multivariable adjusted model on the relationship between the METS-IR and OSA, we considered the following covariates: (1) Demographic characteristics: age, gender, race; (2) Physical measurement indicators: BMI; (3) Lifestyle factors: smoking status, drinking status; (4) Disease status: hypertension, diabetes mellitus, CHD, stroke, COPD, CKD, hyperlipidemia. All (except hyperlipidemia) were defined based on self-reported prior clinical diagnoses.

Statistical Analysis

Fasting subsample weights were used according to NCHS guidance during the analysis of the American population in this study. Continuous variables are described as medians [interquartile range (IQR)], and categorical variables as counts and percentages. The American population was divided into four groups according to METS-IR quartiles. Chinese patients were divided into four groups based on the severity of OSA or quartiles of METS-IR. Comparisons were made using the Kruskal–Wallis test or Pearson's chi-square test. A multivariate logistic regression model was constructed to explore the association between METS-IR and the risk of OSA in the American population. And multivariate logistic regression and multiple linear regression model were constructed in Chinese patients to test the correlation between METS-IR and OSA, AHI, ODI, and T90. Subgroup analyses were performed to examine variations in the association between METS-IR and the prevalence of OSA in the Chinese population. P-values for interaction were calculated to assess the interaction effect of subgroups in the association between METS-IR and OSA. Receiver operating characteristic (ROC) curve analysis was used to evaluate diagnostic value and cutoff values along with sensitivity and specificity were evaluated in the Chinese population. In addition to METS-IR, BMI, TyG, and TyG-BMI were also included for comparison with METS-IR. Prognostic power was determined by calculating the area under the curve (AUC). The DeLong test was employed to compare the AUCs of the different models to assess their relative performance. Furthermore, the cost-effectiveness of METS-IR was also calculated. All statistical analyses were performed using R software version 4.3.0 (http://www.r-project.org), and double-sided $P < 0.05$ was defined as statistical significance.

Results

Baseline Characteristics of the American Population

Table 1 presents the weighted demographic and clinical characteristics of the subjects according to the quartiles of the METS-IR. The study included a total of 8,297 participants, with 51.6% men and 48.4% women. The median age was 47 years, and the prevalence of OSA was 49.8%. Higher METS-IR quartiles were associated with a greater proportion of individuals who smoked, had diabetes mellitus, hypertension, CHD, COPD, hyperlipidemia, or OSA ($P < 0.001$).

Relationship Between METS-IR and Higher Risk of OSA in the American Population

To better explore the relationship between the METS-IR and the higher risk of OSA, we analyzed three logistic regression models. In the unadjusted model, no variables were adjusted, and the results showed that METS-IR was positively correlated with OSA risk (OR= 1.046; 95% CI: 1.039–1.053; $P < 0.001$). Similarly, in Model I, the association between METS-IR and OSA

Table 1 Characteristics of the American Population Across METS-IR Quartiles

Characteristic	Overall N = 8297[a]	Q1, N = 2075[a]	Q2, N = 2074[a]	Q3, N = 2074[a]	Q4, N = 2074[a]	P value[b]
OSA group						<0.001
Non-OSA	4084 (50.2%)	1,357 (67.9%)	1,090 (52.4%)	928 (45.9%)	709 (32.5%)	
OSA	4213 (49.8%)	718 (32.1%)	984 (47.6%)	1,146 (54.1%)	1,365 (67.5%)	
Age, years	47 (33, 60)	42 (28, 58)	48.0 (35, 63)	49 (35, 61)	47 (35, 60)	<0.001
Gender						<0.001
Female	4203 (51.6%)	1,226 (63.4%)	972 (46.9%)	957 (45.2%)	1,048 (49.1%)	
Male	4094 (48.4%)	849 (36.6%)	1,102 (53.1%)	1,117 (54.8%)	1,026 (50.9%)	
BMI, kg/m^2	27.87 (24.13, 32.70)	22.40 (20.60, 23.80)	26.50 (25.10, 27.84)	30.10 (28.40, 32.00)	36.80 (34.00, 41.10)	<0.001
Ethnicity, n (%)						<0.001
Mexican American	1395 (8.5%)	225 (5.5%)	378 (8.9%)	406 (10.0%)	386 (9.9%)	
Non-Hispanic Black	1743 (10.8%)	421 (9.4%)	414 (11.0%)	420 (10.6%)	488 (12.5%)	
Non-Hispanic White	3572 (68.0%)	979 (71.2%)	885 (68.0%)	850 (66.1%)	858 (66.2%)	
Other Hispanic	797 (5.3%)	159 (4.5%)	190 (4.7%)	232 (6.8%)	216 (5.5%)	
Other Race	790 (7.4%)	291 (9.4%)	207 (7.5%)	166 (6.5%)	126 (5.8%)	
Drinking status, n (%)						<0.001
Nondrinkers	2971 (30.3%)	668 (27.4%)	688 (26.8%)	788 (32.0%)	827 (35.2%)	
Drinkers	5326 (69.7%)	1,407 (72.6%)	1,386 (73.2%)	1,286 (68.0%)	1,247 (64.8%)	
Smoking status, n (%)						0.102
Nonsmokers	4377 (52.8%)	1,120 (55.0%)	1,106 (53.2%)	1,114 (52.6%)	1,037 (50.1%)	
Smokers	3920 (47.2%)	955 (45.0%)	968 (46.8%)	960 (47.4%)	1,037 (49.9%)	
Diabetes mellitus, n (%)	1327 (11.3%)	97 (2.8%)	245 (7.5%)	377 (12.5%)	608 (23.3%)	<0.001
Hypertension, n (%)	3010 (31.8%)	443 (17.1%)	716 (30.6%)	802 (35.4%)	1,049 (45.9%)	<0.001
CHD, n (%)	365 (3.7%)	48 (2.1%)	96 (4.0%)	100 (4.2%)	121 (4.6%)	0.004
Stroke, n (%)	330 (2.9%)	55 (2.0%)	83 (3.1%)	66 (2.0%)	126 (4.6%)	<0.001
COPD, n (%)	536 (6.3%)	104 (4.5%)	101 (5.5%)	135 (6.2%)	196 (9.2%)	<0.001
CKD, n (%)	276 (2.3%)	52 (1.7%)	69 (2.1%)	64 (2.1%)	91 (3.5%)	0.001
Hyperlipidemia, n (%)	5312 (63.2%)	921 (43.0%)	1,253 (60.5%)	1,477 (71.3%)	1,661 (80.6%)	<0.001
METS-IR	41 (34, 50)	30 (27, 32)	38 (36, 40)	45 (43, 47)	58 (53, 64)	<0.001
TyG	8.55 (8.12, 8.98)	8.09 (7.79, 8.44)	8.51 (8.15, 8.83)	8.74 (8.37, 9.11)	8.98 (8.58, 9.37)	<0.001
TyG-BMI	241 (200, 288)	181 (166, 194)	224 (214, 235)	263 (250, 277)	329 (304, 367)	<0.001

Notes: [a] n (unweighted) (%); Median (IQR). [b] chi-squared test with Rao & Scott's second-order correction; Wilcoxon rank-sum test for complex survey samples.
Abbreviations: OSA, obstructive sleep apnea; BMI, body mass index; CHD, coronary heart disease; COPD, chronic obstructive pulmonary disease; CKD, chronic kidney disease; METS-IR, metabolic score for insulin resistance; TyG, triglyceride glucose index; TyG-BMI, triglyceride glucose-body mass index.

remained significant after accounting for age, sex, and race (OR= 1.044; 95% CI: 1.037–1.050; P < 0.001). In the fully adjusted Model II, the risk of OSA increased significantly by 4.4% for each unit increase in the METS-IR. Compared with the lowest quartile group, higher risk of OSA in the fourth quartile group increased by 302.8% (OR= 4.028; 95% CI: 3.261–4.975; P < 0.001) (Table 2).

Baseline Characteristics of the Chinese Population

A total of 581 patients who underwent sleep monitoring in Renmin Hospital of Wuhan University were included in this study. The median age of the patients was 60 years, with 71.4% being male and 28.6% female. The prevalence of OSA among these patients was 87.3%. Compared to non-OSA patients, those with OSA exhibited significantly higher TG and BMI, significantly HDL, and a significantly higher prevalence of hypertension and CHD. Notably, METS-IR was significantly elevated in OSA patients and increased with the severity of OSA (P<0.001) (Table 3).

Characteristics of the Chinese Population Stratified by METS-IR Quartiles

Patients were divided into four groups according to METS-IR quartile. Significant differences were observed in age, gender, AHI, ODI, T90, FBG, TG, HDL, smoking, drinking, OSA, hypertension, diabetes mellitus, COPD, CKD, hyperlipidemia, and the use of antihypertensive, lipid-lowering, and antidiabetic medications (P<0.05). As METS-IR increased, the age of patients decreased, and the proportions of men and alcohol drinkers rose. The

Table 2 Multivariate Regression Analysis of METS-IR and OSA in the American Population

Variables	Crude Model		Model I		Model II	
	Crude OR (95% CI)	P value	Adjusted OR (95% CI)	P value	Adjusted OR (95% CI)	P value
METS-IR	1.046 (1.039, 1.053)	<0.001	1.044 (1.037, 1.050)	<0.001	1.044 (1.037, 1.050)	<0.001
Q1	Reference		Reference		Reference	
Q2	1.918 (1.619, 2.272)	<0.001	1.730 (1.461, 2.048)	<0.001	1.727 (1.451, 2.056)	<0.001
Q3	2.491 (2.045, 3.036)	<0.001	2.240 (1.842, 2.725)	<0.001	2.240 (1.855, 2.704)	<0.001
Q4	4.388 (3.569, 5.394)	<0.001	4.086 (3.335, 5.006)	<0.001	4.028 (3.261, 4.975)	<0.001
P for trend	<0.001		<0.001		<0.001	

Notes: The crude model was not adjusted for covariates. Model I was adjusted for age, gender and race. Model II was adjusted for all covariates. N = 8297.
Abbreviations: OR, odds ratio; CI, confidence interval; METS-IR, metabolic score for insulin resistance; OSA, obstructive sleep apnea.

Table 3 Characteristics of the Chinese Population by Different OSA Severity Levels

Characteristic	Overall N = 581[a]	Non-OSA N = 74[a]	Mild N = 146[a]	Moderate N = 142[a]	Severe N = 219[a]	P value[b]
Age, years	60 (48, 69)	61.5 (53, 68)	56 (44, 68)	60 (53, 70)	60 (46, 69)	0.093
Gender						<0.001
Female	166 (28.6%)	28 (37.8%)	53 (36.3%)	49 (34.5%)	36 (16.4%)	
Male	415 (71.4%)	46 (62.2%)	93 (63.7%)	93 (65.5%)	183 (83.6%)	
AHI, events/h	22.0 (9.3, 39.4)	2.5 (1.3, 3.9)	9.5 (7.1, 11.7)	22.0 (18.0, 26.3)	46.2 (37.2, 59.2)	<0.001
ODI, events/h	22.4 (10.7, 37.3)	4.6 (2.2, 7.1)	11.6 (7.5, 18.8)	21.6 (17.0, 27.1)	40.5 (27.7, 56.5)	<0.001
T90, %	4.9 (1.2, 13.2)	0.2 (0.0, 0.7)	1.8 (0.4, 5.7)	5.4 (2.3, 9.2)	13.3 (6.3, 27.5)	<0.001
FBG, mg/dL	88.74 (79.92, 104.04)	84.60 (76.32, 101.16)	86.67 (79.02, 101.70)	89.01 (81.36, 106.20)	91.08 (81.00, 105.30)	0.051
TC, mg/dL	167.44 (143.85, 193.74)	171.31 (149.27, 197.60)	171.89 (151.59, 198.38)	164.93 (143.47, 192.19)	163.57 (138.44, 189.48)	0.184
TG, mg/dL	114.26 (80.60, 172.71)	89.46 (62.88, 128.43)	115.14 (80.60, 164.74)	115.14 (83.26, 200.17)	126.66 (86.80, 184.23)	<0.001
HDL, mg/dL	37.51 (31.32, 45.24)	40.60 (35.58, 50.27)	39.44 (32.87, 44.86)	38.09 (31.71, 46.02)	33.64 (29.00, 42.15)	<0.001
LDL, mg/dL	97.06 (76.18, 117.17)	98.03 (81.21, 117.17)	98.80 (82.37, 122.97)	96.48 (75.02, 110.98)	94.35 (72.31, 114.85)	0.136
TyG	8.57 (8.18, 9.02)	8.24 (7.97, 8.76)	8.52 (8.22, 8.96)	8.60 (8.19, 9.14)	8.70 (8.26, 9.11)	<0.001
BMI, kg/m^2	25.40 (23.01, 28.73)	23.46 (21.26, 25.53)	24.56 (21.77, 27.05)	25.29 (23.14, 28.20)	27.34 (24.22, 30.39)	<0.001
TyG-BMI	221.12 (189.72, 257.26)	194.97 (171.75, 220.59)	208.42 (184.07, 241.52)	221.29 (192.32, 256.45)	241.25 (203.42, 269.11)	<0.001
METS-IR	40.75 (34.66, 47.55)	35.90 (30.88, 39.92)	38.31 (33.65, 44.24)	40.86 (34.97, 47.27)	45.58 (38.38, 51.45)	<0.001
Smoking status, n (%)	160 (27.5%)	13 (17.6%)	37 (25.3%)	35 (24.6%)	75 (34.2%)	0.023
Drinking status, n (%)	125 (21.5%)	10 (13.5%)	28 (19.2%)	33 (23.2%)	54 (24.7%)	0.186
Hypertension, n (%)	316 (54.4%)	26 (35.1%)	67 (45.9%)	87 (61.3%)	136 (62.1%)	<0.001
Diabetes mellitus, n (%)	111 (19.1%)	11 (14.9%)	24 (16.4%)	32 (22.5%)	44 (20.1%)	0.432
CHD, n (%)	140 (24.1%)	10 (13.5%)	26 (17.8%)	32 (22.5%)	72 (32.9%)	0.001
Cerebrovascular disease, n (%)	36 (6.2%)	1 (1.4%)	10 (6.8%)	8 (5.6%)	17 (7.8%)	0.228
COPD, n (%)	119 (20.5%)	26 (35.1%)	28 (19.2%)	26 (18.3%)	39 (17.8%)	0.010
CKD, n (%)	37 (6.4%)	1 (1.4%)	3 (2.1%)	9 (6.3%)	24 (11.0%)	0.001
Hyperlipidemia, n (%)	464 (79.9%)	55 (74.3%)	111 (76.0%)	115 (81.0%)	183 (83.6%)	0.193
Antihypertensive agents, n (%)	293 (50.4%)	22 (29.7%)	64 (43.8%)	79 (55.6%)	128 (58.4%)	<0.001
Antidiabetic drug, n (%)	104 (17.9%)	11 (14.9%)	21 (14.4%)	29 (20.4%)	43 (19.6%)	0.431
Lipid-lowering drug, n (%)	146 (25.1%)	12 (16.2%)	31 (21.2%)	30 (21.1%)	73 (33.3%)	0.004
Antiplatelet drug, n (%)	99 (17.0%)	7 (9.5%)	17 (11.6%)	25 (17.6%)	50 (22.8%)	0.010

Notes: [a] Median (Q1, Q3); n (%). [b] Kruskal–Wallis rank sum test; Pearson's Chi-squared test; Fisher's exact test.
Abbreviations: OSA, obstructive sleep apnea; AHI, apnea hypopnea index; ODI, oxygen desaturation index; T90, the percentage of time with oxygen saturation less than 90%; FBG, fasting blood glucose; TC, total cholesterol; TG, triglyceride; HDL, high density lipoprotein-cholesterol; LDL, low density lipoprotein-cholesterol; TyG, triglyceride glucose index; BMI, body mass index; TyG-BMI, triglyceride glucose-body mass index; METS-IR, metabolic score for insulin resistance; CHD, coronary heart disease; COPD, chronic obstructive pulmonary disease; CKD, chronic kidney disease.

prevalence of OSA, hypertension, diabetes mellitus, and hyperlipidemia also increased. Additionally, patients with higher METS-IR exhibited worse sleep parameters and more common use of antihypertensive, lipid-lowering, and antidiabetic drugs (Table 4).

Table 4 Characteristics of the Chinese Population Across METS-IR Quartiles

Characteristics	OverallN = 581[a]	Q1 N = 146[a]	Q2 N = 145[a]	Q3 N = 145[a]	Q4 N = 145[a]	P value[b]
Age, years	60 (48, 69)	68 (60, 73)	61 (53, 69)	58 (49, 66)	48 (37, 60)	<0.001
Gender, n (%)						0.001
Female	166 (28.6%)	53 (36.3%)	50 (34.5%)	37 (25.5%)	26 (17.9%)	
Male	415 (71.4%)	93 (63.7%)	95 (65.5%)	108 (74.5%)	119 (82.1%)	
OSA, n (%)	507 (87.3%)	115 (78.8%)	118 (81.4%)	132 (91.0%)	142 (97.9%)	<0.001
AHI, events/h	22.0 (9.3, 39.4)	12.5 (5.7, 27.6)	16.0 (6.8, 31.8)	25.5 (11.9, 37.3)	40.1 (19.1, 58.0)	<0.001
ODI, events/h	22.4 (10.7, 37.3)	15.5 (6.9, 24.1)	18.0 (8.8, 29.4)	23.7 (13.3, 33.6)	37.5 (20.1, 56.6)	<0.001
T90, %	4.9 (1.2, 13.2)	2.0 (0.4, 7.0)	4.7 (0.7, 9.8)	4.7 (1.8, 12.2)	12.0 (4.0, 26.8)	<0.001
FBG, mg/dL	88.74 (79.92, 104.04)	83.70 (75.78, 95.94)	86.04 (79.38, 97.56)	92.70 (83.16, 107.10)	96.30 (82.98, 114.30)	<0.001
TC, mg/dL	167.44 (143.85, 193.74)	165.31 (143.47, 189.10)	167.05 (143.08, 187.55)	164.73 (144.24, 194.51)	175.56 (143.85, 202.24)	0.236
TG, mg/dL	114.26 (80.60, 172.71)	73.51 (59.34, 97.43)	100.97 (77.94, 133.74)	139.94 (97.43, 200.17)	181.57 (126.66, 246.22)	<0.001
HDL, mg/dL	37.51 (31.32, 45.24)	47.18 (39.06, 56.07)	39.06 (34.03, 45.63)	36.35 (31.32, 41.38)	31.32 (27.84, 34.80)	<0.001
LDL, mg/dL	97.06 (76.18, 117.17)	94.74 (72.31, 111.76)	100.54 (79.66, 117.17)	97.84 (80.05, 116.01)	95.90 (72.31, 122.97)	0.447
TyG	8.57 (8.18, 9.02)	8.09 (7.76, 8.42)	8.39 (8.15, 8.73)	8.83 (8.50, 9.11)	9.11 (8.71, 9.56)	<0.001
BMI, kg/m^2	25.40 (23.01, 28.73)	21.05 (19.13, 22.77)	24.44 (23.44, 25.39)	26.99 (25.71, 28.44)	30.86 (29.05, 33.22)	<0.001
TyG-BMI	221.12 (189.72, 257.26)	172.17 (155.07, 186.33)	206.96 (196.71, 216.28)	238.20 (225.99, 252.06)	281.45 (263.92, 307.75)	<0.001
METS-IR	40.75 (34.66, 47.55)	31.19 (27.65, 33.47)	37.75 (36.30, 39.48)	44.18 (42.45, 45.96)	54.00 (50.13, 58.22)	<0.001
Smoking status, n (%)	160 (27.5%)	37 (25.3%)	28 (19.3%)	37 (25.5%)	58 (40.0%)	<0.001
Drinking status, n (%)	125 (21.5%)	20 (13.7%)	30 (20.7%)	36 (24.8%)	39 (26.9%)	0.033
Hypertension, n (%)	316 (54.4%)	62 (42.5%)	68 (46.9%)	89 (61.4%)	97 (66.9%)	<0.001
Diabetes mellitus, n (%)	111 (19.1%)	15 (10.3%)	21 (14.5%)	36 (24.8%)	39 (26.9%)	<0.001
CHD, n (%)	140 (24.1%)	26 (17.8%)	36 (24.8%)	36 (24.8%)	42 (29.0%)	0.163
Cerebrovascular disease, n (%)	36 (6.2%)	8 (5.5%)	10 (6.9%)	10 (6.9%)	8 (5.5%)	0.921
COPD, n (%)	119 (20.5%)	54 (37.0%)	35 (24.1%)	20 (13.8%)	10 (6.9%)	<0.001
CKD, n (%)	37 (6.4%)	3 (2.1%)	9 (6.2%)	8 (5.5%)	17 (11.7%)	0.008
Hyperlipidemia, n (%)	464 (79.9%)	80 (54.8%)	114 (78.6%)	129 (89.0%)	141 (97.2%)	<0.001
Antihypertensive agents, n (%)	293 (50.4%)	56 (38.4%)	66 (45.5%)	82 (56.6%)	89 (61.4%)	<0.001
Antidiabetic drug, n (%)	104 (17.9%)	13 (8.9%)	21 (14.5%)	34 (23.4%)	36 (24.8%)	0.001
Lipid-lowering drug, n (%)	146 (25.1%)	18 (12.3%)	33 (22.8%)	44 (30.3%)	51 (35.2%)	<0.001
Antiplatelet drug, n (%)	99 (17.0%)	14 (9.6%)	25 (17.2%)	30 (20.7%)	30 (20.7%)	0.037

Notes: [a]Median (Q1, Q3); n (%). [b]Kruskal–Wallis rank sum test; Pearson's Chi-squared test.

Abbreviations: OSA, obstructive sleep apnea; AHI, apnea hypopnea index; ODI, oxygen desaturation index; T90, the percentage of time with oxygen saturation less than 90%; FBG, fasting blood glucose; TC, total cholesterol; TG, triglyceride; HDL, high density lipoprotein-cholesterol; LDL, low density lipoprotein-cholesterol; TyG, triglyceride glucose index; BMI, body mass index; TyG-BMI, triglyceride glucose-body mass index; METS-IR, metabolic score for insulin resistance; CHD, coronary heart disease; COPD, chronic obstructive pulmonary disease; CKD, chronic kidney disease.

Association Between METS-IR and OSA Prevalence, AHI, ODI, and T90 in the Chinese Population

After adjusting for all confounding factors, models revealed a significant positive correlation between METS-IR and OSA, AHI, ODI, and T90. For each 1-unit increase in METS-IR, the prevalence of OSA increased by 6.7% (OR= 1.067; 95% CI: 1.035–1.103), AHI increased by 0.732 (β= 0.732; 95% CI: 0.573–0.732), ODI increased by 0.777 (β= 0.777; 95% CI: 0.614–0.940), and T90 increased by 0.389 (β= 0.389; 95% CI: 0.285–0.492). Compared with the Q1 group, AHI, ODI, and T90 were higher in the Q3 and Q4 groups, with the prevalence of OSA increasing by 109.5% and 877.4%, respectively. As the METS-IR quartile increased, the probability of OSA, as well as AHI, ODI, and T90, rose significantly (P<0.001) (Table 5).

Subgroup Analysis of the Chinese Population

Subgroup analyses were conducted to determine if the relationship between METS-IR and OSA prevalence remained consistent across various demographic groups. The positive correlation between METS-IR and OSA prevalence was observed in all groups except for women, individuals with a BMI ≥ 28 kg/m², and those without hyperlipidemia, hypertension, coronary heart disease, COPD, or those not taking antiplatelet drugs. Additionally, gender significantly influenced the relationship between METS-IR and OSA (P for interaction = 0.047) (Figure 2).

Table 5 Association of METS-IR with OSA, AHI, ODI and T90 in the Chinese Population

Characteristics	OSA		AHI		ODI		T90	
	OR (95% CI)	P value	β (95% CI)	P value	β (95% CI)	P value	β (95% CI)	P value
METS-IR METS-IR quartile	1.067 (1.035, 1.103)	<0.001	0.732 (0.573, 0.890)	<0.001	0.777 (0.614, 0.940)	<0.001	0.389 (0.285, 0.492)	<0.001
Q1	Reference		Reference		Reference		Reference	
Q2	1.032 (0.571, 1.869)	0.917	3.412 (−0.734, 7.567)	0.107	4.595 (0.294, 8.896)	0.037	2.170 (−0.543, 4.882)	0.118
Q3	2.095 (1.033, 4.421)	0.045	8.170 (3.840, 12.500)	<0.001	8.062 (3.574, 12.549)	<0.001	2.998 (0.167, 5.823)	0.038
Q4	9.774 (3.124, 41.729)	<0.001	20.643 (15.856, 25.430)	<0.001	21.383 (16.422, 26.344)	<0.001	10.966 (7.837, 14.095)	<0.001
P for trend	<0.001		<0.001		<0.001		<0.001	

Notes: The results were adjusted for age, gender, smoking status, drinking status, hypertension, diabetes mellitus, CHD, cerebrovascular disease, COPD, CKD, antihypertensive agents, antidiabetic drug, lipid-lowering drug, antiplatelet drug. N = 581.

Abbreviations: OSA, obstructive sleep apnea; AHI, apnea hypopnea index; ODI, oxygen desaturation index; T90, the percentage of time with oxygen saturation less than 90%; METS-IR, metabolic score for insulin resistance; OR, odds ratio; CI, confidence interval.

ROC Curve Analysis of the Chinese Population

The ROC curve analysis demonstrated the diagnostic effectiveness of BMI, TyG, TyG-BMI, and METS-IR for OSA. In the unadjusted model, the AUC for METS-IR was 0.703, which was higher than that for TyG-BMI (AUC: 0.697), BMI (AUC: 0.668) and TyG (AUC: 0.649) (Figure 3A). After adjusting for gender, age, smoking and drinking status, COPD, hypertension, diabetes mellitus, CKD, CHD, cerebrovascular disease, and the use of antihypertensive, antidiabetic, lipid-lowering, and antiplatelet drugs, TyG-BMI and METS-IR showed superior diagnostic performance, each with an AUC of 0.777, compared to BMI and TyG alone. TyG had the lowest diagnostic performance (AUC: 0.749), followed by BMI (AUC: 0.769). Furthermore, TyG-BMI was significantly more effective in diagnosing OSA than TyG (P = 0.044) (Figure 3B). The optimal cutoff points for BMI, TyG, TyG-BMI, and METS-IR were 25.34, 8.19, 225.20, and 41.97 respectively. Using these cutoff points, the obtained sensitivity values were 0.548, 0.771, 0.517, and 0.503, while the specificity values were 0.743, 0.486, 0.838, and 0.838.

Cost-Effectiveness Analysis of METS-IR in Evaluating OSA in the Chinese Population

Next, the cost-effectiveness of METS-IR and UWB in the diagnosis of OSA was evaluated. According to prior research, UWB demonstrated a sensitivity of 100% and a specificity of 70% for OSA diagnosis.[26] The respective costs per test for UWB, METS-IR, TyG, and TyG-BMI were 150, 20, 11, and 11 yuan. The average cost-effectiveness ratios (ACERs) for these tests were 171.8, 45.5, 16.3, and 24.4 yuan. When comparing METS-IR to UWB, TyG, and TyG-BMI, the incremental cost-effectiveness ratios (ICERs) were 299.6 yuan, −38.5 yuan, and −736.4 yuan, respectively.

Discussion

This study represents a preliminary investigation into the association between METS-IR and OSA risk in Chinese and American populations. The results indicate that METS-IR is associated with an increased risk of OSA after adjusting for confounding factors. Specifically, the risk of OSA significantly increases with higher METS-IR levels. Therefore, METS-IR is likely to be an important predictor of OSA.

Obesity, particularly visceral obesity, is widely recognized as a core driver of metabolic abnormalities.[28] Excess adipose tissue disrupts metabolic homeostasis by altering adipokine secretion and activating pro-inflammatory pathways.[29] Studies have shown that visceral obesity and neck circumference are associated with the occurrence of OSA, and that neck circumference has an independent correlation with both metabolic syndrome and OSA.[30] When obesity and OSA coexist, they have potential synergistic effects. Obesity increases the effort required to maintain airway patency, especially during REM sleep, leading to OSA. Furthermore, long-term obesity can cause related conditions such as hypertension, insulin resistance, and dyslipidemia, which can exacerbate OSA. Conversely, OSA impairs insulin

Variables	No.	OR	lower 95%CI	upper 95%CI	pvalue	P for interaction
Gender						0.047
Male	415	1.111	1.065	1.163	<.001	
Female	166	1.023	0.978	1.076	0.338	
Age						0.084
< 60 years	277	1.112	1.052	1.184	<.001	
≥ 60 years	304	1.050	1.004	1.102	0.042	
Drinking status						0.268
Nondrinkers	456	1.060	1.027	1.098	<.001	
Drinkers	125	1.202	1.058	1.407	0.012	
Smoking status						0.524
Nonsmokers	421	1.062	1.027	1.101	<.001	
Smokers	160	1.115	1.037	1.214	0.008	
Diabetes mellitus						0.132
No	470	1.055	1.021	1.092	0.002	
Yes	111	1.133	1.023	1.255	0.015	
Hypertension						0.946
No	265	1.067	1.024	1.117	0.004	
Yes	316	1.051	0.999	1.111	0.065	
CHD						0.553
No	441	1.063	1.029	1.102	<.001	
Yes	140	1.069	0.980	1.165	0.128	
COPD						0.190
No	462	1.083	1.043	1.129	<.001	
Yes	119	1.038	0.972	1.114	0.286	
Antihypertensive agents						0.312
No	288	1.057	1.018	1.101	0.007	
Yes	293	1.074	1.012	1.145	0.023	
Antidiabetic drug						0.112
No	477	1.055	1.021	1.093	0.002	
Yes	104	1.134	1.020	1.260	0.018	
Lipid−lowering drug						0.345
No	435	1.063	1.026	1.103	0.001	
Yes	146	1.081	1.006	1.177	0.053	
Antiplatelet drug						0.728
No	482	1.067	1.033	1.106	<.001	
Yes	99	1.082	0.951	1.256	0.260	
BMI						NA
< 28 kg/m2	415	1.056	1.010	1.106	0.019	
≥ 28 kg/m2	166	1.095	0.970	1.288	0.204	
Hyperlipidemia						NA
No	117	1.047	0.974	1.149	0.283	
Yes	464	1.088	1.047	1.134	<.001	

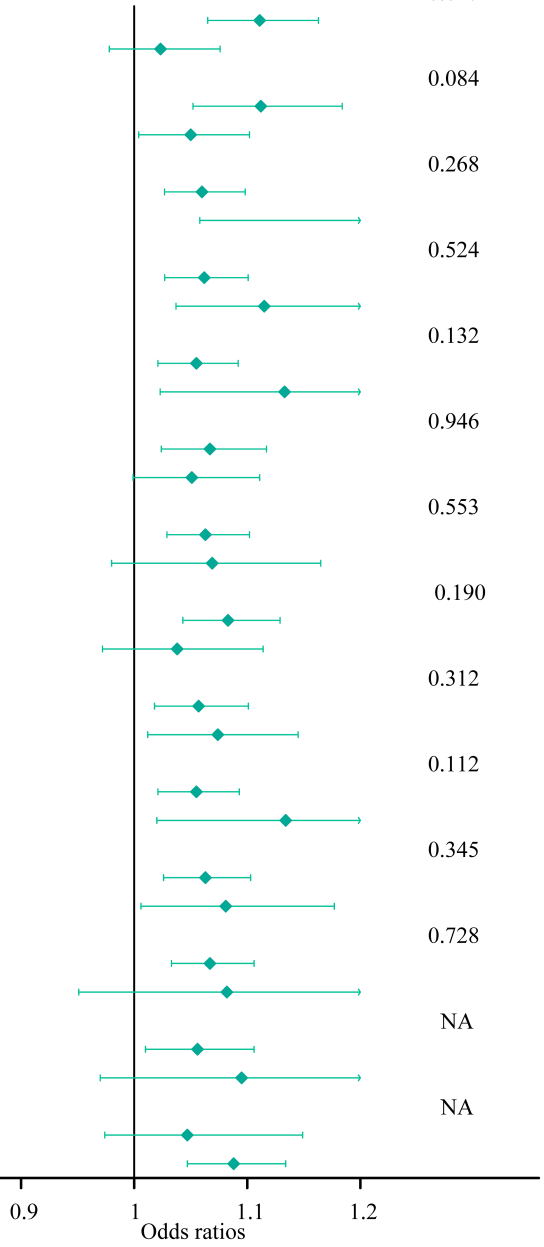

Figure 2 Subgroup analysis of the Chinese population.

Abbreviations: OSA, obstructive sleep apnea; AHI, apnea hypopnea index; ODI, oxygen desaturation index; T90, the percentage of time with oxygen saturation less than 90%; FBG, fasting blood glucose; TC, total cholesterol; TG, triglyceride; HDL, high density lipoprotein-cholesterol; LDL, low density lipoprotein-cholesterol; EGFR, estimated glomerular filtration rate; TyG, triglyceride glucose index; BMI, body mass index; TyG-BMI, triglyceride glucose-body mass index; METS-IR, metabolic score for insulin resistance; CHD, coronary heart disease; COPD, chronic obstructive pulmonary disease; CKD, chronic kidney disease.

sensitivity and increases inflammation, leading to obesity and poor glycemic control.[31] Therefore, given obesity's critical role in linking metabolic disorders and OSA, weight management is of paramount importance.

IR is physiologically described as a diminished sensitivity and reaction of target tissues to insulin. This condition significantly impacts various adverse clinical outcomes, including metabolic syndrome, non-alcoholic fatty liver disease, atherosclerosis, and type 2 diabetes mellitus (T2DM). The underlying mechanisms of IR are generally believed to include ectopic lipid accumulation, endoplasmic reticulum (ER) stress, and inflammation in the liver and skeletal muscle.[32] A randomized crossover trial found that chronic intermittent hypoxia (CIH) induces high sympathetic tone, lipolysis, and

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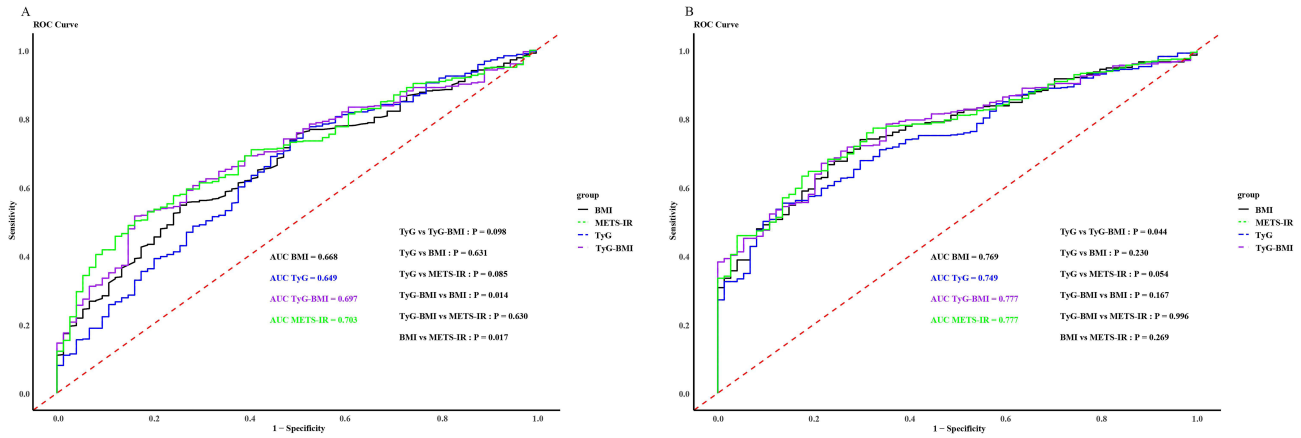

Figure 3 Receiver-operating characteristic curve of BMI, TyG, TyG-BMI and METS-IR for predicting OSA in the Chinese population. (**A**) Unadjusted model. (**B**) The model was adjusted for gender, age, smoking status, drinking status, COPD, hypertension, diabetes mellitus, CKD, CHD, cerebrovascular disease, antihypertensive agents, antidiabetic drug, lipid-lowering drug, antiplatelet drug.
Abbreviations: ROC, receiver operating characteristic; AUC, the area under the curve; TyG, triglyceride glucose index; BMI, body mass index; TyG-BMI, triglyceride.

reduced sensitivity of free fatty acids to insulin in healthy individuals. This may contribute to the development of systemic insulin resistance and diabetes in OSA patients. Moreover, IR can regulate CIH-induced lipolysis.[33] Some scholars have proposed that systemic "lipotoxicity" caused by excessive lipolysis during sleep in OSA is the root cause of metabolic dysfunction.[34] Recent studies have shown increased intramyocellular and extramyocellular lipids in OSA patients, particularly in non-obese individuals. This is related to inappropriate lipolysis triggered by OSA-related intermittent hypoxia, which in turn triggers a free fatty acid storm. Treatment with continuous positive airway pressure significantly improves these characteristics.[35]

Insulin-based measures of IR are often limited by the need for invasive procedures. In contrast, non-insulin-based measures, including the TyG index, TyG-BMI index, and METS-IR, offer advantages such as cost-effectiveness and simplified acquisition techniques. Additionally, these measures have a stronger association with the gold standard protocol for measuring insulin resistance.[36] Recent studies have demonstrated that METS-IR is positively associated with the risk of various diseases, including lung cancer, heart failure, T2DM, and neurological impairment.[37–40] METS-IR is emerging as a potential tool for assessing disease risk. Similarly, TyG related indices have shown remarkable predictive power. With each 1-unit rise in TyG-BMI, the likelihood of OSA rose by 54%.[41] Additionally, an increase in the TyG-waist circumference (TyG-WC) is linked to a 4.29-fold increase in the risk of the first myocardial infarction in OSA patients within the highest quartile of TyG-WC.[42] Furthermore, some studies have employed machine learning methods to differentiate between OSA and non-OSA individuals, with results indicating that the TyG index is the most reliable predictor compared to BMI.[16]

This study evaluated the predictive value of METS-IR against BMI and TyG-related measures for assessing OSA risk. The AUC in ROC analysis was as follows: METS-IR = TyG-BMI > BMI > TyG. Although the improvement in AUC for METS-IR relative to BMI was modest, the added value of METS-IR may be particularly beneficial in clinical settings where a more nuanced assessment of metabolic dysfunction is required to identify patients at risk for OSA, particularly those who have not yet shown overt symptoms but have underlying metabolic abnormalities. Additionally, Gender was identified as the only variable with an interaction effect between METS-IR and OSA. This may be related to sex differences in fat distribution and propensity for lipid metabolism. Studies have shown that men generally accumulate more visceral fat in the abdomen, while women accumulate more subcutaneous fat in the hips and thighs.[43] Compared to subcutaneous fat, visceral fat is easier to mobilize and dissolve, and is more sensitive to the anti-lipolytic effect of insulin.[44] Moreover, men are more likely to have high levels of very-low-density lipoprotein lipids, particularly triglycerides, whereas women typically have higher HDL levels.[45] As women enter menopause, their estrogen levels decrease, increasing the risk of lipid metabolic disorders, which is manifested by the accumulation of visceral fat and aggravation of insulin resistance. The fact that women have less abdominal fat accumulation may contribute to their

lower risks for cardiovascular disease, metabolic syndrome, and diabetes.[43,46] More caution is needed when applying this indicator to predict the risk of OSA in females. In the cost-effectiveness analysis, METS-IR proved to be more cost-effective compared to UWB, with a lower cost per identified true positive case. TyG-BMI demonstrated the highest cost-effectiveness, offering greater benefits at a lower cost. Moreover, TyG-BMI showed a clear advantage over both METS-IR and TyG in terms of cost-effectiveness.

This study has several strengths. Firstly, it is the first to assess the link between METS-IR and OSA risk, highlighting METS-IR as a potential predictive marker for OSA. Secondly, OSA diagnosis in the Chinese population was based on actual sleep monitoring using the AHI, rather than questionnaires or subjective reports. Subgroup analysis was used to enhance the validity and reliability of our findings. Nevertheless, this study has several limitations that warrant consideration. The cross-sectional design precludes the establishment of causal relationships between METS-IR and OSA. While a significant association was observed, it remains unclear whether elevated METS-IR contributes to the development of OSA or whether OSA may influence METS-IR levels. Large-scale epidemiological studies like NHANES rely on questionnaires, as sleep monitoring is impractical for thousands of participants. This approach may introduce misclassification bias, potentially failing to identify mild or asymptomatic OSA cases. Another important limitation lies in the hospital-based recruitment and single-center nature of this study. Our Chinese cohort comprised hospitalized patients with suspected OSA, which may limit the generalizability of the findings to asymptomatic individuals or those with mild OSA typically managed in primary care. Additionally, as the study was conducted at a single institution, the results may not apply to populations with different demographic, clinical, or environmental characteristics. Our study did not systematically analyze night/day symptom patterns. Future research should combine METS-IR with symptom scales to explore how metabolic features interact with specific OSA phenotypes. Incorporating METS-IR as a routine screening tool for patients at risk for OSA, especially those with metabolic disorders, is essential. Early identification of high-risk individuals allows for targeted interventions, such as lifestyle modifications and continuous positive airway pressure therapy, to reduce OSA incidence and severity. For instance, non-obese patients with metabolic dysregulation (elevated TG, low HDL-C) could be flagged for OSA screening even if their BMI is normal, addressing the limitations of BMI-centric approaches. As a simple, non-invasive, and readily calculable index (requiring routine laboratory tests), METS-IR could be integrated into primary care settings to prioritize high-risk patients for costly and time-consuming sleep studies like polysomnography. This is particularly valuable in resource-limited regions where OSA remains underdiagnosed. METS-IR's ability to quantify metabolic dysfunction in OSA patients may help subclassify OSA phenotypes, such as "metabolically unhealthy OSA" vs "metabolically healthy OSA", enabling precision medicine strategies. Additionally, given the association between METS-IR and OSA, clinicians should consider incorporating metabolic health assessments into the evaluation and management of OSA patients. By combining METS-IR with traditional OSA screening tools (such as the STOP-Bang Questionnaire and NoSAS Score), the sensitivity and specificity of screenings can be enhanced. This approach can aid in the early identification of individuals with metabolic abnormalities who may not yet exhibit obvious OSA symptoms, potentially improving health outcomes and reducing the risk of long-term complications.

Conclusions

The results suggest that an elevated METS-IR correlates with a heightened risk of OSA. Early identification and intervention for individuals with higher METS-IR may help prevent the onset of OSA and mitigate its negative impact on health.

Abbreviations

OSA, obstructive sleep apnea; IR, insulin resistance; FBG, fasting blood glucose; TG, triglycerides; TC, total cholesterol; METS-IR, metabolic score for insulin resistance; BMI, body mass index; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; COPD, chronic obstructive pulmonary disease; CHD, coronary heart disease; CKD, chronic kidney disease; TyG, triglyceride glucose; TyG-BMI, triglyceride glucose-body mass index; TyG-WC, triglyceride glucose-waist circumference; AHI, apnea-hypopnea index; ODI, oxygen desaturation index; T90, the

percentage of time with oxygen saturation less than 90%; ROC, receiver operating characteristic; AUC, area under the curve; T2DM, type 2 diabetes mellitus; ER, endoplasmic reticulum; CIH, chronic intermittent hypoxia.

Data Sharing Statement

The datasets used and analyzed during the current study are available from the corresponding author on reasonable request.

Ethics Approval and Consent to Participate

The study was approved by the Ethics Committee of Renmin Hospital of Wuhan University. Informed consent was waived due to the retrospective analysis of anonymized clinical data, which posed no additional risks to participants and involved no direct patient interaction. All data were de-identified before analysis and handled according to hospital confidentiality protocols to ensure privacy. This study adhered to the ethical standards of the Declaration of Helsinki.

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Author Contributions

Beini Zhou and Yan Yao: Conceptualization, Methodology, Writing - original draft. Yuhan Wang and Wuriliga Yue: Data curation, Formal analysis, Writing - review & editing. Jingyi Zhang and Yang He: Data curation, Writing - review & editing. Qingfeng Zhang and Yixuan Wang: Resources, Formal analysis, Writing - review & editing. Ke Hu: Supervision, Methodology, Writing - review & editing. All authors have reviewed and agreed on the journal to which the article will be submitted. They have contributed to and approved all versions of the article before submission, during revision, the final version accepted for publication, and any significant changes introduced at the proofing stage. All authors have agreed to take responsibility and be accountable for the contents of the article.

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Disclosure

The authors report no conflicts of interest in this work.

References

1. Franklin KA, Lindberg E. Obstructive sleep apnea is a common disorder in the population-a review on the epidemiology of sleep apnea. *J Thorac Dis*. 2015;7(8):1311–1322. doi:10.3978/j.issn.2072-1439.2015.06.11
2. Mao Z, Zheng P, Zhu X, et al. Obstructive sleep apnea hypopnea syndrome and vascular lesions: an update on what we currently know. *Sleep Med*. 2024;119:296–311. doi:10.1016/j.sleep.2024.05.010
3. Maniaci A, Lavalle S, Parisi FM, et al. Impact of obstructive sleep apnea and sympathetic nervous system on cardiac health: a comprehensive review. *J Cardiovasc Dev Dis*. 2024;11(7). doi:10.3390/jcdd11070204
4. Rochlani Y, Pothineni NV, Kovelamudi S, Mehta JL. Metabolic syndrome: pathophysiology, management, and modulation by natural compounds. *Ther Adv Cardiovasc Dis*. 2017;11(8):215–225. doi:10.1177/1753944717711379
5. Alenezi MA, Alabdulathim S, Alhejaili SAM, et al. The association between obesity and the development and severity of obstructive sleep apnea: a systematic review. *Cureus*. 2024;16(9):e69962. doi:10.7759/cureus.69962
6. Framnes SN, Arble DM. The bidirectional relationship between obstructive sleep apnea and metabolic disease. *Front Endocrinol*. 2018;9:440. doi:10.3389/fendo.2018.00440
7. Young T, Palta M, Dempsey J, Skatrud J, Weber S, Badr S. The occurrence of sleep-disordered breathing among middle-aged adults. *N Engl J Med*. 1993;328(17):1230–1235. doi:10.1056/nejm199304293281704
8. Zheng M, Duan X, Zhou H, et al. Association between glycolipids and risk of obstructive sleep apnea: a population-based study. *Front Nutr*. 2023;10:974801. doi:10.3389/fnut.2023.974801
9. Drager LF, Jun JC, Polotsky VY. Metabolic consequences of intermittent hypoxia: relevance to obstructive sleep apnea. *Best Pract Res Clin Endocrinol Metab*. 2010;24(5):843–851. doi:10.1016/j.beem.2010.08.011
10. Azman M, Sani A, Kamaruddin NA. Insulin resistance using HOMA model in obstructive sleep apnea: a cross sectional study. *Ann Saudi Med*. 2014;34(6):476–481. doi:10.5144/0256-4947.2014.476

11. Sharma P, Dong Y, Somers VK, et al. Intermittent hypoxia regulates vasoactive molecules and alters insulin-signaling in vascular endothelial cells. *Sci Rep*. 2018;8(1):14110. doi:10.1038/s41598-018-32490-3
12. Tenda ED, Henrina J, Cha JH, et al. Obstructive sleep apnea: overlooked comorbidity in patients with diabetes. *World J Diabetes*. 2024;15 (7):1448–1460. doi:10.4239/wjd.v15.i7.1448
13. Behnoush AH, Khalaji A, Ghondaghsaz E, et al. Triglyceride-glucose index and obstructive sleep apnea: a systematic review and meta-analysis. *Lipids Health Dis*. 2024;23(1):4. doi:10.1186/s12944-024-02005-3
14. Després JP, Lemieux I. Abdominal obesity and metabolic syndrome. *Nature*. 2006;444(7121):881–887. doi:10.1038/nature05488
15. Lam JC, Mak JC, Ip MS. Obesity, obstructive sleep apnoea and metabolic syndrome. *Respirology*. 2012;17(2):223–236. doi:10.1111/j.1440-1843.2011.02081.x
16. Huang J, Zhuang J, Zheng H, et al. A machine learning prediction model of adult obstructive sleep apnea based on systematically evaluated common clinical biochemical indicators. *Nat Sci Sleep*. 2024;16:413–428. doi:10.2147/nss.S453794
17. Bello-Chavolla OY, Almeda-Valdes P, Gomez-Velasco D, et al. METS-IR, a novel score to evaluate insulin sensitivity, is predictive of visceral adiposity and incident type 2 diabetes. *Eur J Endocrinol*. 2018;178(5):533–544. doi:10.1530/eje-17-0883
18. Han KY, Gu J, Wang Z, et al. Association between METS-IR and prehypertension or hypertension among normoglycemia subjects in japan: a retrospective study. *Front Endocrinol*. 2022;13:851338. doi:10.3389/fendo.2022.851338
19. Qian T, Sheng X, Shen P, Fang Y, Deng Y, Zou G. Mets-IR as a predictor of cardiovascular events in the middle-aged and elderly population and mediator role of blood lipids. *Front Endocrinol*. 2023;14:1224967. doi:10.3389/fendo.2023.1224967
20. Zhou Y, Xue F. Exploring the association between triglyceride-glucose indices and their derivatives with obstructive sleep apnea: insights from the national health and nutrition examination survey. *Nat Sci Sleep*. 2025;17:143–155. doi:10.2147/nss.S487596
21. Scinicariello F, Buser MC, Feroe AG, Attanasio R. Antimony and sleep-related disorders: NHANES 2005-2008. *Environ Res*. 2017;156:247–252. doi:10.1016/j.envres.2017.03.036
22. Chen Y, Xie K, Han Y, Ju H, Sun J, Zhao X. The association between triglyceride-glucose index and its combination with systemic inflammation indicators and all-cause and cardiovascular mortality in the general US population: NHANES 1999-2018. *Lipids Health Dis*. 2024;23(1):289. doi:10.1186/s12944-024-02277-9
23. Wu X, Zhao D, Hu W, et al. Randomised, controlled crossover trial of intermittent and continuous transcutaneous electrical stimulation of the genioglossus muscle for obstructive sleep apnoea. *Thorax*. 2023;78(7):713–720. doi:10.1136/thorax-2021-218277
24. Zhang Q, Wang Z, Ding J, et al. Effect of obstructive sleep apnea on in vitro fertilization outcomes in women with polycystic ovary syndrome. *J Clin Sleep Med*. 2024;20(1):31–38. doi:10.5664/jcsm.10780
25. Zhang J, Liu X, Zha S, Chen H, Zhang Q, Hu K. Physiological effects and tolerance of wearing surgical and N95 masks during sleep in normal individuals and patients with mild-moderate obstructive sleep apnea: a randomized crossover trial. *Am J Med*. 2024;137(11):1128–1135.e4. doi:10.1016/j.amjmed.2024.06.013
26. Wei Z, Xu J, Li W, et al. Evaluation of a non-contact ultra-wideband bio-radar sleep monitoring device for screening of sleep breathing disease. *Sleep Breath*. 2022;26(2):689–696. doi:10.1007/s11325-021-02424-x
27. Zhao R, Xue J, Zhang X, et al. Comparison of ring pulse oximetry using reflective photoplethysmography and PSG in the detection of OSA in Chinese adults: a pilot study. *Nat Sci Sleep*. 2022;14:1427–1436. doi:10.2147/nss.S367400
28. Alam I, Lewis K, Stephens JW, Baxter JN. Obesity, metabolic syndrome and sleep apnoea: all pro-inflammatory states. *Obes Rev*. 2007;8 (2):119–127. doi:10.1111/j.1467-789X.2006.00269.x
29. Bonsignore MR, McNicholas WT, Montserrat JM, Eckel J. Adipose tissue in obesity and obstructive sleep apnoea. *Eur Respir J Mar*. 2012;39 (3):746–767. doi:10.1183/09031936.00047010
30. Onat A, Hergenç G, Yüksel H, et al. Neck circumference as a measure of central obesity: associations with metabolic syndrome and obstructive sleep apnea syndrome beyond waist circumference. *Clin Nutr*. 2009;28(1):46–51. doi:10.1016/j.clnu.2008.10.006
31. Pugliese G, Barrea L, Laudisio D, et al. Sleep apnea, obesity, and disturbed glucose homeostasis: epidemiologic evidence, biologic insights, and therapeutic strategies. *Curr Obes Rep*. 2020;9(1):30–38. doi:10.1007/s13679-020-00369-y
32. Lee SH, Park SY, Choi CS. Insulin resistance: from mechanisms to therapeutic strategies. *Diabetes Metab J*. 2022;46(1):15–37. doi:10.4093/dmj.2021.0280
33. Briançon-Marjollet A, Netchitaïlo M, Fabre F, et al. Intermittent hypoxia increases lipid insulin resistance in healthy humans: a randomized crossover trial. *J Sleep Res*. 2024;34(2):e14243. doi:10.1111/jsr.14243
34. Gu C, Younas H, Jun JC. Sleep apnea: an overlooked cause of lipotoxicity? *Med Hypotheses*. 2017;108:161–165. doi:10.1016/j.mehy.2017.09.007
35. Koenig AM, Koehler U, Hildebrandt O, et al. The effect of obstructive sleep apnea and continuous positive airway pressure therapy on skeletal muscle lipid content in obese and nonobese men. *J Endocr Soc*. 2021;5(8):bvab082. doi:10.1210/jendso/bvab082
36. Mirjalili SR, Soltani S, Meybodi ZH, et al. Which surrogate insulin resistance indices best predict coronary artery disease? A machine learning approach. *Cardiovasc Diabetol*. 2024;23(1):214. doi:10.1186/s12933-024-02306-y
37. Wang G, Zhu Z, Wang Y, et al. The association between METS-IR, an indirect index for insulin resistance, and lung cancer risk. *Eur J Public Health*. 2024;34(4):800–805. doi:10.1093/eurpub/ckad234
38. Su X, Zhao C, Zhang X. Association between METS-IR and heart failure: a cross-sectional study. *Front Endocrinol*. 2024;15:1416462. doi:10.3389/fendo.2024.1416462
39. Dong J, Liu YH, Lu YK, et al. Association between surrogate indicators of insulin resistance and risk of type 2 diabetes combined with hypertension among Chinese adults: two independent cohort studies. *Nutr Metab*. 2022;19(1):85. doi:10.1186/s12986-022-00720-1
40. Hou Y, Wu X, Shi Y, et al. METS-IR as an important predictor of neurological impairment severity in patients with severe cerebral infarction: a multicenter study based on the Chinese population. *Front Neurol*. 2024;15:1450825. doi:10.3389/fneur.2024.1450825
41. Meng X, Wen H, Lian L. Association between triglyceride glucose-body mass index and obstructive sleep apnea: a study from NHANES 2015-2018. *Front Nutr*. 2024;11:1424881. doi:10.3389/fnut.2024.1424881
42. Hu J, Cai X, Li N, et al. Association between triglyceride glucose index-waist circumference and risk of first myocardial infarction in Chinese hypertensive patients with obstructive sleep apnoea: an observational cohort study. *Nat Sci Sleep*. 2022;14:969–980. doi:10.2147/nss.S362101
43. Williams CM. Lipid metabolism in women. *Proc Nutr Soc*. 2004;63(1):153–160. doi:10.1079/pns2003314

44. Kahn SE, Hull RL, Utzschneider KM. Mechanisms linking obesity to insulin resistance and type 2 diabetes. *Nature*. 2006;444(7121):840–846. doi:10.1038/nature05482
45. Bell JA, Santos Ferreira DL, Fraser A, et al. Sex differences in systemic metabolites at four life stages: cohort study with repeated metabolomics. *BMC Med*. 2021;19(1):58. doi:10.1186/s12916-021-01929-2
46. Power ML, Schulkin J. Sex differences in fat storage, fat metabolism, and the health risks from obesity: possible evolutionary origins. *Br J Nutr*. 2008;99(5):931–940. doi:10.1017/s0007114507853347