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A novel nomogram for predicting optimal weight loss response following diet and exercise intervention in patients with obesity

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This study aimed to identify factors associated with optimal weight loss response by analyzing pre-weight loss data from a cohort of 2577 patients with obesity who visited weight management clinics between 2013 and 2022. Out of these, 1276 patients had follow-up data available. Following dietary and exercise interventions, 580 participants achieved optimal weight loss outcomes. Participants were subsequently divided into two groups based on their weight loss outcomes: those who achieved optimal weight loss response and those who did not. Statistical analysis, conducted using RStudio, identified thirteen predictor variables through LASSO and logistic regression, with age emerging as the most influential predictor. A nomogram was developed to predict optimal weight loss response, showing good predictive performance (AUC = 0.807) and clinical applicability, validated by internal validation methods. Decision curve analysis (DCA) further illustrated the nomogram's clinical utility. The developed nomogram prediction model for optimal weight loss response is user-friendly, highly accurate, and demonstrates excellent discriminative and calibration capabilities.

Keywords Obesity, Weight loss, Nomogram, Prediction model

Obesity is a chronic metabolic disease influenced by genetic and environmental factors, characterized by excessive total body fat and/or abnormal fat distribution[1]. The global prevalence of obesity has nearly tripled over the past 40 years. In 2016, the World Health Organization estimated that 1.9 billion adults and over 340 million children and adolescents aged 5–19 were overweight or obese[2]. The World Obesity Federation predicts that by 2030, approximately one billion people worldwide will be obese, including one in five women and one in seven men[3]. Obesity is associated with a higher risk of early death and increased overall mortality[4]. Furthermore, the mass effect of excess adipose tissue and its direct metabolic effects make obesity a risk factor for various chronic diseases, including diabetes, stroke, coronary artery disease, hypertension, respiratory disease, and obstructive sleep apnea[5–7]. Obesity is also linked to the development of various tumors[8]. Additionally, obesity has well-documented adverse psychological and social consequences. Multiple studies have identified over 200 comorbidities associated with obesity, including type 2 diabetes, cardiovascular diseases, and certain cancers[9]. Research indicates that even a modest weight loss of 5–10% of total body weight can significantly improve these comorbidities[10]. This degree of weight loss can enhance blood sugar control, reduce blood pressure, and improve cholesterol levels, thereby lowering the risk of associated diseases[11].

Approaches to weight loss include lifestyle modifications, dietary adjustments, high-intensity physical activity, pharmacotherapy, and surgery[12]. The cornerstone of therapy is lifestyle intervention, which is resource-intensive and challenging for many people to sustain over time[13]. Additionally, the body's "energy compensation," where increased physical activity leads to increased appetite and calorie intake, often diminishes the effectiveness of exercise alone for weight loss in patients with obesity compared to comprehensive lifestyle interventions

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that include both diet and exercise modifications[14]. Pharmacotherapy for weight loss has lagged and is often inaccessible[15]. The use of minimally invasive bariatric surgery has increased, but not all patients are suitable candidates or desire surgery[16]. Ultimately, a multifaceted approach is needed to optimize disease control for all patients with obesity.

With reliable methods to identify individuals likely to have an optimal response to weight loss, clinicians can implement more comprehensive interventions earlier, thereby improving the rates of optimal weight loss responders. While some weight loss prediction models exist[17,18], there is a lack of such models specifically for Asians. Based on data from weight loss clinics in Nanjing, China, this study constructed an prediction model aimed at identifying early characteristics of individuals prone to optimal weight loss response before they begin, thereby guiding more effective interventions.

Methods

Study design

This study employed a case–control design to develop a risk model for predicting optimal weight loss responders. The sample size was calculated considering 12 expected predictors, a power of 50%, and a significance level of 0.05. At least 1170 cases were needed, with 487 samples required to have outcome events. To account for potential loss to follow-up and missing data, a total of 1276 participants were included, with 580 achieving optimal weight loss. The sample size was calculated using an interactive tool (https://mvansmeden.shinyapps.io/BeyondEPV/)[19].

This study utilized data from 2577 patients with obesity who visited weight management clinics between 2013 and 2022. A subset of 1276 patients, each with at least one follow-up visit after 3 months, was selected for detailed analysis. Participants provided written informed consent for data use, and the study was conducted in accordance with the Declaration of Helsinki. The study protocol was approved by the Institutional Review Board of Nanjing Drum Tower Hospital (Ethics No. 2015-099-01).

Inclusion criteria: Adults aged 18–65 years with a BMI ≥ 24 kg/m^2 who visited weight management clinics between 2013 and 2022, and had at least one follow-up visit after 3 months. Exclusion criteria: Patients with secondary causes of obesity (e.g., hypothyroidism, Cushing's syndrome), those who underwent bariatric surgery or used weight loss medication within 6 months before enrollment, those with severe comorbid conditions such as advanced cardiovascular disease, uncontrolled diabetes, or other significant medical conditions that could independently affect weight loss outcomes, those with poorly controlled mental disorders, alcoholism, or drug abuse history, and pregnant or breastfeeding women.

Study treatment

Calorie-restricted diet (CRD) mode. CRD is a dietary mode that guarantees basic nutritional needs while limiting energy intake. Its macronutrient energy supply ratio aligns with balanced diet requirements: protein 20%, fat 30%, and carbohydrate 50%. Energy provision is based on the Katch-McArdle formula to estimate basal metabolic rate (BMR), BMR = 370 + 21.6 × lean body mass, and recommended energy intake (Kcal) = (1.2 or 1.3) × BMR − 500. Carbohydrates should come from low glycemic index foods, mainly complex carbohydrates, with coarse grains making up half of the staple food. Participants should consume 500 g of vegetables daily (half leafy), 200 g of fruit, ensuring dietary fiber intake of 25–30 g/day, and strictly limit simple sugars (monosaccharides, disaccharides). Patients are encouraged to choose healthy fats (monounsaturated and polyunsaturated fats) and proteins (fish, nuts, beans, and poultry). Patients were advised to engage in 30 to 60 min of moderate-intensity aerobic exercise daily and perform resistance exercises 2 to 3 times a week for 10 to 20 min each session. Metformin was provided to some patients with impaired glucose tolerance. After the initial visit, patients followed up by telephone every two weeks and visited the clinic every four weeks, keeping a diet and exercise diary and interacting with the doctor through the Big Doctor Internet + Medical Platform, telephone, text messages, and WeChat.

Study end points

The primary endpoint was achieving at least 5% total weight loss (TWL) from baseline during the 3-month follow-up, defined as optimal weight loss responder[20]. Secondary endpoints included improvements in metabolic indicators such as blood glucose and blood lipids.

Study hypothesizes

The primary hypothesis posited that specific pre-weight loss factors could predict optimal weight loss responder. Secondary hypotheses aimed to identify potential demographic and clinical variables affecting weight loss outcomes.

Data preprocessing

First, data from 1276 patients were extracted from 2577 weight loss patients. Variables of interest included age, height, weight, BMI, waist-to-hip ratio, obstetric history, diabetes history, hypertension history, alcohol consumption history, hypothyroidism, anxiety score, depression score, age at menarche, menstrual abnormality, hirsutism, acne, hair loss, galactorrhea, acanthosis nigricans, polycystic ovary (PCO), fatty liver, blood pressure, blood glucose, insulin, hemoglobin A1c (HbA1c), thyroid-stimulating hormone (TSH), free triiodothyronine (FT3), free thyroxine (FT4), thyroglobulin antibody (TgAb), alanine aminotransferase (ALT), aspartate aminotransferase (AST), γ-glutamyl transferase (γ-GT), total bilirubin (TBIL), direct bilirubin (DBIL), uric acid (UA), blood urea nitrogen (BUN), serum creatinine (SCr), triglycerides (TG), total cholesterol (TC), high-density lipoprotein (HDL), low-density lipoprotein (LDL), apoprotein (Apo), calcium (Ca), dehydroepiandrosterone sulfate (DHEAS), sex hormone-binding globulin (SHBG), adrenocorticotropic hormone (ACTH) at 8:00 AM, cortisol (F) at 8:00 AM, vitamin D, albumin, c-reactive protein (CRP), and metformin treatment regimen (0:

without; 1: with metformin). Anxiety and depression scores were derived from the GAD-7 and PHQ-9 scales, respectively[21,22], and PCO and fatty liver were diagnosed by ultrasound. Through dietary and exercise interventions, patients with impaired glucose tolerance or T2DM were treated with metformin. After 3 months to 1 year of follow-up, 580 participants achieved 5%TWL or more, which were considered optimal weight loss responders. Based on this result, we divided the patients into two groups: the optimal weight loss responder group (TWL ≥ 5%) and the non-optimal weight loss responder group (TWL < 5%).

Statistical analysis

The methods described here were reported previously[23] and are briefly outlined below. This study used RStudio (https://www.rstudio.com) for all statistical analyses, with all data expressed as follows. Participant characteristics were expressed as mean (SD) for continuous variables and frequency (percentage) for categorical variables. One-way ANOVA and Kruskal–Wallis tests were used to analyze differences in normally and skewed continuous variables, respectively, while chi-squared tests were used for categorical variables. The least absolute shrinkage and selection operator (LASSO) regression was used to identify the most predictive variables from the dataset. Logistic regression analysis was then conducted to construct a predictive model for optimal weight loss responders. Based on this model, a nomogram was created.

Internal validation of the nomogram's performance was conducted using bootstrap resampling (500 resamples). The nomogram's predictive accuracy was assessed using the area under the receiver operating characteristic curve (AUC). Additionally, decision curve analysis (DCA) was used to evaluate the clinical utility of the nomogram. DCA quantifies the net benefit at different threshold probabilities by comparing the nomogram's performance against two reference lines: one assuming all patients are optimal weight loss responders and receive the intervention (representing the highest clinical cost) and one assuming no patients are optimal weight loss responders (representing no clinical benefit).

Results

Baseline characteristics

Of the 1276 patients in the cohort who had follow-up, 580 were classified as optimal weight loss responders. Table 1 presents the baseline data for the 580 optimal weight loss responders and 696 non-optimal weight loss responders. At baseline, significant differences were observed between the two groups in weight, BMI, fasting blood glucose, fasting insulin, 30-min glucose, 60-min glucose, HOMA-IR, HbA1c, Apo-B, and CRP. These differences highlight the necessity for a personalized approach in weight loss interventions.

Development of role selection and personalization prediction models

To identify key predictors of optimal weight loss response, we initially considered 61 features. Using the Lasso regression model, we reduced this number to 8 potential predictors with nonzero coefficients: hirsutism, hair loss, BMI, blood glucose at 0 min, insulin at 0 min, blood glucose at 60 min, ALT, and Ca (Fig. 1A,B).

Additionally, backward logistic regression analysis was employed, which identified 9 predictor variables: blood glucose at 0 min, ALT, TC, LDLC, Ca, F-8:00, HOMA β, hirsutism, and hair loss. By combining the results of both screening methods, we identified 12 predictor variables. Including age as an additional factor, we finalized 13 predictor variables (Table 2). These variables were used to develop a nomogram for predicting optimal weight loss response, shown in Fig. 2.

Nomogram performance

The nomogram calibration curve showed good agreement across the cohort (Fig. 3A). Using a bootstrap sampling method for internal validation, we found that the AUC of the nomogram was 0.807 (95% CI 0.736–0.868), indicating that the model has good predictive power (Fig. 3B).

Nomogram decision curve

Decision curve analysis (DCA) further validated the clinical utility of the nomogram. As depicted in Fig. 4, the DCA results show that the nomogram provides a higher net benefit across a range of threshold probabilities compared to the reference curves. The nomogram curve being positioned farther from the reference lines suggests that it offers better clinical benefit. These findings indicate that the nomogram can effectively guide clinical decision-making for weight loss interventions.

Discussion

Obesity is a significant global health issue[9]. Given the generally low rate of achieving optimal weight loss through diet and exercise alone, alternative and more comprehensive strategies are often required to achieve and sustain significant weight loss. In this study, we developed a nomogram to predict optimal weight loss responders using pre-weight loss data.

Validation of the nomogram demonstrated its strong discriminatory ability and calibration capabilities. Furthermore, the prediction model for optimal weight loss responder constructed in this study can be applied before weight loss attempts begin, thus providing more individualized weight loss guidance for individuals at different risk levels and potentially improving the rates of optimal weight loss responder.

Obesity is associated with an increased risk of type 2 diabetes, cardiovascular disease, certain cancers, and premature death[24]. In addition to adverse health outcomes, obesity also impacts the healthcare system, creating direct healthcare costs as well as indirect costs such as lost productivity[25]. Once weight is gained, it is extremely difficult to lose, with only 40% of those who attempt weight loss achieving ≥ 5% weight loss and 20%

Characteristic	Optimal weight loss responder (n = 696)	Non-optimal weight loss responder (n = 580)	*P* value
Age (year)	30.38 (4.442)	30.37 (4.239)	0.996
Height (cm)	160.32 (5.714)	159.77 (5.670)	0.449
Weight (kg)	80.89 (11.032)	77.06 (7.858)	0.003
BMI (kg/m^2)	31.47 (3.940)	30.25 (2.690)	0.009
Waist to hip (%)	0.96 (0.053)	0.95 (0.048)	0.094
Obstetric history	42 (43.75%)	79 (43.89%)	0.982
Diabetes history	1 (1.04%)	4 (2.22%)	0.492
Hypertension history	1 (1.04%)	4 (2.22%)	0.492
Alcohol consumption history	2 (2.08%)	6 (3.33%)	0.566
Hypothyroidism	14 (14.58%)	19 (10.56%)	0.328
Anxiety score	4.65 (4.693)	4.09 (4.036)	0.309
Depression score	4.08 (4.010)	3.80 (3.770)	0.568
Age at merche (year)	13.60 (1.475)	13.47 (1.442)	0.484
Menstrual abnormality	18 (18.75%)	37 (20.56%)	0.722
Hirsutism	16 (16.67%)	39 (21.67%)	0.310
Acne	7 (7.29%)	16 (8.89%)	0.649
Hair loss	25 (26.04%)	30 (16.67%)	0.072
Galactorrhea	2 (2.08%)	2 (1.11%)	0.515
Acanthosis nigricans	6 (6.25%)	10 (5.56%)	0.807
PCO	3 (3.13%)	2 (1.11%)	0.296
Fatty liver	60 (62.50%)	107 (59.44%)	0.796
SBP (mmHg)	124.11 (11.337)	122.92 (14.097)	0.458
DBP (mmHg)	82.27 (10.037)	80.78 (10.836)	0.275
Blood glucose during OGTT (mg/dL)			
0 min	5.72 (1.751)	5.32 (1.005)	0.017
30 min	9.31 (2.400)	8.55 (1.739)	0.003
60 min	9.70 (3.139)	8.86 (2.701)	0.026
120 min	8.07 (3.457)	7.56 (2.714)	0.180
Blood insulin during OGTT (uU/mL)			
0 min	23.86 (11.526)	19.51 (9.508)	0.000
30 min	119.96 (68.958)	121.80 (87.079)	0.861
60 min	148.55 (89.051)	132.42 (87.905)	0.159
120 min	148.45 (99.466)	129.084 (107.751)	0.160
HOMA IR	6.50 (5.94)	4.73 (2.84)	0.001
HOMA β	250.98 (120.77)	193.45 (493.88)	0.177
HbA1c (%)	5.697 (1.176)	5.43 (0.650)	0.049
TSH (mIU/L)	3.21 (1.743)	3.13 (2.840)	0.811
FT3 (pmol/L)	4.89 (0.849)	4.99 (0.453)	0.308
FT4 (pmol/L)	17.03 (2.288)	17.04 (2.536)	0.974
TgAb (IU/mL)	16.30 (13.497)	19.43 (25.709)	0.266
ALT (U/L)	43.99 (29.761)	37.50 (33.030)	0.111
AST (U/L)	22.59 (7.495)	22.90 (14.354)	0.843
γ-GT (U/L)	72.28 (20.911)	69.44 (25.941)	0.361
TBIL (umol/L)	11.37 (17.704)	9.99 (6.219)	0.351
DBIL (umol/L)	2.84 (1.940)	3.38 (8.185)	0.530
UA (umol/L)	374.43 (78.694)	369.93 (87.704)	0.676
BUN (mmol/L)	4.50 (1.099)	4.56 (1.157)	0.699
SCr (umol/L)	49.30 (8.223)	50.20 (7.612)	0.367
TG (mmol/L)	1.79 (1.141)	1.64 (1.063)	0.287
TC (mmol/L)	4.69 (1.002)	4.60 (0.866)	0.453
HDL (mmol/L)	1.21 (0.421)	1.19 (0.347)	0.671
LDL (mmol/L)	2.66 (0.783)	2.62 (0.636)	0.711
Apo-A (g/L)	1.07 (0.196)	1.09 (0.236)	0.445
Apo-B (g/L)	0.97 (0.335)	0.89 (0.206)	0.021
Ca (mmol/L)	2.43 (0.200)	2.46 (0.131)	0.119
DHEAS (umol/L)	230.32 (117.525)	234.49 (111.011)	0.778
Continued			

Characteristic	Optimal weight loss responder (n = 696)	Non-optimal weight loss responder (n = 580)	*P* value
SHBG (nmol/L)	27.26 (16.156)	30.08 (22.072)	0.287
ACTH-8:00 (pmol/L)	6.84 (3.785)	6.66 (4.363)	0.731
F-8:00 (nmol/L)	354.96 (124.783)	336.71 (139.961)	0.298
Vitamin D (ng/mL)	17.83 (5.599)	17.93 (6.212)	0.898
Albumin (g/L)	45.95 (3.753)	46.51 (3.766)	0.247
CRP (mg/L)	4.07 (4.730)	2.87 (3.651)	0.046
Metformin treatment regimen (0: without; 1: with metformin)	69 (71.88%)	114 (59.44%)	0.146

Table 1. Baseline clinical and laboratory data characteristics of patients with obesity in optimal weight loss responder and non-optimal weight loss responder groups. Data are shown as means (SD), *P* value. *PCO* polycystic ovary, *SBP* systolic blood pressure, *DBP* diastolic blood pressure, *HOMA IR* homeostatic model assessment of insulin resistance, *HOMA β* homeostatic model assessment of β-cell function, *HbA1c* hemoglobin A1C, *TSH* thyroid-stimulating hormone, *FT3* free triiodothyronine, *FT4* free thyroxine, *TgAb* thyroglobulin antibody, *ALT* alanine aminotransferase, *AST* aspartate aminotransferase, *γ-GT* γ-glutamyl transferase, *TBIL* total bilirubin, *DBIL* direct bilirubin, *UA* uric acid, *BUN* blood urea nitrogen, *SCr* serum creatinine, *TG* triglyceride, *TC* total cholesterol, *HDL* high density lipoprotein, *LDL* low density lipoprotein, *Apo* apoprotein, *DHEAS* dehydroepiandrosterone sulfate, *SHBG* sex hormone-binding globulin, *ACTH* adrenocorticotropic hormone, *F* cortisol, *CRP* C-reactive protein.

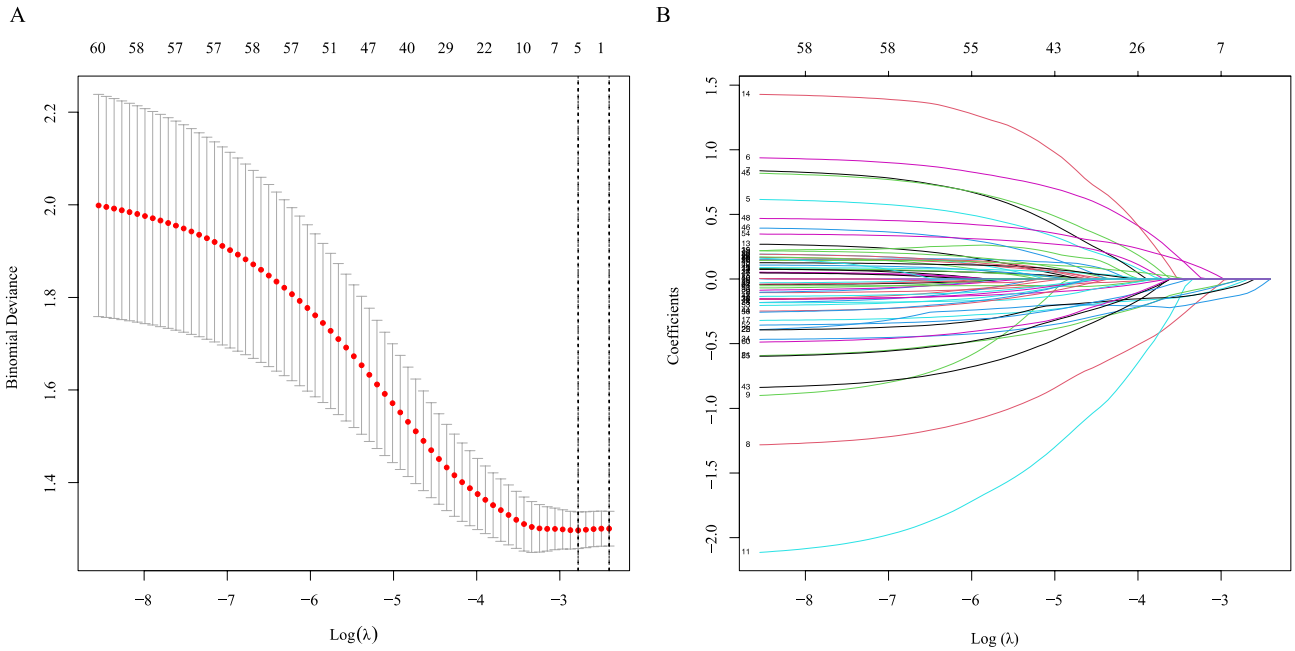

Figure 1. LASSO model for feature selection in predicting optimal weight loss responder. (**A**) Determine the optimal coefficient lambda (λ) in the LASSO model by tenfold cross-validation. (**B**) Distribution plot of LASSO coefficients for 13 features. The LASSO coefficient curve depicts how each characteristic related to optimal weight loss responder changes as lambda varies. The optimal lambda, marked by non-zero coefficients, is crucial for constructing a predictive model.

achieving ≥ 10% weight loss. However, maintaining such weight loss is challenging, with reported weight regain of 30%–50% within one year[26].

Various methods for weight loss include behavioral interventions, nutritional adjustments, exercise, drug therapy, and surgery[11]. However, lifestyle therapy alone typically results in moderate weight loss, which is often difficult to sustain. Drug-assisted lifestyle modifications can achieve greater weight loss than lifestyle changes alone. Approved weight loss drugs include sympathomimetic drugs, pancreatic lipase inhibitors, and glucagon-like peptide-1 (GLP-1) receptor agonists[27]. The efficacy and side effects of these drugs vary, and due to insufficient medical insurance coverage, few patients utilize them. Furthermore, surgery is invasive and has limited indications, thus only a small population undergoes it.

Diet and exercise are fundamental to weight loss. Identifying the risks associated with optimal and non-optimal weight loss responders before commencing diet and exercise interventions can aid individuals struggling with

	β	Odds ratio (95% CI)	*P* value
Age	− 0.074	0.928	0.783
Hair loss	− 0.971	0.379	0.006
Hirsutism	− 0.242	0.785	0.183
ALT	− 0.587	0.556	0.025
BMI	0.671	1.956	0.009
Insulin at 0 min	0.428	1.536	0.016
Blood glucose at 0 min	− 0.263	0.769	0.134
Blood glucose at 60 min	− 0.400	0.670	0.153
TC	0.892	2.441	0.020
LDL	− 0.230	0.795	0.194
Ca	− 0.448	0.639	0.061
F-8:00	− 0.154	0.857	0.558
HOMA β	− 0.400	0.803	0.259

Table 2. Multivariate logistic regression analysis of 13 predictor variables in the final model.

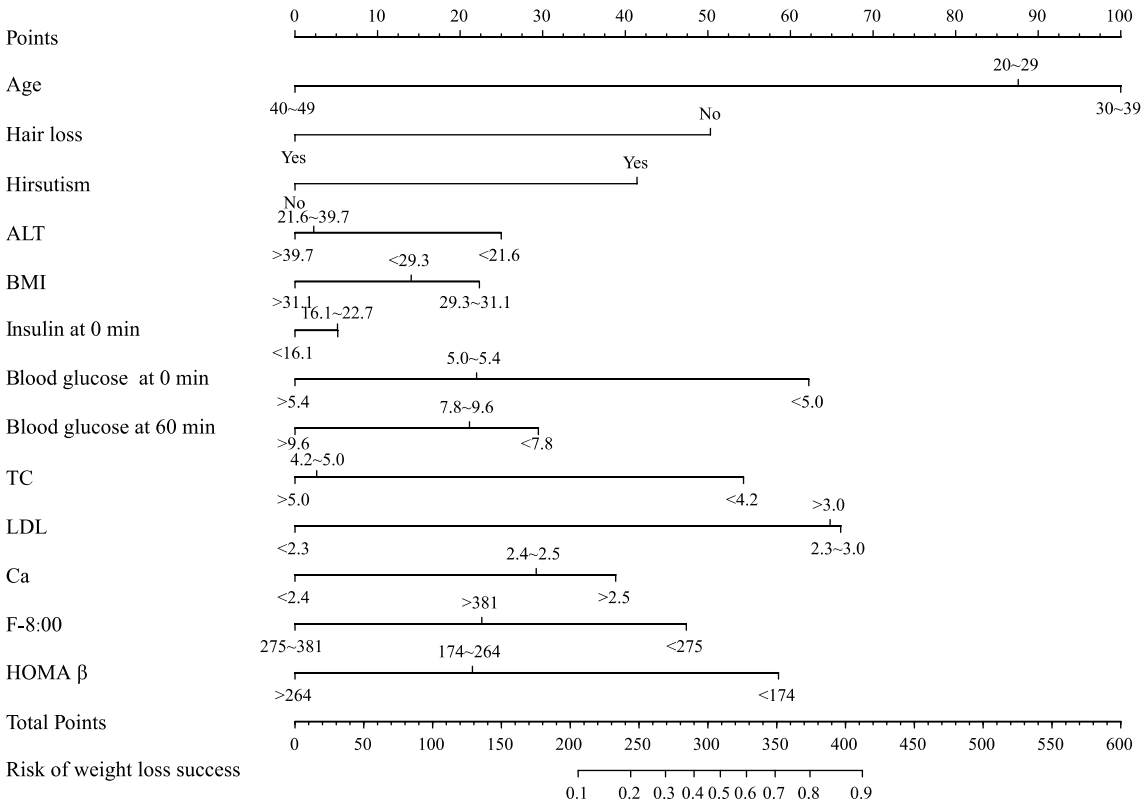

Figure 2. Nomogram for predicting risk of optimal weight loss responders in patients with obesity. Each variable contributes to a corresponding score, which is then summed to calculate the total score. This total score is then used to determine the probability of being an optimal weight loss responder.

weight loss in enhancing their behavioral, dietary, and other interventions to improve their chances of optimal weight loss. Currently, some studies have developed weight loss prediction models[17,18,28], but these models are based on small sample sizes and lack data on Asian populations. This study utilizes data from individuals who visited a weight loss clinic for diet and exercise interventions. Their baseline data were analyzed by dividing them into two groups based on whether they achieved more than 5% weight loss during the three-month follow-up.

In this study, 12 variables were selected based on LASSO regression and logistic regression, and these were included in the nomogram along with age. Each variable's line segment in the nomogram is marked with a scale representing the variable's possible value range. The total score, termed total points, is obtained by summing the individual scores of all variables. The length of the line segment indicates the factor's contribution to the outcome. Our nomogram identified age as the most influential predictor, followed by LDL, blood glucose at 0 min, HOMA β, TC, hair loss, F-8:00, hirsutism, Ca, blood glucose at 60 min, ALT, BMI, and insulin at 0 min.

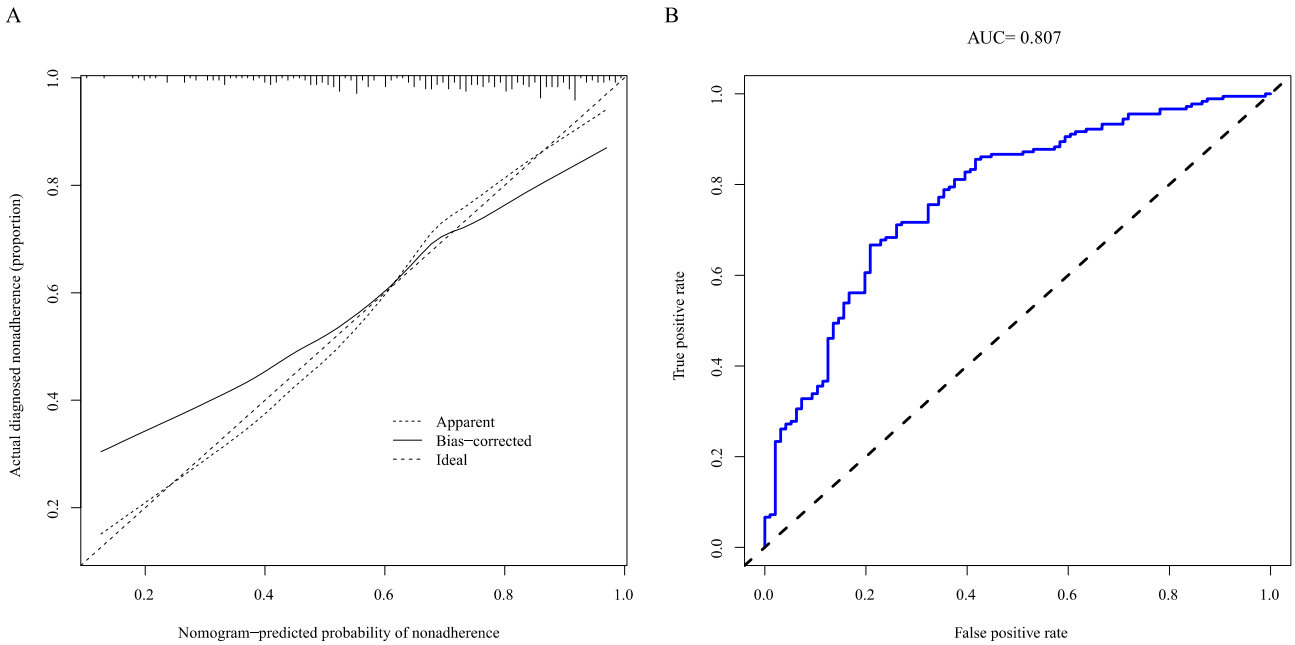

Figure 3. Calibration and ROC curve of the nomogram. (**A**) Calibration curve of nomogram. The x-axis represents the nomogram predicted probability and the y-axis represents the actual probability of optimal weight loss responder. The dashed line at a 45-degree angle represents perfect prediction, while the solid line represents observed nomogram performance corrected for bias using bootstrapping (B = 1000 replicates). (**B**) ROC curve of the nomogram. When using bootstrap resampling (number = 500), the nomogram achieved an AUC of 0.807 (95% CI 0.736–0.868).

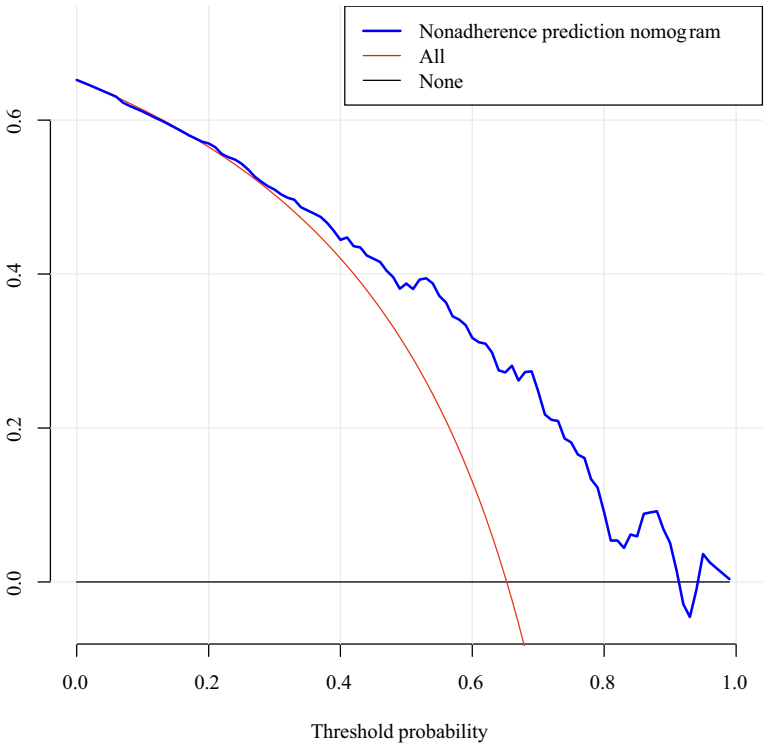

Figure 4. Decision curve analysis for the nomogram. The black horizontal line represents a net benefit of 0 when all patients with obesity are not predicted according to the nomogram. The solid red line shows the scenario where all patients with obesity are treated according to the nomogram. The area enclosed by the three lines (black, red, and blue) signifies the clinical utility of the nomogram. A larger area indicates greater clinical value in using the nomogram.

Our nomogram also indicated that hair loss and hirsutism are significant factors, potentially exerting more influence than BMI and fasting insulin. This suggests that greater hair loss may be associated with a higher likelihood of optimal weight loss response. Excessive body hair may result from the body's sensitivity to androgens. Indicators of abdominal obesity in men are negatively correlated with testosterone levels. In women, unlike men, high androgen levels are typically a significant risk factor for obesity and are closely linked to the development of abdominal obesity[29]. This study also identified ALT as a prognostic factor, with lower ALT levels being associated with a higher likelihood of optimal weight loss response. The calibration curve indicated that the nomogram was well calibrated, and the AUC (0.807) demonstrated good predictive performance. Additionally, our DCA validated the clinical utility of the nomogram, enhancing its potential application in real-world settings.

Although the model's predictions were favorable, this study has several major limitations: the follow-up period was too short, and it did not account for weight regain after weight loss. Another significant limitation is the small number of participants at the weight management clinic, resulting in a limited sample size and only internal validation without external validation. Additionally, only diet and exercise interventions were studied, excluding other methods such as drugs and surgery. Many individuals desire weight loss but are not actively engaged due to the perceived difficulty and low probability of optimal weight loss responders. Thus, improving accurate prediction models for optimal weight loss is crucial to enhance public confidence.

Furthermore, this study did not consider factors such as sleep[30], support from friends and family[31], eating habits[32], reasons for weight loss, previous non-optimal weight loss attempts, or other elements that might influence optimal weight loss. Future studies should validate our nomogram in external populations and explore integrating variables such as genetics and psychosocial factors to enhance prediction accuracy further.

In conclusion, we developed a nomogram to predict optimal weight loss responders following diet and exercise interventions. Our study fills a gap in existing knowledge on weight loss prediction models in Asian populations, providing a tool that is easy to use, highly accurate, and possesses excellent effect differentiation and calibration capabilities. It can aid clinicians in making personalized predictions about the likelihood of optimal weight loss response for each patient with obesity, thereby facilitating more tailored weight loss interventions to improve outcomes.

Data availability

All relevant data can be requested through the corresponding author.

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Author contributions

L.Y. was responsible for writing the article. J.W. was responsible for patient recruitment and data collection. Z.H. was responsible for the final modification. T.X. was responsible for the design and analysis of the project, and W.Z. was responsible for data compilation.

Competing interests

The authors declare no competing interests.

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