








Triglyceride-Glucose Index in Chinese Patients with Obstructive Sleep Apnea in the Absence of Traditional Confounding Factors: A Propensity Score-Matched Cross-Sectional Study

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Background: Obstructive sleep apnea (OSA) affects nearly one billion adults globally and significantly increases cardiovascular and metabolic risks, largely due to insulin resistance (IR). The triglyceride-glucose (TyG) index is a validated, cost-effective surrogate for IR. However, studies report conflicting associations between the TyG index and the presence or severity of OSA, possibly due to confounding factors such as age, gender, and obesity (BMI). This study aimed to clarify the independent TyG–OSA relationship by adjusting for confounders using propensity score matching (PSM).

Methods: This cross-sectional study included 394 patients with OSA (apnea-hypopnea index [AHI] ≥ 5 events/hour) and 285 controls (AHI < 5 events/hour). PSM (1:1) balanced groups for age, gender, height, weight, and BMI. Differences in the TyG index between groups and across OSA severity (mild, moderate, severe) were analyzed pre- and post-PSM. Predictive performance was assessed using receiver operating characteristic (ROC) curves.

Results: Pre-PSM, the TyG index was significantly higher in patients with OSA than in controls ($p < 0.001$) and increased with severity ($p < 0.001$). Post-PSM (185 matched pairs), the TyG index remained significantly higher in moderate or severe OSA versus matched controls ($p < 0.05$) but not in mild OSA. ROC analysis demonstrated that PSM reduced the area under the curve (AUC) for predicting any OSA (from 0.709 to 0.628; $p < 0.001$) but substantially increased the AUC for predicting severe OSA (from 0.752 to 0.843; $p < 0.001$), improving sensitivity (0.754 to 0.796) and specificity (0.796 to 0.843).

Conclusion: This PSM analysis provides robust evidence of an independent association between the TyG index and OSA, particularly in moderate-to-severe cases. The TyG index demonstrates strong predictive value for severe OSA, supporting its utility for risk stratification and monitoring in clinical practice.

Keywords: triglyceride-glucose index, retrospective study, insulin resistance, cross-sectional study, propensity score matching, obstructive sleep apnea-hypopnea syndrome

Background

Obstructive sleep apnea (OSA), a global major public health burden, is characterized by recurrent upper airway collapse during sleep, leading to intermittent hypoxia, sleep fragmentation, and hypercapnia.^{1–3} Current estimates suggest that nearly one billion individuals aged 30–69 years worldwide are affected.³ OSA is linked to increased risks of cardiovascular disease (CVD), metabolic dysfunction, and all-cause mortality, independent of traditional risk factors.^{4–8}

The adverse cardiometabolic consequences of OSA are increasingly linked to its induction of insulin resistance (IR), systemic inflammation, and oxidative stress.^{9–16} As a core pathophysiological mechanism, IR requires accurate yet practical assessment in clinical practice. While the hyperinsulinemic-euglycemic clamp and the homeostatic model assessment of insulin resistance (HOMA-IR) are established methods, the Triglyceride-Glucose (TyG) index has emerged as a simple, reliable, cost-effective surrogate marker of IR, requiring only fasting triglyceride and glucose measurements.^{17–20} The TyG index shows a strong correlation with gold-standard IR measures and predicts incident type 2 diabetes mellitus and CVD outcomes.^{21–23}

Existing studies report conflicting associations between the TyG index and OSA. Several cross-sectional and observational studies report a significant positive association between OSA severity (typically measured by the apnea-hypopnea index, AHI) and elevated TyG index values, suggesting TyG as a potential biomarker reflecting OSA-related metabolic dysregulation.^{23–25} Conversely, other investigations, such as the analysis by Pei H et al, found no significant independent association between the TyG index and OSA presence or severity after multivariate adjustment, particularly when accounting for body mass index (BMI).²⁶

This discrepancy highlights critical methodological challenges and potential confounding. Key factors known to profoundly influence both OSA risk and TyG index values include obesity (a primary driver of both IR and upper airway collapsibility),²⁷ age (associated with increased OSA prevalence and metabolic changes),²⁷ and gender (with documented differences in TyG index and cardiovascular risk factors).²⁸ We hypothesize that the previously reported conflicting associations between the TyG index and OSA may be substantially confounded by age, gender, and obesity (as measured by BMI). To rigorously test this hypothesis and elucidate the nature of the TyG–OSA relationship independent of these key confounders, we propose a novel analytical approach.

This cross-sectional study aims to determine the independent association between the TyG index and both the presence and severity of OSA. We will employ propensity score matching (PSM) to meticulously balance participants with and without OSA for the critical confounding variables of age, gender, and BMI. By minimizing the influence of these factors through PSM, this study aims to provide robust, unbiased evidence regarding the intrinsic link between the TyG index and OSA, resolving existing contradictions in the literature and clarifying the clinical utility of the TyG index in the context of OSA.

Materials and Methods

Study Design and Participants

This retrospective cross-sectional study followed the STROBE guidelines. We enrolled adults (≥ 18 years) with clinically suspected OSA who underwent overnight polysomnography (PSG) at our tertiary sleep center between June 2022 and February 2024. Inclusion criteria: (1) clinical indicators of OSA (snoring, witnessed apneas, or excessive daytime sleepiness); (2) completion of standardized PSG and fasting laboratory tests within 48 hours after admission; and (3) complete demographic and biochemical data. Exclusion criteria: (1) chronic liver disease (viral hepatitis, alcoholic liver disease, or hepatocellular carcinoma); (2) severe cardiopulmonary disease (NYHA class III–IV heart failure or COPD GOLD stage III–IV); and (3) active malignancy or immunosuppressive therapy. Participants were categorized into two groups: the OSA group (AHI ≥ 5 events/hour) and the control group (AHI < 5 events/hour). [Figure 1](#) details the enrollment process.

Data Collection Protocol

All measurements were completed within 48 hours of admission under standardized conditions. Baseline anthropometrics (age, gender, height, weight) were extracted from medical records. BMI was calculated as $\text{weight}(\text{kg})/\text{height}(\text{m})^2$. Laboratory analysis required 8–10h overnight fasting before venous blood collection for tests including red blood cell count (RBC), white blood cell count (WBC), hemoglobin, low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), non-HDL-C, total cholesterol (TC), triglycerides (TG), and fasting blood glucose (FBG). Participants abstained from caffeine, alcohol, smoking, and vigorous exercise for 12 hours pre-phlebotomy. The TyG index was calculated as: $\text{Ln}[\text{TG}(\text{mg/dL}) \times \text{FBG}(\text{mg/dL})/2]$. All participants who underwent ≥ 7 h PSG scored per

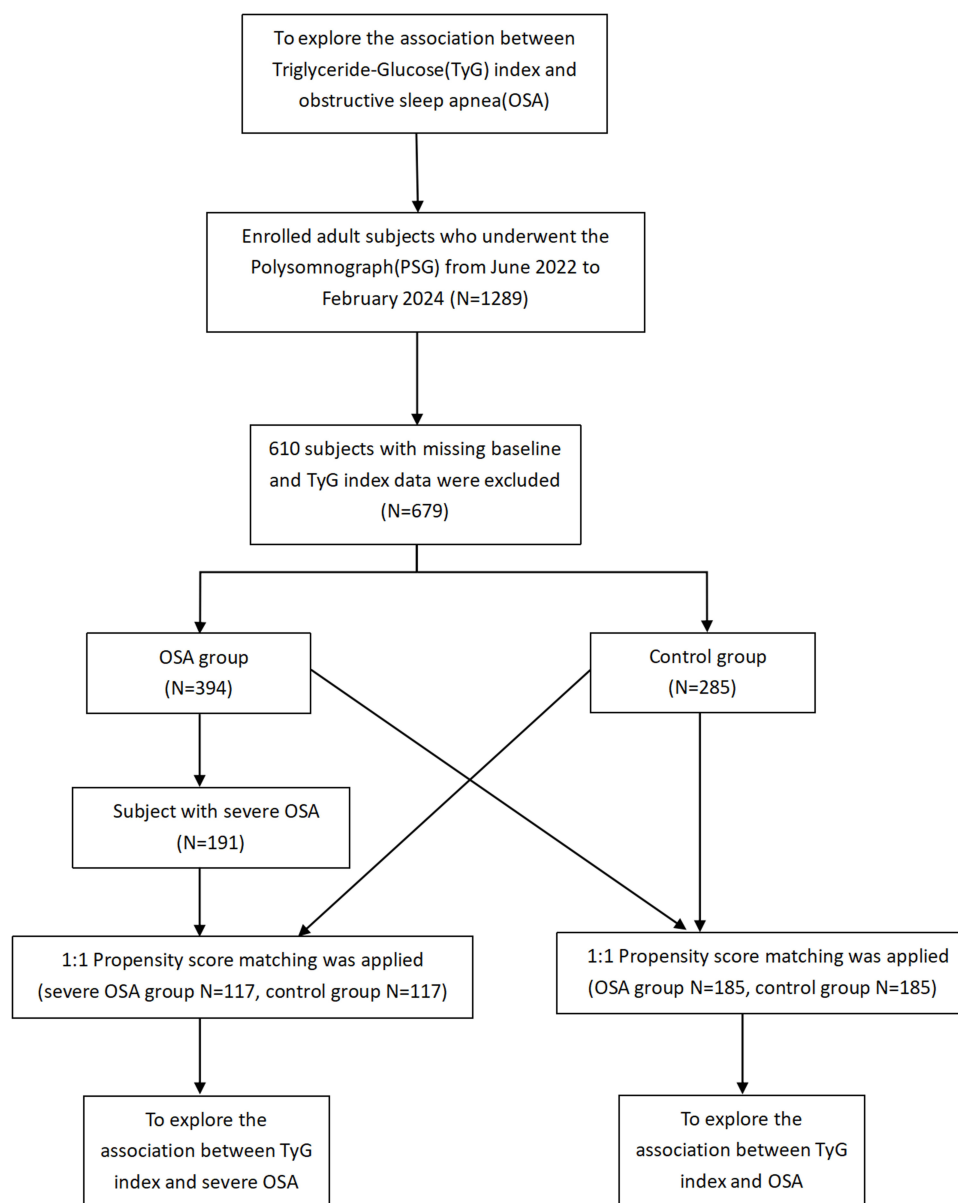


Figure 1 Flow chart of the selection.

American Academy of Sleep Medicine (AASM) guidelines.²⁹ OSA severity was stratified as: mild ($5 \leq \text{AHI} < 15$), moderate ($15 \leq \text{AHI} < 30$), and severe ($\text{AHI} \geq 30$).

Propensity Score Matching (PSM)

To control for confounding factors like age, gender, and obesity, the following approach was used: Matching variables: Age, gender, height, weight, and BMI. Methodology: (1) Propensity scores were generated using logistic regression (OSA vs control as the outcome). (2) 1:1 nearest-neighbor matching was performed with a caliper width of 0.05 SD of the logit propensity score. (3) The R package MatchIt (v4.2.1) was used for implementation. (4) Covariate balance was confirmed using standardized mean differences (< 0.10).

Statistical Analyses

Statistical analyses were performed using SPSS 24.0 software (SPSS, Inc.; Chicago, IL, USA) and R 4.2.1 software. Continuous variables are presented as mean \pm SD, and categorical variables as percentages. Differences between groups were assessed using independent sample *t*-tests (normal variables) and Mann–Whitney *U*-tests (non-normal variables). Categorical variables were compared using Chi-square tests. The Benjamini-Hochberg procedure was applied to all subgroup analyses. Statistical significance was set at $p < 0.05$. The TyG index was divided into tertiles to explore its relationship with OSA severity. The receiver operating characteristic (ROC) curve was analyzed to calculate the area under the curve (AUC), evaluating the TyG index's predictive ability.

Results

Overall Baseline Characteristics

Before PSM, there were 285 controls and 394 OSA patients. As shown in Table 1, the OSA group had more males than the control group ($p < 0.01$). OSA patients had significantly higher height, weight, BMI, FBG, TG, and TyG index values. They also had higher hemoglobin, WBC, and RBC levels but lower HDL-C levels (all $p < 0.05$). After PSM, 185 well-matched controls and OSA patients were obtained. Table 1 compares the groups' characteristics before and after matching. The matching eliminated significant differences in key variables like age, height, weight, BMI, and gender. Even after adjusting for age, height, weight, gender, and BMI, OSA patients still had higher hemoglobin, RBC, FBG, TG, and TyG index levels and lower HDL-C levels than controls (all $p < 0.05$).

Baseline Characteristics of Subgroups of OSA Patients According to TyG Index Tertiles

The TyG index values were divided into three tertiles: group 1 (7.34–8.53), group 2 (8.54–9.08), and group 3 (9.10–10.88). Of the 394 OSA patients, 131, 131, and 132 were in tertiles 1, 2, and 3, respectively. Table 2 compares the baseline characteristics of these three subgroups. Age, weight, BMI, TG, FBG, WBC, RBC, hemoglobin, TC, and HDL-C differed significantly across the tertiles (all $p < 0.05$). As TyG index tertiles increased, BMI, TG, FBG, hemoglobin, and

Table 1 Baseline Characteristics of OSA and Control Group Before and After Propensity Score Matching

| | Before Propensity Score Matching | | | After Propensity Score Matching | | |
|--------------------------------|----------------------------------|-------------------|---------|---------------------------------|-------------------|---------|
| | OSA (n=394) | Control (n=185) | p-value | OSA (n=185) | Control (n=185) | p-value |
| Age (years) | 52.06 \pm 13.13 | 49.99 \pm 14.40 | 0.052 | 51.78 \pm 12.38 | 52.54 \pm 14.06 | 0.583 |
| Gender (Males n, %) | 297(75.38) | 153(82.70) | <0.001* | 120(64.86) | 123(66.49) | 0.743 |
| Height (m) | 166.67 \pm 8.12 | 164.20 \pm 8.64 | <0.001* | 165.27 \pm 8.44 | 165.48 \pm 8.55 | 0.816 |
| Weight (kg) | 77.50 \pm 15.49 | 65.74 \pm 13.77 | <0.001* | 71.77 \pm 12.92 | 71.32 \pm 12.60 | 0.734 |
| BMI (kg/m ²) | 27.75 \pm 4.29 | 24.23 \pm 3.84 | <0.001* | 26.13 \pm 3.28 | 25.93 \pm 3.33 | 0.570 |
| WBC (10 ⁹ /L) | 6.87 \pm 1.90 | 6.44 \pm 1.96 | 0.004* | 6.64 \pm 1.76 | 6.55 \pm 1.92 | 0.636 |
| Hemoglobin (g/l) | 143.29 \pm 17.22 | 88.53 \pm 40.84 | <0.001* | 140.71 \pm 16.88 | 88.60 \pm 41.75 | <0.001* |
| RBC (10 ⁹ /L) | 4.84 \pm 0.62 | 4.10 \pm 1.35 | <0.001* | 4.75 \pm 0.58 | 4.15 \pm 1.24 | <0.001* |
| Triglyceride (mmol/L) | 2.08 \pm 1.57 | 1.45 \pm 0.92 | <0.001* | 2.16 \pm 1.85 | 1.64 \pm 1.02 | 0.001* |
| TyG index | 8.88 \pm 0.67 | 8.40 \pm 0.63 | <0.001* | 8.88 \pm 0.72 | 8.57 \pm 0.64 | <0.001* |
| Total cholesterol (mmol/L) | 4.73 \pm 1.04 | 4.67 \pm 1.09 | 0.435 | 4.84 \pm 1.03 | 4.63 \pm 1.12 | 0.058 |
| HDL-C (mmol/L) | 1.07 \pm 0.27 | 1.27 \pm 0.35 | <0.001* | 1.11 \pm 0.31 | 1.19 \pm 0.31 | 0.012* |
| Non-HDL-C (mmol/L) | 3.66 \pm 1.03 | 3.39 \pm 1.07 | 0.001* | 3.73 \pm 1.01 | 3.44 \pm 1.12 | 0.008* |
| LDL-C (mmol/L) | 2.79 \pm 0.93 | 2.72 \pm 0.99 | 0.334 | 2.84 \pm 0.93 | 2.69 \pm 1.03 | 0.151 |
| Fasting Blood Glucose (mmol/L) | 6.48 \pm 2.48 | 5.47 \pm 2.09 | <0.001* | 6.40 \pm 2.57 | 5.74 \pm 2.45 | 0.012* |

Notes: *There is a statistically significant difference.

Abbreviations: OSA, obstructive sleep apnea; TyG index, triglyceride-glucose index; BMI, body mass index; WBC, white blood cell; RBC, red blood cell; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol, non-HDL-C, non-high-density lipoprotein cholesterol.

Table 2 Baseline Characteristics of Subgroups of OSA Patients According to TyG Index Tertiles

| | Overall | Tertiles of Triglyceride-Glucose Index (n=394) | | | p-value | adj. p-value |
|--------------------------------|--------------|--|------------------------------------|----------------------------------|---------|--------------|
| | | Tertile1 (n=131) (7.34~8.53) | Tertile2 (n=131) (8.54~9.08) | Tertile3 (n=132) (9.10~10.88) | | |
| Age (years) | 52.06±13.13 | 55.69±13.70 | 49.25±12.24 | 51.24±12.66 | 0.006* | 0.010* |
| Gender (Males n, %) | 297(97) | 89(42) | 104(27) | 104(28) | 0.057 | 0.075 |
| Height (m) | 166.67±8.12 | 165.19±8.15 | 167.76±7.91 | 167.07±8.15 | 0.061 | 0.075 |
| Weight (kg) | 77.50±15.49 | 72.72±14.53 | 80.16±17.69 | 79.60±12.87 | <0.001* | <0.001* |
| BMI (kg/m ²) | 27.75±4.29 | 26.54±4.50 | 28.29±4.60 | 28.42±3.45 | <0.001* | <0.001* |
| WBC (10 ⁹ /L) | 6.87±1.90 | 6.35±1.82 | 7.17±1.77 | 7.10±2.02 | 0.001* | 0.002* |
| RBC (10 ⁹ /L) | 4.84±0.62 | 4.70±0.68 | 4.94±0.56 | 4.88±0.59 | 0.020* | 0.029* |
| Hemoglobin (g/l) | 143.29±17.22 | 138.12±17.21 | 144.99±17.33 | 146.73±16.01 | <0.001* | <0.001* |
| Triglyceride (mmol/L) | 2.08±1.57 | 1.05±0.27 | 1.72±0.39 | 3.45±2.01 | <0.001* | <0.001* |
| Total cholesterol (mmol/L) | 4.73±1.04 | 4.41±0.96 | 4.64±0.95 | 5.14±1.08 | <0.001* | <0.001* |
| HDL-C (mmol/L) | 1.07±0.27 | 1.24±0.30 | 1.05±0.22 | 0.94±0.18 | <0.001* | <0.001* |
| Non-HDL-C (mmol/L) | 3.66±1.03 | 3.18±0.88 | 3.59±0.89 | 4.20±1.05 | <0.001* | <0.001* |
| LDL-C (mmol/L) | 2.79±0.93 | 2.66±0.84 | 2.86±0.88 | 2.85±1.04 | 0.086 | 0.092 |
| Fasting Blood Glucose (mmol/L) | 6.48±2.48 | 5.31±0.89 | 6.04±1.46 | 8.08±3.38 | <0.001* | <0.001* |
| AHI (times) | 33.93±23.80 | 31.48±23.17 | 34.59±24.01 | 35.72±24.18 | 0.149 | 0.149 |
| Lowest SaO ₂ | 78.19±10.20 | 79.60±9.55 | 77.56±10.84 | 77.42±10.12 | 0.085 | 0.092 |

Notes: adj. p-value is the p-value adjusted using the Benjamini-Hochberg correction. *There is a statistically significant difference.

Abbreviations: OSA, obstructive sleep apnea; BMI, body mass index; WBC, white blood cell; RBC, red blood cell; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol, non-HDL-C, non-high-density lipoprotein cholesterol; AHI, apnea-hypopnea index.

TC levels rose significantly, while HDL-C levels decreased. Additionally, AHI tended to increase and the lowest SaO₂ tended to decrease with higher TyG index tertiles.

Baseline Characteristics of OSA Patients with Various Disease Severity

Of the 394 OSA patients, 109 had mild, 94 had moderate, and 191 had severe OSA. As shown in Table 3, significant differences existed among the three groups in age, gender, height, weight, BMI, RBC, TG, and HDL-C (all p<0.05). Severe OSA patients had higher height, weight, BMI, and TG levels but lower age and HDL-C levels. Stratified analysis indicated that males with severe OSA had a significantly higher TyG index (9.12 ± 0.71 vs 8.79 ± 0.69 , p=0.003) independently of BMI.

TyG Index Comparisons

Before PSM, the TyG index differed significantly between OSA patients and controls (p<0.001; Figure 2a) and among the three OSA severity groups compared to controls (p<0.001; Figure 3a), but no significant differences were found among the three OSA severity levels (p>0.05; Figure 3a). TyG index increased with worsening OSA severity (p=0.038; Table 3). After PSM, the TyG index remained significantly higher in OSA patients than in controls (p<0.001; Figure 2b), even after adjusting for gender, age, height, weight, and BMI. TyG index values were significantly higher in moderate-to-severe OSA groups than in controls (both p<0.05), but not in the mild OSA group (p>0.05; Figure 3b). A significant difference was found between the mild and severe OSA groups (p<0.05; Figure 3b). This indicates the TyG index is independently associated with OSA, particularly in moderate to severe cases, and may be more effective in identifying these patients than those with mild OSA.

Predictive Value of the TyG Index for OSA

A ROC curve analysis was conducted to assess the TyG index's predictive ability for OSA. Before PSM, the AUC was 0.709 (95% CI: 0.670–0.748), with a sensitivity of 0.538, specificity of 0.789, and a cut-off value of 8.758 (p<0.001,

Table 3 Baseline Characteristics of OSA Patients with Various Disease Severity

| | Overall | Severity of OSA (n=394) | | | p-value | adj.p-value |
|--|--------------|-------------------------|-----------------|----------------|---------|-------------|
| | | Mild (n=109) | Moderate (n=94) | Severe (n=191) | | |
| Age (years) | 52.06±13.13 | 54.02±13.00 | 54.30±13.27 | 49.84±12.83 | 0.004* | 0.019* |
| Gender (Males n, %) | 297(75.38) | 72(66.06) | 71(75.53) | 154(80.63) | 0.019* | 0.046* |
| Height (m) | 166.67±8.12 | 164.98±8.67 | 166.85±7.66 | 167.55±7.91 | 0.010* | 0.035* |
| Weight (kg) | 77.50±15.49 | 72.24±13.69 | 74.90±12.84 | 81.77±16.48 | <0.001* | <0.001* |
| BMI (kg/m ²) | 27.75±4.29 | 26.35±3.19 | 26.83±3.83 | 29.00±4.69 | <0.001* | <0.001* |
| WBC (10 ⁹ /L) | 6.87±1.90 | 6.75±2.06 | 6.64±1.72 | 7.05±1.89 | 0.137 | 0.192 |
| RBC (10 ⁹ /L) | 4.84±0.62 | 4.75±0.65 | 4.78±0.59 | 4.91±0.60 | 0.023* | 0.046* |
| Hemoglobin (g/l) | 143.29±17.22 | 141.14±16.14 | 143.02±15.95 | 144.65±18.35 | 0.088 | 0.137 |
| Triglyceride (mmol/L) | 2.08±1.57 | 1.81±0.88 | 2.07±1.75 | 2.23±1.75 | 0.027* | 0.047* |
| Total cholesterol (mmol/L) | 4.73±1.04 | 4.70±1.07 | 4.79±1.06 | 4.72±1.02 | 0.979 | 0.979 |
| HDL-C (mmol/L) | 1.07±0.27 | 1.11±0.28 | 1.10±0.29 | 1.04±0.25 | 0.020* | 0.046* |
| Non-HDL-C (mmol/L) | 3.66±1.03 | 3.59±1.06 | 3.69±1.04 | 3.68±1.02 | 0.518 | 0.659 |
| LDL-C (mmol/L) | 2.79±0.93 | 2.81±0.94 | 2.83±0.95 | 2.76±0.92 | 0.637 | 0.686 |
| Fasting Blood Glucose (mmol/L) | 6.48±2.48 | 6.64±2.67 | 6.33±1.91 | 6.46±2.62 | 0.616 | 0.686 |
| Tertiles of Triglyceride-Glucose Index | | | | | 0.038* | |
| Tertile1 (7.34–8.53) | 131 | 31(23.66) | 40(30.53) | 60(45.80) | | |
| Tertile2 (8.54–9.08) | 131 | 46(35.11) | 21(16.03) | 64(48.85) | | |
| Tertile3 (9.10–10.88) | 132 | 32(24.24) | 33(25.00) | 67(50.76) | | |

Notes: adj. p-value is the p-value adjusted using the Benjamini-Hochberg correction. *There is a statistically significant difference.

Abbreviations: OSA, obstructive sleep apnea; BMI, body mass index; WBC, white blood cell; RBC, red blood cell; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol, non-HDL-C, non-high-density lipoprotein cholesterol.

Figure 4a). After PSM, the AUC decreased to 0.628 (95% CI: 0.572–0.685), sensitivity rose to 0.541, specificity fell to 0.676, and the cut-off value dropped to 8.71 (p<0.001, Figure 4b). This indicates that while the TyG index has some predictive power for OSA, its predictive efficacy is influenced by factors like age, gender, and obesity.

Predictive Value of the TyG Index for Severe OSA

Of the 394 OSA patients, 191 had severe OSA. After using PSM to match 117 controls and severe OSA patients, we assessed the TyG index's ability to predict severe OSA via ROC curve analysis. Before PSM, the AUC was 0.752 (95% CI: 0.707–0.796), with a sensitivity of 0.602, specificity of 0.790, and a cut-off value of 8.712 (p<0.001, Figure 5a). Post-

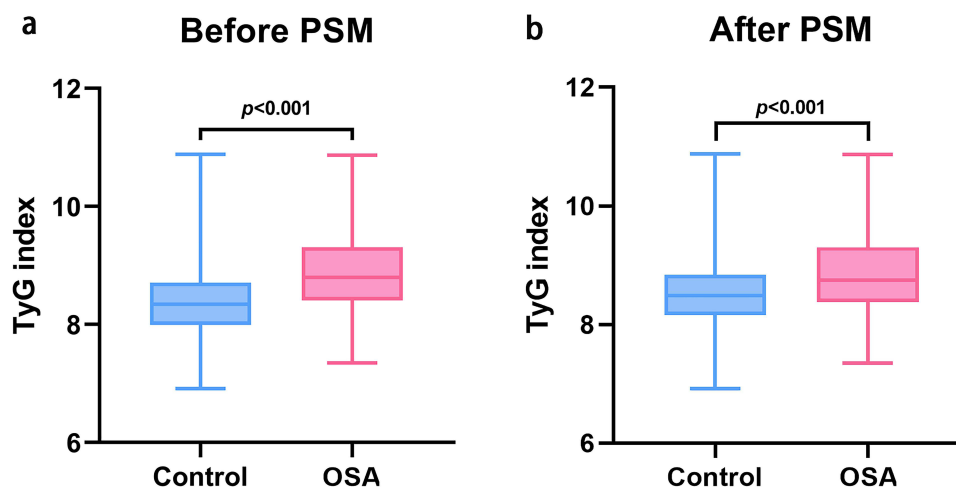


Figure 2 TyG index compared between the control and OSA group before and after propensity score matching. (a) before PSM and (b) after PSM. Data are presented with median, minimum, and maximum values.

Abbreviation: OSA, obstructive sleep apnea; TyG index, Triglyceride-glucose index; PSM, propensity score matching.

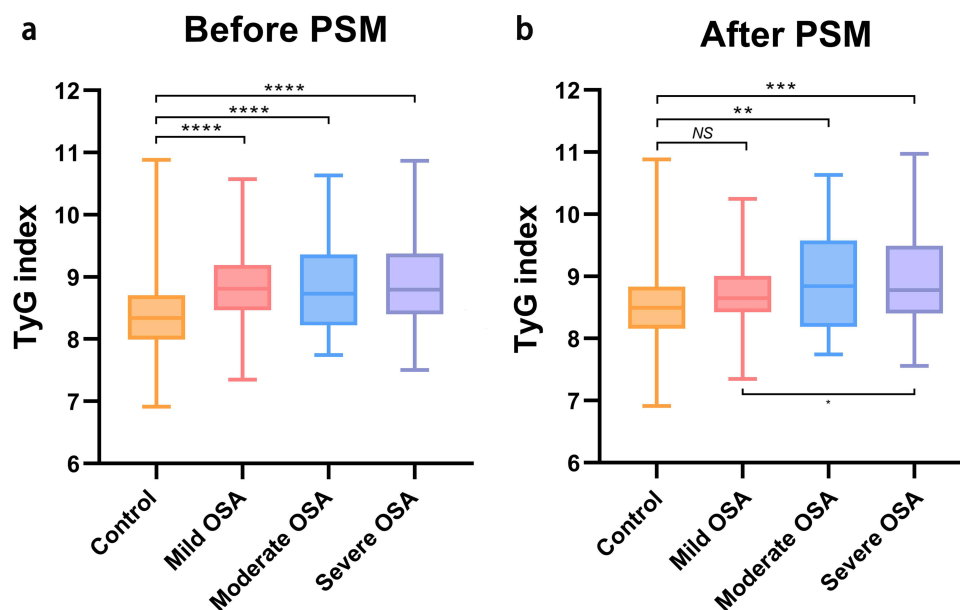


Figure 3 TyG index compared between the severity groups of OSA before and after propensity score matching. (a) before PSM; (b) after PSM. Data are presented with median, minimum, and maximum values. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$, **** $p < 0.0001$.

Abbreviations: NS, not significant; OSA, obstructive sleep apnea; TyG index, Triglyceride-glucose index; PSM, propensity score matching.

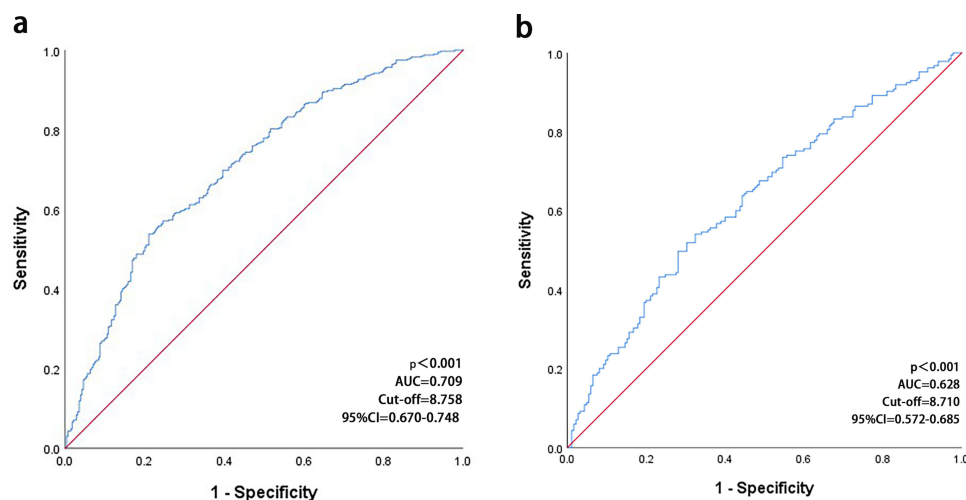


Figure 4 Receiver operating characteristic (ROC) curve analyses. ROC curves were applied to the prediction of OSA by the TyG index before and after propensity score matching. (a) before PSM: sensitivity (0.538), specificity (0.789); (b) after PSM: sensitivity (0.541), specificity (0.676).

Abbreviation: AUC, area under the curve; OSA, obstructive sleep apnea; TyG index, Triglyceride-glucose index.

PSM, the AUC rose to 0.843 (95% CI: 0.790–0.896), sensitivity increased to 0.754, specificity was 0.796, and the cut-off value slightly dropped to 8.701 ($p < 0.001$, Figure 5b). This indicates the TyG index is more effective in predicting severe OSA and its predictive power is less influenced by factors like age, gender, and obesity.

Discussion

This study employed PSM to minimize selection bias and covariate imbalance, affirming a significant link between the TyG index and OSA (especially moderate-to-severe cases), independent of age, gender, and BMI. This clarifies prior conflicting findings and underscores the TyG index's relevance to OSA pathophysiology beyond demographic and anthropometric factors.

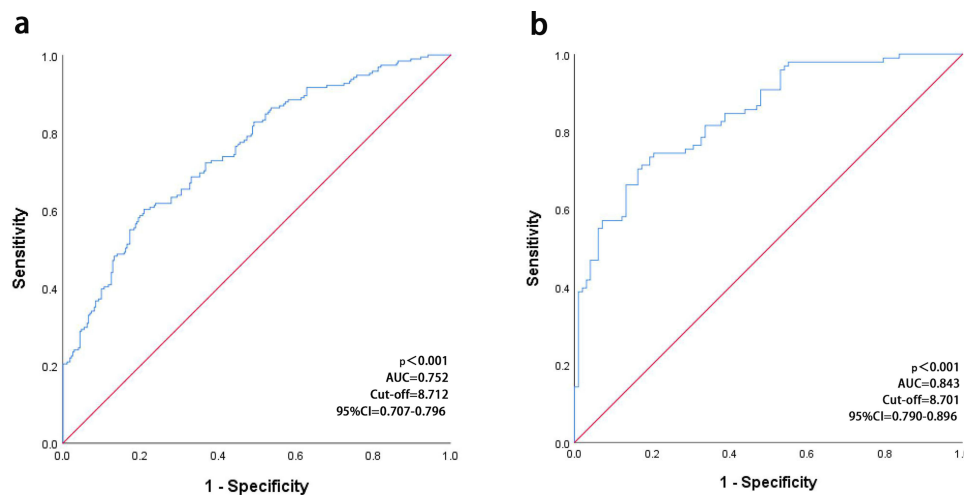


Figure 5 Receiver operating characteristic (ROC) curve analyses. ROC curves were applied to the prediction of severe OSA by the TyG index before and after propensity score matching. (a) before PSM: sensitivity (0.602), specificity (0.790); (b) after PSM: sensitivity (0.754), specificity (0.796).
Abbreviation: AUC, area under the curve; OSA, obstructive sleep apnea; TyG index, Triglyceride-glucose index.

Previous studies on the TyG index and OSA relationship have reported conflicting results.^{19,24–26,29,30} Our analysis reveals that age, gender, and obesity (BMI) are major confounders. Before PSM, we found significant TyG index differences between OSA patients and controls, consistent with some previous studies.^{19,24,25,30} However, after PSM, the TyG index remained significantly higher only in moderate to severe OSA cases compared to controls, while the difference in mild OSA disappeared. This suggests that the mild OSA association may be driven by shared risk factors like age, gender, and obesity, whereas the moderate-to-severe OSA link is more intrinsic. The reduced ROC-AUC for predicting OSA after PSM highlights how these confounders can inflate the TyG index's predictive ability in unadjusted analyses. Our approach addresses prior methodological challenges of residual confounding,^{25,26} showing that inadequate control for these factors, especially BMI, can lead to false-positive associations or potentially mask true associations in specific subgroups.

Obesity emerged as a paramount confounder, significantly influencing both OSA risk and TyG index values, as evidenced by baseline disparities and post-matching association attenuation. This reinforces that obesity must be strictly considered when investigating metabolic markers in OSA. The persistent TyG-OSA association after PSM, particularly for moderate to severe disease, suggests OSA-related mechanisms might independently contribute to IR or interact with adiposity to worsen metabolic dysfunction. This aligns with known pathophysiological pathways where OSA-induced intermittent hypoxia can directly promote IR, inflammation, and dyslipidemia,^{9–16} explaining why the TyG index remained elevated in more severe OSA even after BMI matching. The stratified analysis also showed a significantly higher TyG index in males with severe OSA, independent of BMI, further supporting that OSA severity directly impacts metabolic regulation.

Our results strongly indicate a positive link between the TyG index and OSA severity. Post-PSM, TyG levels were significantly higher in moderate and severe OSA groups compared to controls, and significant differences were evident between mild and severe OSA groups. Additionally, categorizing OSA patients by TyG index tertiles showed a clear trend: TyG index increases were associated with higher AHI and lower Lowest SaO₂, even after controlling for key confounders. This strengthens the case for the TyG index as a marker of metabolic burden in advanced OSA. Notably, the TyG index's predictive performance for severe OSA improved substantially after PSM (AUC rose from 0.752 to 0.843), suggesting it may better identify patients with significant, metabolically consequential OSA than detect OSA presence or mild cases.

The ROC analysis needs careful interpretation for clinical use. The post-PSM AUC of 0.628 (95% CI: 0.572–0.685) shows a low - moderate discriminative ability to distinguish OSA from controls. While statistically significant, this limits the TyG index's use as a single diagnostic tool for OSA. However, it can indicate insulin resistance, especially in

moderate-to-severe OSA, and is useful for risk assessment in those already suspected of sleep-disordered breathing or for identifying OSA patients at higher metabolic risk. The post-PSM AUC for severe OSA (0.843) supports using the TyG index to prioritize high-risk patients for expedited sleep studies or intensive metabolic management.

An intriguing observation was that severe OSA patients in our cohort tended to be younger. Though age is a known OSA risk factor, this finding is not coincidental. Importantly, severe OSA patients had significantly higher BMIs and weights than other groups (see Results Baseline Characteristics of OSA Patients with Various Disease Severity). This suggests obesity's impact on OSA severity may outweigh age's impact on our population. The pronounced metabolic burden and increased upper airway collapsibility linked to higher BMI may lead to more severe OSA at a younger age in susceptible individuals. However, our PSM methodology successfully balanced age and BMI between groups in the matched analyses, ensuring the observed TyG-OSA associations were not due to these differences.

The male predominance in our cohort aligns with established OSA epidemiology^{19,24,25,30} and reflects gender-based differences in disease manifestation and metabolic risk profiles.^{27,31} This baseline imbalance was a key reason for including gender as a matching variable in PSM. Successful elimination of gender differences in the matched cohort (SMD <0.10) strengthens our conclusion that the TyG-OSA association persists independently of gender. However, residual confounding due to unmeasured gender-specific factors or hormonal influences cannot be entirely excluded.

Several limitations must be acknowledged. First, our control group comprised individuals referred for PSG due to OSA suspicion (eg, snoring, excessive daytime sleepiness) but with an AHI <5. While this group may not represent perfectly healthy individuals, it provides a clinically relevant comparison for assessing the metabolic burden of diagnosed OSA. This definition is a limitation, and the results should be interpreted in this context. Second, we did not collect detailed data on diet, physical activity, or long-term dietary habits, which influence fasting glucose and triglyceride levels. Third, we did not include other IR measures (eg, HOMA-IR or clamp) for direct comparison. Fourth, our single-center design may limit the generalizability of our results. Fifth, the potential influence of medications (eg, statins and antihypertensives) on metabolic parameters or OSA severity cannot be fully ruled out. Finally, the cross-sectional design precludes causal inference.

Conclusions

In summary, this PSM analysis provides robust evidence of an independent association between the TyG index and OSA, particularly in moderate-to-severe cases. The TyG index can serve for initial screening, risk evaluation, and monitoring treatment effects and disease progression in moderate-to-severe OSA, especially in resource-limited settings. However, its diagnostic capacity is restricted and it cannot be used alone to confirm OSA diagnosis. In clinical practice, the TyG index should be used alongside other clinical information for a comprehensive, individualized assessment. Further research is needed to explore the specific mechanisms linking the TyG index to OSA and to optimize its combined use with other indicators to enhance OSA management.

Abbreviations

OSA, obstructive sleep apnea; CVD, cardiovascular disease; IR, insulin resistance; HOMA-IR, homeostasis model assessment of insulin resistance; TyG index, Triglyceride-Glucose index; BMI, body mass index; PSM, propensity score matching; PSG, polysomnography; RBC, red blood cells; WBC, white blood cells; LDL-C, low-density lipoprotein cholesterol; TG, triglycerides; non-HDL-C, non-high-density lipoprotein cholesterol; TC, total cholesterol; FBG, fasting blood glucose; HDL-C, high-density lipoprotein cholesterol; AHI, apnea-hypopnea index; SD, standard deviation; ROC, receiver operating characteristic; AUC, area under the curve; EDS, excessive daytime sleepiness.

Data Sharing Statement

The data that support the findings of this study are available on request from the corresponding author.

Ethics Approval and Consent to Participate

This study was approved by the Medical Ethics Committee of the Fifth Affiliated Hospital of Sun Yat-sen University (Approval number: K217-1), all participants signed informed consent, and the study process followed the ethical guidelines of the Declaration of Helsinki.

Author Contributions

Jun Zhang, Baoyi Chen, and Tengyu Chen are joint first authors. All authors approved the final version for publication, agreed to submit to this journal, and take accountability for all aspects of the work. Specific contributions: Jun Zhang, Baoyi Chen, Tengyu Chen: conceptualization, methodology, formal analysis and investigation, writing-original draft preparation, resources. RenJie Lai, Zhongkang Ye: conceptualization, methodology, formal analysis and investigation, writing-original draft preparation. Zhenpeng Liao, Yingxiang Xu, Shan Zhu: conceptualization, methodology, writing-original draft preparation. Anni Yang: conceptualization, writing-review and editing, supervision. Haiyu Hong: conceptualization, writing-review and editing, funding acquisition, supervision.

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Disclosure

The authors have no conflicts of interest to disclose.

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