British Journal of Hospital Medicine

Br. J. Hosp. Med. (Lond) **2026**; 87(10): 52648
https://doi.org/10.31083/BJHM52648

Article

A Model for Identifying Obesity Status Based on Inflammatory and Metabolic Indicators: The Role of the CRP-TG-GLU Index

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Academic Editor: Dimitrios Patoulias
Submitted: 7 April 2026 Revised: 21 July 2026 Accepted: 20 August 2026 Published: 20 October 2026

Abstract

Aims/Background: Obesity is a major public health concern. However, conventional anthropometric measures do not adequately reflect underlying inflammation and insulin resistance. This study aimed to validate a nomogram model for identifying obesity risk in adults, centered on the C-reactive protein-Triglyceride-Glucose composite index (CTI) and incorporating routinely available clinical indicators. **Methods:** A single-center retrospective observational study was conducted. A total of 360 adult subjects who completed health examinations at our hospital between January 2022 and December 2024 were consecutively enrolled. Participants were categorized into obese and non-obese groups according to World Health Organization (WHO) criteria (body mass index [BMI] ≥30 kg/m^2) and randomly divided into a training set (n = 252) and an internal validation set (n = 108) at a 7:3 ratio. The core predictor, CTI, was calculated as follows: CTI = 0.412 × Ln(hs-CRP, mg/L) + Ln[TG(mg/dL) × FPG(mg/dL) / 2]. In the training set, independently associated factors were identified using univariate and multivariate logistic regression to construct the final model. Model discrimination was assessed using the area under the receiver operating characteristic curve (AUC) with bootstrap correction, while calibration was evaluated using calibration curves and the Hosmer-Lemeshow test. The incremental value of CTI was assessed using the Net Reclassification Improvement (NRI) and Integrated Discrimination Improvement (IDI), and clinical utility was evaluated using decision curve analysis (DCA). **Results:** Among the 360 included subjects, the prevalence of obesity was 17.22%. Baseline characteristics were comparable between the training and validation sets ($p > 0.05$). Multivariate analysis identified waist circumference (WC) and CTI as independent factors associated with obesity (WC: OR = 1.117, 95% CI: 1.072–1.165, $p < 0.001$; CTI: OR = 1.684, 95% CI: 1.262–2.246, $p < 0.001$). The model demonstrated good discriminative performance, with a bootstrap-corrected AUC of 0.795, and satisfactory calibration (H-L test $p > 0.05$). Incremental value analysis showed that adding CTI to WC significantly improved risk reclassification (NRI = 0.242, p = 0.006; IDI = 0.072, p = 0.002). Sensitivity analysis using the Chinese BMI criterion (≥28 kg/m^2) yielded consistent results. Decision curve analysis demonstrated a clinical net benefit across a broad range of threshold probabilities. **Conclusion:** CTI, as a composite indicator reflecting both inflammation and insulin resistance, is independently associated with obesity and provides incremental value for risk stratification beyond WC alone. The proposed nomogram, based on routinely available health examination data, provides a convenient approach for obesity risk assessment in health management settings and may facilitate early risk stratification and targeted prevention. Future multicenter prospective studies are warranted to externally validate the model.

Keywords: obesity; C-reactive protein-triglyceride-glucose composite index; nomogram; inflammation; insulin resistance

1. Introduction

The global prevalence of obesity has reached epidemic proportions, making it one of the major public health threats of the twenty-first century [1]. Extensive epidemiological evidence has established that excess adiposity is associated with elevated risks of type 2 diabetes, hypertension, cardiovascular disease, and several malignancies, collectively contributing to substantial healthcare costs and socioeconomic burdens [2,3,4]. In routine clinical practice, body mass index (BMI) and waist circumference remain the standard anthropometric measures for obesity screening and classification [5]. However, these measurements primarily quantify overall adiposity and its distribution and do not reflect underlying pathophysiological hallmarks of obesity, particularly chronic low-grade systemic inflammation and insulin resistance [6,7]. Contemporary research has established that adipose tissue, especially the visceral adipose tissue, functions as a metabolically active endocrine organ that releases pro-inflammatory mediators, including interleukin-6 and tumor necrosis factor-α, thereby promoting systemic inflammation and increasing circulating levels of acute-phase proteins such as high-sensitivity C-reactive protein (hs-CRP) [8]. Concurrently, obesity is considered a hallmark of metabolic syndrome (MetS), in which disturbances in lipid and glucose metabolism contribute to insulin resistance, a key metabolic abnormality associated with obesity [9].

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Given the central role of inflammation and insulin resistance in the pathophysiology of obesity, biomarkers that capture these two interrelated processes may provide greater predictive value than conventional anthropometric measures. The triglyceride-glucose (TyG) index has gained recognition as a practical surrogate marker of insulin resistance and has demonstrated consistent associations with obesity and its metabolic complications [10]. Concurrently, hs-CRP is one of the most extensively used laboratory biomarkers for assessing systemic inflammatory status. The theoretical rationale for integrating these two biomarkers into a unified inflammation-metabolism composite index is that their combined assessment may better reflect the convergent pathophysiological processes underlying obesity. Preliminary studies have indicated that composite indices incorporating C-reactive protein, triglycerides, and glucose, such as the C-reactive protein-Triglyceride-Glucose index (CTI), are associated with metabolic syndrome and its individual components; however, their potential value in building clinically useful obesity risk prediction models remains underexplored [11,12].

Although various obesity risk assessment tools are currently available, most rely on questionnaires, family history, or complex body composition analyses, which may limit their feasibility for rapid implementation during routine health check-ups or large-scale screening [13]. Therefore, developing a quantitative risk assessment model based on readily available health examination variables, such as waist circumference and routine blood biochemical markers, while incorporating an advanced "inflammation-metabolism" composite indicator, holds significant practical importance for achieving early identification and risk stratification of individuals at increased risk of obesity. Consequently, this study aimed to develop and internally validate an adult obesity risk assessment model centered on CTI and incorporating routinely available clinical indicators using a retrospective observational design. The resulting model was presented as a user-friendly nomogram to provide clinicians and health managers with a practical tool for individualized obesity risk assessment and targeted prevention.

2. Methods

2.1 Study Population

This was a single-center retrospective observational study. Participants were adults who underwent routine health examinations at Shanghai Jinke Clinic between January 2022 and December 2024. All participants were of Asian ethnicity; no non-Asian individuals were included in this cohort. Individuals who had completed health examinations and had comprehensive data records were consecutively screened through the hospital electronic medical record system.

Inclusion criteria: (1) age ≥18 years; (2) complete data available. Exclusion criteria: (1) acute infection, active autoimmune disease, or malignancy; (2) severe hepatic or renal insufficiency; (3) major surgery, severe trauma, acute myocardial infarction, or stroke within the past 3 months; (4) diagnosed endocrine diseases significantly affecting weight or metabolism, such as diabetes (defined as fasting glucose ≥7.0 mmol/L or HbA1c ≥6.5% or self-reported use of antidiabetic medication), hyperthyroidism or hypothyroidism (based on TSH and FT4 levels), or Cushing's syndrome (based on clinical diagnosis records) [14]; (5) long-term use (continuous use for more than 3 months) of glucocorticoids, immunosuppressants, or prescription weight-loss medications.

A total of 360 subjects meeting all criteria were finally included. For participants with multiple health examinations during the study period, only the first complete record was included. To ensure reliability in model development and internal validation, all subjects were randomly divided into two independent datasets using computer-generated random numbers at a 7:3 ratio: the model development training set (n = 252) and the internal validation set (n = 108).

2.2 Data Collection

All predictor variable data were obtained from standardized anthropometric measurements and laboratory tests performed on the day of the health examination. Height and weight were measured with participants wearing light clothing and no shoes, using a calibrated stadiometer and electronic scale. BMI was calculated as weight (kg) divided by height squared (m^2). Waist circumference (WC) was measured at the midpoint between the lower costal margin and the iliac crest at the end of normal expiration, with participants standing upright and their feet shoulder-width apart. Two measurements were obtained to the nearest 0.1 cm, and the average value was recorded. Blood pressure was measured in the seated position after at least 5 minutes of rest using an automated oscillometric device. Two measurements were obtained 1 minute apart, and the mean value was recorded.

Outcome variable (obesity status): Obesity status was treated as a binary variable. Subjects with a body mass index (BMI) ≥30 kg/m^2 were classified as obese according to World Health Organization (WHO) criteria [15], facilitating international comparison of our findings with those of other studies.

Core predictor variable (inflammation-metabolism composite index, CTI): CTI was calculated from fasting blood measurements. High-sensitivity C-reactive protein (hs-CRP, mg/L) was measured by immunoturbidimetry, while triglyceride (TG, mmol/L) and fasting plasma glucose (FPG, mmol/L) were measured using enzymatic methods. For consistency of units, TG and FPG values were converted using the following factors: 1 mmol/L TG = 88.57 mg/dL and 1 mmol/L FPG = 18.02 mg/dL. The CTI was calculated as follows:

CTI = 0.412 × Ln(hs − CRP, mg/L) + Ln[TG(mg/dL) × FPG(mg/dL)/2] [16].

In this formula, 0.412 is a fixed constant established in the original definition of the CTI. This formula has been adopted in subsequent studies across diverse populations, including non-diabetic cohorts [17,18,19].

Demographic and anthropometric variables included age (years), sex (male/female), and waist circumference (cm). Clinical and laboratory variables included systolic and diastolic blood pressure (mmHg); lipid profile parameters, including total cholesterol, high-density lipoprotein cholesterol, and low-density lipoprotein cholesterol (mmol/L); and uric acid (μmol/L).

2.3 Statistical Analysis

All statistical analyses were performed using R software (version 4.5.2; R Core Team, Vienna, Austria). A two-sided p-value < 0.05 was considered statistically significant. The normality of continuous variables was assessed using the Shapiro-Wilk test. Normally distributed continuous variables are presented as mean ± standard deviation and were compared using the independent-samples t-test, while non-normally distributed variables are presented as median (Q_1, Q_3) and were compared using the Mann-Whitney U test. Categorical variables are presented as frequencies (percentages) and were compared using the chi-square test or Fisher's exact test as appropriate.

In the training set, candidate variables were first evaluated using univariate logistic regression analysis. Variables with $p < 0.05$ were subsequently entered into a multivariate logistic regression model using a bidirectional stepwise selection procedure to identify factors independently associated with obesity and establish the final prediction model. Multicollinearity was assessed using the variance inflation factor (VIF <5). Model discrimination was evaluated by calculating the area under the receiver operating characteristic curve (AUC) in both the training and validation sets. Calibration curves were plotted, and the Hosmer-Lemeshow goodness-of-fit test ($p > 0.05$ indicating good calibration) was performed. The optimal cutoff was determined using the Youden index, and the corresponding sensitivity, specificity, positive predictive value (PPV), negative predictive value (NPV), accuracy, and Brier score were calculated. Decision curve analysis (DCA) was performed to evaluate the clinical net benefit of the prediction model across a range of threshold probabilities. A visual nomogram was constructed using the regression coefficients from the final multivariate regression model. Internal validation was performed in the training set using the bootstrap method with 1000 resamples to estimate the optimism-corrected AUC.

To evaluate the incremental predictive value of CTI beyond waist circumference, three logistic regression models were constructed: Model 1, including waist circumference alone; Model 2, including CTI alone; and Model 3, including waist circumference and CTI. The area under the ROC curves (AUCs) was compared using the DeLong test. Additionally, thecategorical (classified) Net Reclassification Improvement (NRI) and Integrated Discrimination Improvement (IDI) were calculated using the PredictABEL package (version 4.5.2, R Core Team). For the sensitivity analysis based on the Chinese BMI criterion for obesity (BMI ≥28 kg/m^2), the same model development and validation procedures were repeated.

3. Results

3.1 Baseline Characteristics of the Study Population

A total of 360 subjects were included in the analysis, with an overall obesity prevalence of 17.22% according to WHO criteria. No statistically significant differences were observed between the training and validation sets in any baseline characteristics, including demographic, anthropometric, clinical, and laboratory variables (all $p > 0.05$), indicating comparable distributions between the two datasets (Table 1).

Compared with non-obese participants, obese participants had significantly higher waist circumference, CTI, TG, LDL-C, and hs-CRP levels, as well as a higher proportion of males (all $p < 0.05$), while age, blood pressure, FPG, HDL-C, TC, and UA were comparable between the two groups (Table 2).

3.2 Predictor Screening and Model Construction

Univariate logistic regression analysis in the training set (Table 3) showed that waist circumference (WC), CTI, and sex were significantly associated with the risk of obesity ($p < 0.05$). Sex was subsequently excluded during stepwise regression. Collinearity diagnostics showed VIF values <5 for both waist circumference and CTI, indicating no significant multicollinearity. Multivariate logistic regression analysis (Table 4) ultimately identified two variables as independently associated factors: waist circumference (WC) and CTI.

3.3 Model Performance Evaluation

3.3.1 ROC Analysis

As shown in Fig. 1 and Table 5, the model achieved an AUC of 0.802 (95% CI: 0.723–0.882) in the training set, with an optimism-corrected AUC of 0.795 after 1000 bootstrap resamples. In the 7:3 split validation set, the AUC was 0.857 (95% CI: 0.759–0.955); however, this estimate should be interpreted with caution, given the wide confidence interval and the limited number of obesity events (n = 16) in this subset. Therefore, the bootstrap-corrected AUC of 0.795 from the training set provides a more stable estimate of the discriminative performance of the model, while the validation set results should be considered preliminary and exploratory.

Based on the Youden index, the optimal cutoff was 0.151 in the training set, yielding a sensitivity of 0.804,

Table 1. Comparison of baseline characteristics between the training and validation sets.

Variables	Total (n = 360)	Train Set (n = 252)	Validation Set (n = 108)	Test Statistic	*p*-value
SBP (mmHg), mean ± SD	124.67 ± 12.55	125.11 ± 12.41	123.64 ± 12.88	$t = 1.02$	0.309
DBP (mmHg), mean ± SD	80.70 ± 7.33	81.02 ± 7.25	79.96 ± 7.52	$t = 1.25$	0.211
FPG (mmol/L), mean ± SD	5.39 ± 0.57	5.38 ± 0.57	5.40 ± 0.57	$t = –0.35$	0.728
CTI, mean ± SD	5.47 ± 1.16	5.42 ± 1.28	5.59 ± 0.81	$t = 1.50$	0.134
HDL-C (mmol/L), mean ± SD	1.37 ± 0.23	1.37 ± 0.24	1.37 ± 0.21	$t = 0.23$	0.819
LDL-C (mmol/L), mean ± SD	2.83 ± 0.65	2.82 ± 0.66	2.84 ± 0.60	$t = –0.32$	0.750
UA (μmol/L), mean ± SD	312.30 ± 54.76	314.02 ± 55.47	308.30 ± 53.11	$t = 0.91$	0.364
Age (years), median (Q_1, Q_3)	48.50 (42.00, 55.00)	48.00 (42.00, 55.00)	49.00 (41.00, 55.00)	$Z = –0.31$	0.756
Waist circumference (cm), median (Q_1, Q_3)	84.90 (79.28, 90.33)	84.95 (78.55, 90.63)	84.85 (80.30, 89.53)	$Z = –0.06$	0.949
hs-CRP (mg/L), median (Q_1, Q_3)	1.24 (0.92, 1.73)	1.23 (0.93, 1.70)	1.32 (0.92, 1.75)	$Z = –0.83$	0.408
BMI (kg/m^2), median (Q_1, Q_3)	27.00 (24.90, 29.20)	27.00 (24.87, 29.22)	26.90 (25.08, 29.13)	$Z = –0.35$	0.729
TG (mmol/L), median (Q_1, Q_3)	1.45 (1.14, 1.86)	1.45 (1.12, 1.90)	1.44 (1.17, 1.79)	$Z = –0.36$	0.722
TC (mmol/L), median (Q_1, Q_3)	4.86 (4.43, 5.38)	4.84 (4.41, 5.35)	4.89 (4.51, 5.43)	$Z = –0.93$	0.353
Sex, n (%)				$\chi^2 = 0.85$	0.358
Female	180 (50.00)	122 (48.41)	58 (53.70)		
Male	180 (50.00)	130 (51.59)	50 (46.30)		
Obesity, n (%)				$\chi^2 = 0.63$	0.428
No	298 (82.78)	206 (81.75)	92 (85.19)		
Yes	62 (17.22)	46 (18.25)	16 (14.81)		

Abbreviations: SBP, systolic blood pressure; DBP, diastolic blood pressure; FPG, fasting plasma glucose; TG, triglycerides; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; TC, total cholesterol; UA, uric acid; hs-CRP, high-sensitivity C-reactive protein; CTI, C-reactive protein-triglyceride-glucose composite index; BMI, body mass index.

Table 2. Baseline characteristics of obese and non-obese participants.

Variable	Total (n = 360)	Non-Obese (n = 298)	Obese (n = 62)	Test Statistic	*p*-value
Age (years), median (Q_1, Q_3)	48.50 (42.00, 55.00)	48.50 (42.00, 55.00)	48.50 (44.00, 55.75)	$Z = –0.72$	0.472
Sex, n (%)				$\chi^2 = 7.79$	0.005
Female	180 (50.00)	159 (53.36)	21 (33.87)		
Male	180 (50.00)	139 (46.64)	41 (66.13)		
Waist circumference (cm), median (Q_1, Q_3)	84.90 (79.28, 90.33)	83.45 (78.20, 88.68)	92.25 (86.03, 97.55)	$Z = –6.59$	<0.001
SBP (mmHg), mean ± SD	124.67 ± 12.55	124.91 ± 12.58	123.52 ± 12.45	$t = 0.79$	0.427
DBP (mmHg), mean ± SD	80.70 ± 7.33	80.92 ± 7.17	79.65 ± 8.05	$t = 1.16$	0.250
FPG (mmol/L), mean ± SD	5.39 ± 0.57	5.40 ± 0.56	5.32 ± 0.60	$t = 1.09$	0.275
hs-CRP (mg/L), median (Q_1, Q_3)	1.24 (0.92, 1.73)	1.22 (0.90, 1.65)	1.54 (1.00, 2.09)	$Z = –2.29$	0.022
BMI (kg/m^2), median (Q_1, Q_3)	27.00 (24.90, 29.20)	26.40 (24.52, 27.90)	36.05 (33.30, 38.00)	$Z = –12.39$	<0.001
TG (mmol/L), median (Q_1, Q_3)	1.45 (1.14, 1.86)	1.38 (1.11, 1.76)	1.99 (1.39, 2.64)	$Z = –5.37$	<0.001
HDL-C (mmol/L), mean ± SD	1.37 ± 0.23	1.38 ± 0.22	1.34 ± 0.26	$t = 1.12$	0.262
LDL-C (mmol/L), mean ± SD	2.83 ± 0.65	2.79 ± 0.64	3.01 ± 0.65	$t = –2.51$	0.013
TC (mmol/L), median (Q_1, Q_3)	4.86 (4.43, 5.38)	4.87 (4.44, 5.41)	4.82 (4.39, 5.28)	$Z = –0.81$	0.419
UA (μmol/L), mean ± SD	312.30 ± 54.76	311.38 ± 54.77	316.73 ± 54.91	$t = –0.70$	0.485
CTI, mean ± SD	5.47 ± 1.16	5.33 ± 1.02	6.17 ± 1.52	$t = –4.17$	<0.001

Abbreviations: SBP, systolic blood pressure; DBP, diastolic blood pressure; FPG, fasting plasma glucose; TG, triglycerides; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; TC, total cholesterol; UA, uric acid; CTI, C-reactive protein-triglyceride-glucose composite index; hs-CRP, high-sensitivity C-reactive protein; BMI, body mass index.

specificity of 0.684, PPV of 0.363, NPV of 0.940, and accuracy of 0.706 (Youden index = 0.489). Applying this fixed cutoff to the validation set yielded a sensitivity of 0.875, specificity of 0.674, PPV of 0.318, NPV of 0.969, and accuracy of 0.704 (Youden index = 0.549). The full logistic regression equation was: logit(P) = –14.223 + 0.111 × Waist circumference (cm) + 0.521 × CTI. The higher AUC observed in the validation set than in the training set may reflect sampling variation due to the relatively small validation sample size. Nevertheless, both AUC estimates ex-

Table 3. Univariate logistic regression analysis of factors associated with obesity in the training set.

Predictor	β	SE	Wald	*p*-value	OR (95% CI)
Age	–0.003	0.019	0.031	0.859	0.997 (0.961–1.034)
Sex	–0.687	0.340	4.093	0.043	0.503 (0.259–0.979)
Waist circumference	0.112	0.021	29.919	<0.001	1.119 (1.075–1.165)
SBP	–0.020	0.014	2.121	0.145	0.981 (0.955–1.007)
DBP	–0.027	0.023	1.378	0.234	0.973 (0.93–1.018)
hs-CRP	0.287	0.220	1.702	0.191	1.332 (0.866–2.049)
CTI	0.552	0.137	16.270	<0.001	1.736 (1.328–2.27)
HDL-C	–0.757	0.690	1.205	0.272	0.469 (0.121–1.813)
TC	0.229	0.249	0.845	0.358	1.257 (0.772–2.047)
LDL-C	0.477	0.248	3.695	0.055	1.612 (0.991–2.623)
UA	0.002	0.003	0.364	0.546	1.002 (0.996–1.008)

Abbreviations: SBP, systolic blood pressure; DBP, diastolic blood pressure; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; TC, total cholesterol; UA, uric acid; hs-CRP, high-sensitivity C-reactive protein; CTI, C-reactive protein-triglyceride-glucose composite index; β, regression coefficient; OR, odds ratio; SE, standard error; CI, confidence interval. Sex was coded as male = 0 (reference) and female = 1.

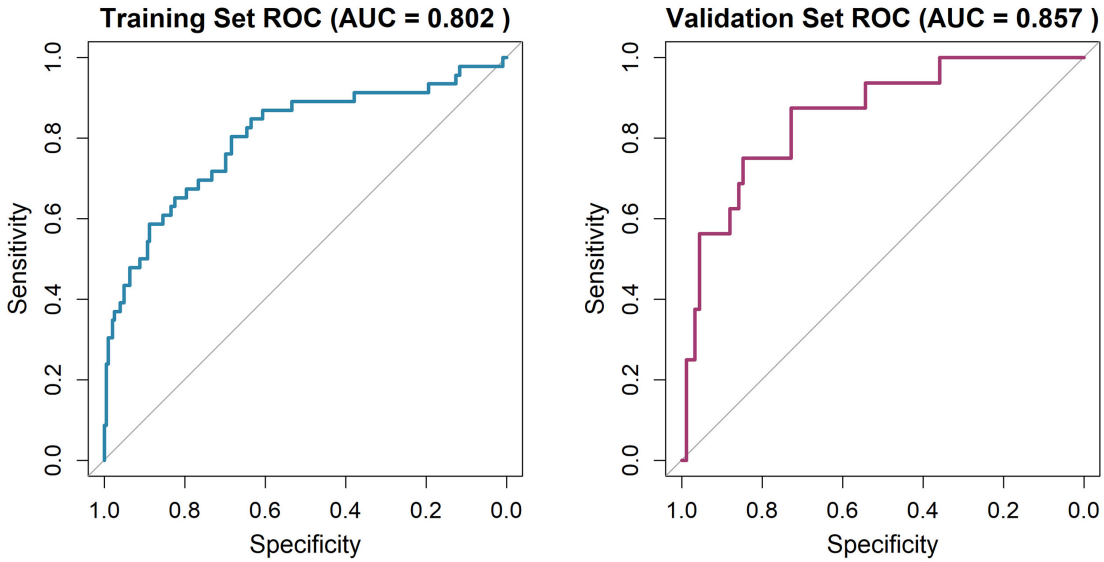


Fig. 1. Receiver operating characteristic curves of the model in the training and validation sets.

Table 4. Multivariate logistic regression analysis of the final model in the training set.

Predictor	β	SE	Wald	*p*-value	OR (95% CI)
Waist circumference	0.111	0.021	27.939	<0.001	1.117 (1.072–1.165)
CTI	0.521	0.147	12.561	<0.001	1.684 (1.262–2.246)

Abbreviations: β, regression coefficient; CI, confidence interval; CTI, C-reactive protein-triglyceride-glucose composite index; OR, odds ratio; SE, standard error.

ceeded 0.7, indicating acceptable to good discriminative performance.

3.3.2 Calibration

In the training set, the calibration curve (Fig. 2) showed good agreement between predicted probabilities and observed outcomes. In the validation set (Fig. 2), the calibration curve exhibited some fluctuation at higher predicted-risk ranges, likely reflecting the small sample size and limited number of obesity events (n = 16), warranting cautious interpretation. The Hosmer-Lemeshow test *p*-values were >0.05 for both sets (training: $\chi^2 = 9.17$, $p = 0.241$; validation: $\chi^2 = 3.81$, $p = 0.282$), indicating that no statistically significant lack of fit was detected. However, these results do not establish perfect calibration. The Brier score was 0.147 for the training set and 0.186 in the validation set. The calibration intercept and slope were –0.000

Table 5. Model performance at the optimal cutoff determined by the Youden index.

Dataset	Cutoff	Sensitivity	Specificity	PPV	NPV	Accuracy	Youden Index	AUC (95% CI)
Training set	0.151	0.804	0.684	0.363	0.940	0.706	0.489	0.802 (0.723–0.882)
Validation set	0.151	0.875	0.674	0.318	0.969	0.704	0.549	0.857 (0.759–0.955)

Abbreviations: PPV, positive predictive value; NPV, negative predictive value; AUC, area under the ROC curve; CI, confidence interval.

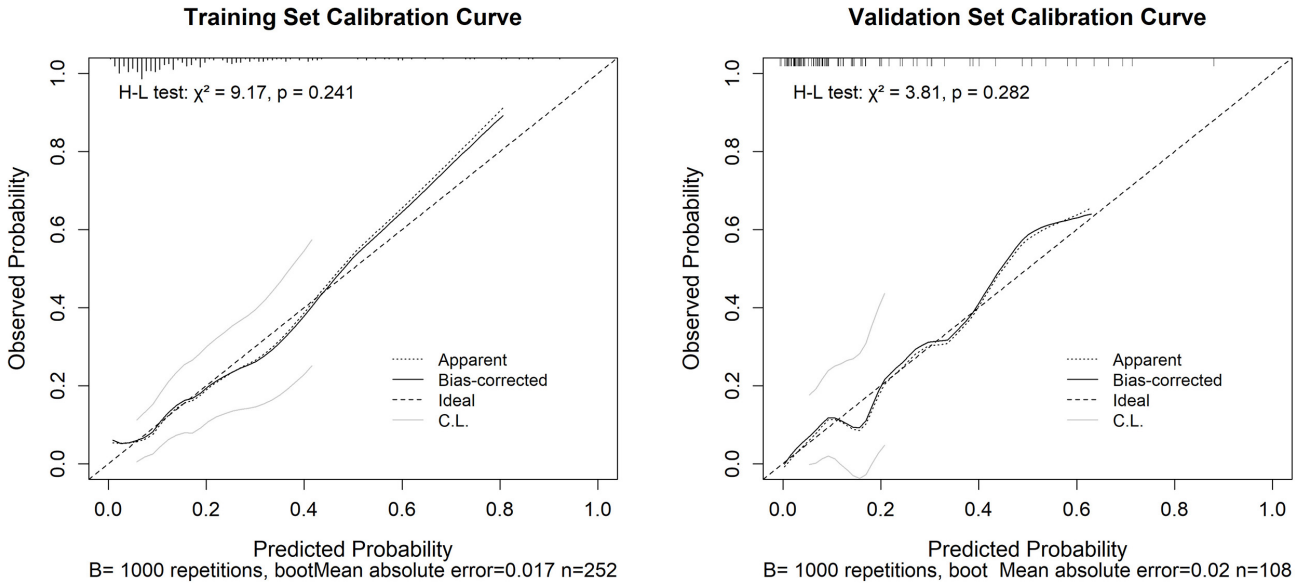


Fig. 2. Calibration curves of the obesity risk prediction model.

(95% CI: –0.526 to 0.552) and 1.000 (95% CI: 0.697 to 1.345), respectively, in the training set, and 0.860 (95% CI: –0.351 to 2.200) and 1.753 (95% CI: 0.948 to 2.759), respectively, in the validation set. The calibration slope was 1.753 in the validation set, indicating under-dispersion of predicted risks and suboptimal calibration, despite a non-significant Hosmer–Lemeshow test.

3.3.3 Decision Curve Analysis

Decision curve analysis (DCA) (Fig. 3) demonstrated that, across a threshold probability range of approximately 10% to 65%, use of the model provided a greater clinical net benefit than the two extreme strategies of "intervention for all" (i.e., providing intervention to everyone) and "intervention for none" (i.e., providing intervention to no one).

3.3.4 Prediction Nomogram

Based on the final multivariate logistic regression model in the training set (n = 252), a visual nomogram incorporating the two predictors, waist circumference and CTI, was constructed (Fig. 4). By locating an individual's waist circumference and CTI values on the corresponding axes to obtain the respective points, summing these points yields a total score. The corresponding probability of obesity can then be read from the total risk axis, thereby enabling individualized quantitative risk assessment.

3.3.5 Incremental Value of CTI

To assess whether CTI provides additional predictive information beyond waist circumference, we compared three logistic regression models: Model 1 (waist circumference only), Model 2 (CTI only), and Model 3 (waist circumference + CTI). The combined model (Model 3) achieved an AUC of 0.802, compared with 0.760 for the WC-only model and 0.669 for the CTI-only model (Table 6). The DeLong test showed no statistically significant difference between the combined and WC-only models (ΔAUC = 0.043, 95% CI: –0.019 to 0.105, $p = 0.172$).

However, the Net Reclassification Improvement (NRI) was 0.242 (95% CI: 0.068 to 0.416, $p = 0.006$), indicating an overall improvement in risk classification after the addition of CTI. The Integrated Discrimination Improvement (IDI) was 0.072 (95% CI: 0.027 to 0.117, $p = 0.002$), further supporting the incremental discriminatory value of CTI (Table 6).

These findings suggest that although the overall improvement in AUC was modest and did not reach statistical significance, CTI may provide complementary information for individual risk stratification, capturing inflammatory-metabolic signals beyond those reflected by waist circumference alone.

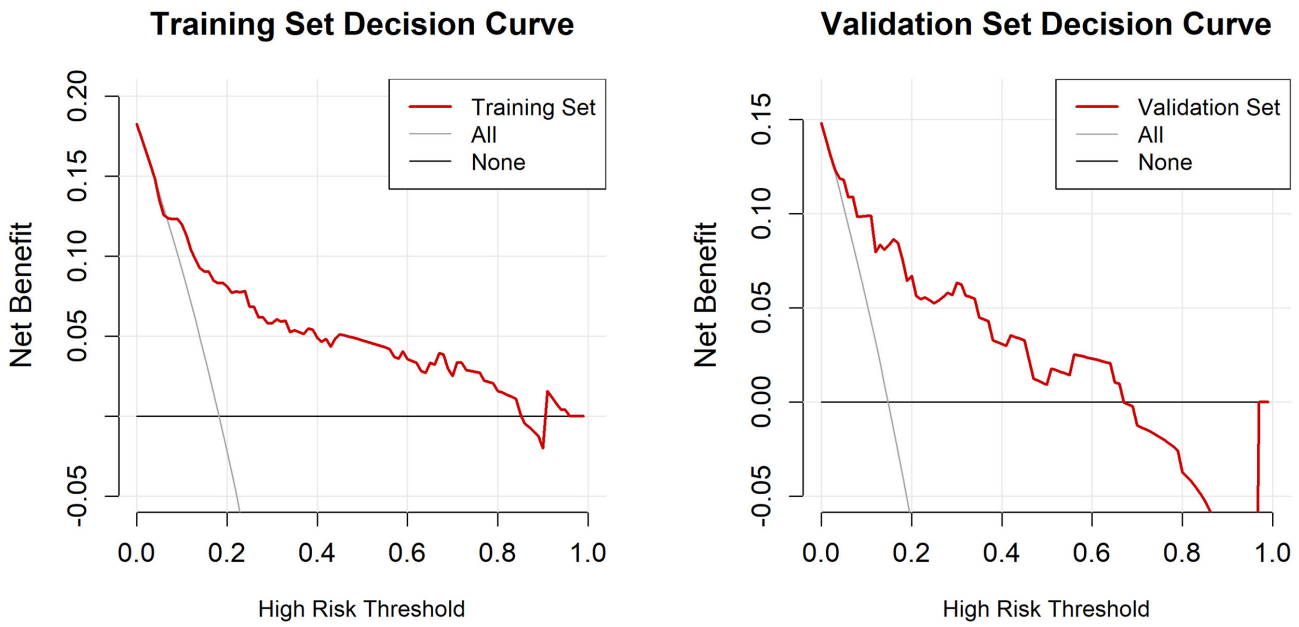


Fig. 3. Decision curve analysis of the obesity risk prediction model.

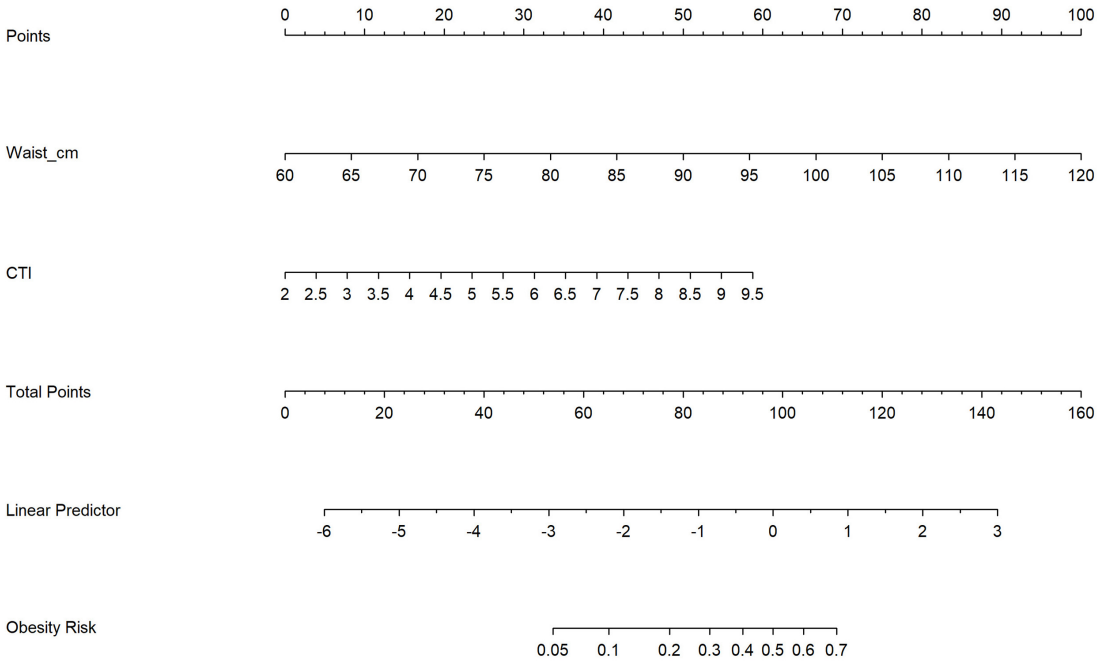


Fig. 4. Nomogram for predicting the risk of obesity. Note: The nomogram was constructed based on the final multivariate logistic regression model from the training set (n = 252). Points, point scale for each predictor; Waist_cm, waist circumference in cm; CTI, C-reactive protein–triglyceride–glucose composite index; Total Points, sum of points from all predictors; Obesity Risk, predicted probability of obesity.

3.3.6 Sensitivity Analysis Using Chinese BMI Criteria

To evaluate the robustness of our model across different obesity definitions, we repeated the analysis using the Chinese-specific BMI cutoff of ≥28 kg/m^2. Multivariate logistic regression again identified waist circumference (OR = 1.154, 95% CI: 1.106–1.205, $p < 0.001$) and CTI (OR = 1.445, 95% CI: 1.132–1.845, $p = 0.003$) as independent predictors. The model achieved an AUC of 0.808 in the training set and 0.795 in the validation set, with a bootstrap-corrected AUC of 0.806. These results were consistent with those of the primary analysis, supporting the robustness of the model across different obesity definitions (Table 7, Fig. 5).

Table 6. Incremental value of CTI over waist circumference alone.

Model	AUC	ΔAUC (95% CI)	*p*-value	NRI (95% CI)	*p*-value	IDI (95% CI)	*p*-value
WC-only	0.760	Reference					
CTI-only	0.669	–0.091 (–0.137 to –0.045)	<0.001				
Combined (WC + CTI)	0.802	0.043 (–0.019 to 0.105)	0.172	0.242 (0.068 to 0.416)	0.006	0.072 (0.027 to 0.117)	0.002

Abbreviations: AUC, area under the ROC curve; CI, confidence interval; CTI, C-reactive protein-triglyceride-glucose composite index; ΔAUC, difference in AUC compared to the WC-only model; NRI, net reclassification improvement; IDI, integrated discrimination improvement; WC, waist circumference.

Table 7. Sensitivity analysis using the Chinese BMI criteria.

Predictor	β	SE	Wald	*p*-value	OR (95% CI)
Waist circumference	0.143	0.022	43.137	<0.001	1.154 (1.106–1.205)
CTI	0.368	0.125	8.730	0.003	1.445 (1.132–1.845)

Abbreviations: β, regression coefficient; BMI, body mass index; CI, confidence interval; CTI, C-reactive protein-triglyceride-glucose composite index; OR, odds ratio; SE, standard error.

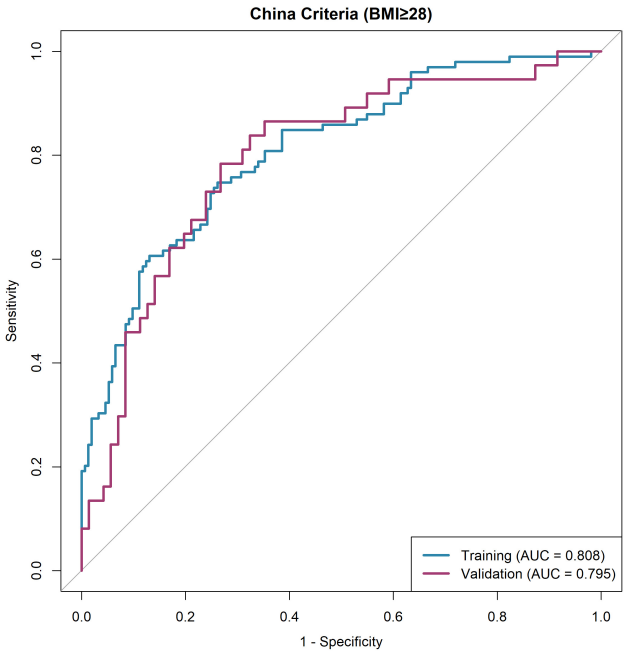


Fig. 5. Receiver operating characteristic curves of the model in the sensitivity analysis.

4. Discussion

The core of this study lies in exploring the significance of a composite index that integrates inflammatory and metabolic signals, namely CTI, for assessing obesity status. Our findings indicate that the combined use of CTI and waist circumference provides information beyond that captured by traditional anthropometric measures. This observation suggests that obesity may involve not only the quantity and distribution of adipose tissue but also alterations in adipose tissue function.

The contemporary view of obesity positions it as a chronic inflammatory condition associated with adipose tissue dysfunction and concurrent systemic metabolic derangements [20,21]. In this state, hypertrophied and stressed adipocytes exhibit altered secretory profiles, releasing elevated quantities of free fatty acids and pro-inflammatory adipokines while producing lower levels of protective factors such as adiponectin. These changes collectively contribute to the development of insulin resistance [22,23,24]. Among the downstream consequences, hepatic synthesis of hs-CRP, a prototypical acute-phase reactant, is upregulated, supporting the use of hs-CRP as a reliable peripheral marker of obesity-related inflammatory activity [25].

Under conditions of adipocyte hypertrophy and hypoxia, adipose tissue may recruit and interact with infiltrating immune cells, particularly pro-inflammatory macrophages. This cellular crosstalk establishes a paracrine signaling network that can amplify local and systemic inflammatory responses [26,27]. The resulting inflammatory mediators may, in turn, interfere with insulin receptor signaling and contribute to a feed-forward process in which metabolic dysfunction and inflammation synergistically reinforce one another [26,28].

The CTI employed in this investigation was designed to capture three distinct components of the obesity-related metabolic-inflammatory axis: hs-CRP as a marker of systemic inflammation, triglycerides as an indicator of lipid dysmetabolism, and fasting glucose as a marker of glycemic status and a component of the assessment of insulin resistance. We propose that CTI may serve as an exploratory composite marker of systemic inflammatory-metabolic status associated with obesity. However, this interpretation remains hypothetical and cannot be established directly from the present observational data. Further studies incorporating direct assessments of adipose tissue function, reference-standard measures of insulin sensitivity, or imaging-based quantification of fat distribution are required to determine whether CTI accurately reflects adipose tissue dysfunction or specific pathophysiological processes. Our finding that CTI remained independently associated with obesity even after adjustment for waist circumference, the morphological indicator of central obesity, suggests that obesity risk may depend not only on the amount and distribution of fat but also on the functional state of adipose tissue. This find-

ing may provide a mechanistic explanation for the clinical heterogeneity observed in obesity.

Our incremental value analysis revealed an important nuance: while the addition of CTI to waist circumference did not significantly increase the AUC (ΔAUC = 0.043, p = 0.172), the significant NRI (0.242, p = 0.006) and IDI (0.072, p = 0.002) suggested improved risk classification and discrimination after CTI was incorporated into the model. We acknowledge that the incremental value of CTI is modest, and its clinical application should be considered complementary to waist circumference rather than a replacement. The potential value of CTI may extend beyond obesity identification to reflect underlying inflammatory-metabolic dysregulation, a hypothesis that warrants future investigation. This apparent discrepancy may reflect the fact that AUC is a global measure of discrimination that can be relatively insensitive to modest improvements in risk stratification, whereas NRI and IDI directly assess the proportion of individuals correctly reclassified into more appropriate risk categories [29]. Additionally, although waist circumference and BMI are conceptually related measures of adiposity, our model uses waist circumference as an indicator of central obesity. CTI remained independently associated with obesity after adjustment for waist circumference, suggesting that CTI may capture additional inflammatory-metabolic information. In clinical practice, even without a substantial improvement in overall AUC, improved risk classification may have potential practical value for guiding clinical decision-making, particularly in primary prevention settings where accurate risk stratification is paramount.

The algorithm underlying this prediction model is readily adaptable for integration into electronic health records or smartphone-based health platforms, potentially facilitating automated risk estimation and clinical alerts. Such integration could support population-level risk screening and stratified prevention strategies at relatively low operational cost [30]. However, successful translation into clinical practice also faces several challenges, including the standardization of hs-CRP assays across institutions and the need for implementation studies to determine its feasibility, real-world clinical utility, and potential impact on clinical decision-making and patient outcomes [29].

5. Limitation

This study has several limitations. First, the single-center retrospective design and limited sample size may restrict the generalizability of the model to other populations and healthcare settings. Furthermore, as the model was developed in a non-diabetic population, its applicability to individuals with diabetes requires further investigation. Second, the weighting coefficients in the CTI formula were derived from a specific study, and their applicability across different populations requires further validation. Third, the model does not incorporate key variables such as lifestyle factors and genetic background, which may limit its predictive performance and broader applicability. Future work could consider developing a more integrated predictive system that utilizes multi-omics data. Future research directions should focus on: (1) conducting multicenter prospective cohort studies for external validation; and (2) conducting prospective intervention studies based on the risk stratification results of this model to evaluate its potential effects on metabolic health outcomes in high-risk populations.

6. Conclusion

This study developed an exploratory model for identifying obesity that integrates waist circumference and CTI. CTI, as a composite index of inflammation and metabolism, may provide additional information for identifying individuals with obesity and may be associated with concomitant inflammatory-metabolic dysregulation. However, the present study did not classify obesity subtypes, and this exploratory finding requires further validation. Given the single-center retrospective design, the proposed model should therefore be considered exploratory. Future multicenter prospective studies are warranted to externally validate the model before consideration of routine clinical implementation.

Key Points

• This study developed and internally validated a risk prediction model incorporating waist circumference and the C-reactive protein-triglyceride-glucose composite index (CTI) for identifying obesity in adults undergoing routine health examinations.

• The CTI, a composite biomarker integrating inflammatory (hs-CRP) and metabolic (triglycerides and fasting glucose) signals, was independently associated with obesity even after adjustment for waist circumference.

• Sensitivity analysis using Chinese BMI criteria (≥28 kg/m^2) yielded consistent results, supporting the robustness of the model across different obesity definitions in this Asian population.

Availability of Data and Materials

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

Author Contributions

LX and JZ designed the research study. LX, CLZ, and JZ performed the research. CLZ and JZ analyzed the data. JZ drafted the manuscript. All authors contributed to important editorial changes in the manuscript. All authors read and approved the final manuscript. All authors participated sufficiently in the work and agreed to be accountable for all aspects of the work.

Ethics Approval and Consent to Participate

The study protocol was approved by the Ethics Review Committee of Shanghai Jinke Clinic (Approval No.2025-01). As a retrospective data analysis, all data were anonymized prior to analysis, and the ethics committee waived the requirement for individual informed consent. The study was conducted in accordance with the principles of the Declaration of Helsinki.

Acknowledgment

Not applicable.

Funding

This research received no external funding.

Conflicts of Interest

Changlin Zhou was an employee of Shanghai Jinke Clinic. This organization was not involved in the conception and design of this study, data collection, analysis, interpretation, paper writing, or submission decisions. The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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