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# Construction of a Diagnostic Model for Non-Alcoholic Fatty Liver Disease in Populations with BMI<24 kg/m² Based on Basic Biochemical Indicators

**Chunxia Guo**
Linyi Central Hospital
**Jingguo Bai**
sdlygcx2025@163.com
Linyi Third People's Hospital
**Zhenyan Jia**
Linyi Central Hospital
**Fankun Lin**
Linyi Central Hospital

**Research Article**

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# Construction of a Diagnostic Model for Non-Alcoholic Fatty Liver Disease in Populations with BMI<24 kg/m² Based on Basic Biochemical Indicators

Chunxia Guo[1], Jingguo Bai[2]*, Zhenyan Jia*, Fankun Lin[1]

[1] Health Management Center, Linyi Central Hospital, Linyi, Shandong 276400, China; [2] Health Management Center, The Third People's Hospital of Linyi, Linyi, Shandong 276000, China

Corresponding authors: Bai Jingguo, E-mail: sdlygcx2025@163.com; Jia Zhenyan, E-mail: wjpaxl2021@163.com

**Abstract**

Objective To construct a diagnostic model for non-alcoholic fatty liver disease (NAFLD) among individuals with body mass index (BMI) <24 kg/m² and evaluate its diagnostic performance for NAFLD. Methods A retrospective analysis was performed on 9173 physical examinees with BMI<24 kg/m² from the Health Management Center of Linyi Central Hospital between June 2022 and May 2025. Subjects were randomly divided into a derivation cohort (6356 cases) and an internal validation cohort (2817 cases) at a 7:3 ratio. Within the derivation cohort, participants were assigned to the NAFLD group (761 cases) and control group (5595 cases) according to ultrasonography and computed tomography (CT) findings. Non-parametric rank-sum test was adopted for statistical analysis. Logistic regression was used to build the diagnostic model. The Hosmer-Lemeshow test was applied to assess model goodness-of-fit, and the receiver operating characteristic (ROC) curve was employed to evaluate diagnostic efficiency. A total of 3199 physical examinees with BMI<24 kg/m² enrolled from May 2023 to November 2024 at the Health Management Center of The Third People's Hospital of Linyi served as the external validation set. Both internal and external

validation cohorts were used to further verify model performance. Results The detection rate of NAFLD was 12.0% in the BMI<24 kg/m² population; the detection rate was higher in males than in females (17.0% vs. 9.4%, $\chi^2$=104.335, P<0.001). Age ($X_1$), BMI ($X_2$), triglyceride ($X_3$), creatinine ($X_4$), uric acid ($X_5$), fasting blood glucose ($X_6$), alanine aminotransferase ($X_7$), aspartate aminotransferase ($X_8$), γ-glutamyl transferase ($X_9$) and albumin ($X_{10}$) were independent risk factors for NAFLD. The diagnostic model was established as follows: $Logit(P)= -16.357+0.015X_1+0.349X_2+1.006X_3-0.026X_4+0.006X_5+0.219X_6+0.041X_7-0.038X_8+0.055X_9+0.043X_{10}$. Taking the Youden index of 0.621 as the diagnostic cut-off value, the area under the ROC curve (AUC) was 0.884, with a sensitivity (Se) of 81.9% and specificity (Sp) of 80.2%. The AUCs of internal validation and external validation cohorts were 0.851 and 0.840 respectively; their Youden indices were 0.542 and 0.552; sensitivity values were 74.0% and 79.8%; specificity values were 80.2% and 75.3%. Conclusion The diagnostic model for NAFLD in individuals with BMI<24 kg/m² constructed on basic biochemical indicators shows favourable diagnostic performance, and possesses practical value for clinical diagnosis and follow-up.

**Key words** non-alcoholic fatty liver disease; diagnostic model; biochemical indicators; non-overweight

## Introduction

Non-alcoholic fatty liver disease (NAFLD) has overtaken viral hepatitis to become the most prevalent chronic liver disease worldwide[1]. The number of NAFLD patients in China keeps rising, and China is projected to become the country with the highest global morbidity and case burden by 2030, imposing a heavy burden on chronic disease prevention and control[2]. Data from Asia's largest biopsy-confirmed international NAFLD registry

demonstrated that non-obese NAFLD accounts for 21.6% of cases[3]. The proportion of patients with non-obese or lean NAFLD is increasing. Notably, NAFLD patients with normal waist circumference carry higher risks of severe liver disease compared with obese NAFLD patients[4]. This subgroup often overlooks or fails to receive NAFLD diagnosis. American studies indicate that 95% of NAFLD patients are unaware of their liver disease[5], and this proportion is even higher among non-obese and lean populations. Existing evidence shows that among non-obese or lean NAFLD patients, 39.0% develop non-alcoholic steatohepatitis, 29.2% suffer from advanced fibrosis, and 3.2% progress to cirrhosis[6]. Therefore, active screening for NAFLD in non-obese or lean populations is of great significance. This study aimed to construct a NAFLD diagnostic model for people with body mass index (BMI) <24 kg/m² using routine biochemical parameters, to explore a novel, simple and non-invasive screening tool for clinical practice.

# Materials and Methods

## 1 Study Subjects

This was a cross-sectional study. (1) A total of 9173 subjects with complete clinical data and BMI<24 kg/m² undergoing health check-ups from June 2022 to May 2025 at the Health Management Center of Linyi Central Hospital were enrolled. Participants were randomly assigned to the derivation cohort (n=6356) and internal validation cohort (n=2817) at a ratio of 7:3. In the derivation cohort, subjects with hepatic steatosis identified by liver ultrasonography or computed tomography (CT) were allocated to the Lean-NAFLD group (n=761), and the remaining subjects formed the control group (n=5595). (2) A total of 3199 examinees with BMI<24 kg/m² from May 2023 to November 2024 at the Health Management Center of The Third People's Hospital of Linyi were recruited for external validation.

**Inclusion criteria**

(1) Aged between 18 and 90 years old (18 ⩽ age ⩽ 90 years);

(2) BMI ＜ 24 kg/m² ;

(3) Hepatic steatosis confirmed by liver ultrasonography and CT. The diagnostic criteria for fatty liver were based on the Guidelines for the Prevention and Treatment of Metabolic-Associated (Non-Alcoholic) Fatty Liver Disease (2024 Edition)[7];

(4) Complete examination data, including general information, liver and renal function, blood lipid profiles, liver ultrasonography, CT and other relevant records.

**Exclusion criteria**

(1) Alcoholic liver disease, diagnosed according to the Primary Care Guidelines for Alcoholic Liver Disease (2019)[8];

(2) Organic diseases such as cardiac insufficiency and pre-existing abnormal liver or kidney function;

(3) Pregnant or lactating women;

(4) Patients receiving medications for hyperlipidaemia, diabetes mellitus, renal insufficiency, acute or chronic hepatitis, as well as patients with uncontrolled thyroid dysfunction;

(5) Patients with malignant tumours including hepatocellular carcinoma, breast cancer, lung cancer, etc.

This study was approved by the Ethics Committee of Linyi Central Hospital (IRB) (Approval No. LCH-LW-2025069). Written informed consent was waived by the ethics committee due to the retrospective nature of the study, and all patient data were anonymized to protect privacy.

(1) Metabolic biochemical measurements: Fasting venous blood samples were collected after at least 8 hours of overnight fasting. An Olympus AU5421 automatic biochemical analyser was used to detect alanine aminotransferase (ALT), aspartate aminotransferase

(AST), γ-glutamyl transferase (GGT), low-density lipoprotein (LDL), total cholesterol (TC), triglyceride (TG) and other biochemical markers.

(2) Liver ultrasonography: A PHILIP-IU ultrasonic diagnostic instrument (probe frequency: 2.8–5.0 MHz) was used to evaluate liver echo intensity and deep-region attenuation. All examinations were performed by experienced physicians. Subjects fasted for ≥10 hours and lay supine. Multiple positive-and-lateral-position scans of the liver were performed. Fatty liver diagnosis complied with criteria in Guidelines for the Prevention and Treatment of Metabolic-Associated (Non-Alcoholic) Fatty Liver Disease (2024 Edition)[7].

(3) Liver CT: A uCT 550+ X-ray computed tomography system (United Imaging Healthcare, Shanghai, China) was used following standard low-dose CT protocols; scanning extended inferiorly to the lower margin of the 3rd lumbar vertebral body. Image acquisition and interpretation were performed by qualified radiologists. NAFLD was defined per 2024 MASLD/NAFLD guidelines[7]: diffuse hepatic hypodensity with a liver-to-spleen CT attenuation ratio <1.0 on non-contrast scans.

## 3 Statistical Analysis

SPSS 25.0 software (SPSS Inc., Chicago, Illinois, USA) was used for statistical analyses. Count data were compared using the $\chi^2$ test and presented as $n$ (%). Normally-distributed continuous variables were compared by independent-samples t-test and expressed as mean ± standard deviation. Non-normally-distributed continuous variables were analysed by rank-sum tests and reported as median (interquartile range) [$M(P_{25}, P_{75})$]. Multivariate binary logistic regression was applied for risk-factor screening and model construction. The Hosmer-Lemeshow test assessed model goodness-of-fit; $P$>0.1 indicated satisfactory fit. ROC curves were plotted to calculate Youden index, sensitivity (Se) and specificity (Sp). $P$<0.05 was considered statistically significant.

# Results

## 1 Baseline Characteristics

Among the 9173 subjects with BMI<24 kg/m², there were 3229 males and 5944 females. NAFLD was diagnosed in 1100 participants, corresponding to an overall detection rate of 12.0% (1100/9173). The detection rate was 17.0% (539/3229) in males and 9.4% (561/5944) in females; males had significantly higher NAFLD prevalence ($\chi^2$=104.335, $P$<0.001).

No statistically significant inter-group differences were observed between derivation and internal-validation cohorts for all measured indicators ($P$>0.05, Table 1). The NAFLD detection rate within the derivation cohort was 12.0% (761/6357). The NAFLD subgroup contained a higher proportion of male participants. Age, BMI, TC, TG, LDL, creatinine (Cr), uric acid (UA), fasting blood glucose (FBG), blood urea nitrogen (BUN), ALT, AST, GGT, total protein (TPO), albumin (ALB), alkaline phosphatase (ALP), total bilirubin (TBIL), indirect bilirubin (IBIL) and cholyglycine (CG) levels were higher in the NAFLD group than in controls, whereas the AST/ALT ratio was lower; all differences reached statistical significance (Table 2).

**Table 1 Comparison of baseline characteristics between derivation cohort and internal-validation cohort**

| | Derivation cohort (n=6356) | Internal-validation cohort (n=2817) | t/$\chi^2$/Z value | P value |
|---|---|---|---|---|
| Male, n(%) | 2231(35.1%) | 992(35.2%) | 0.011 | 0.924 |
| Age, years | 48(35, 57) | 48(36, 57) | −1.117 | 0.264 |
| BMI, kg/m² | 22.1(20.86, 23.15) | 22.19(20.98, 23.18) | −1.265 | 0.206 |
| LDL, mmol/L | 2.91(2.40, 3.51) | 2.92(2.42, 3.54) | −0.654 | 0.513 |
| TG, mmol/L | 0.94(0.71, 1.33) | 0.94(0.70, 1.31) | −0.806 | 0.420 |
| TC, mmol/L | 4.85(4.22, 5.54) | 4.84(4.24, 5.58) | −0.269 | 0.788 |
| Cr, mmol/L | 61(54, 71) | 61(54, 71) | −0.229 | 0.819 |

| | Derivation cohort (n=6356) | Internal-validation cohort (n=2817) | t/$\chi^2$/Z value | P value |
|---|---|---|---|---|
| UA, mmol/L | 267(226, 314) | 266(225, 316) | −0.004 | 0.997 |
| FBG, mmol/L | 5.02(4.73, 5.42) | 5.04(4.72, 5.43) | −0.182 | 0.856 |
| BUN, mmol/L | 4.70(3.90, 5.50) | 4.70(3.90, 5.60) | −0.145 | 0.884 |
| ALT, U/L | 14(10, 19) | 14(11, 19) | −0.593 | 0.553 |
| AST, U/L | 18(15, 21) | 18(15, 21) | −0.391 | 0.696 |
| ALT/AST | 1.30(1.05, 1.58) | 1.29(1.05, 1.57) | −0.994 | 0.320 |
| GGT, U/L | 14(11, 20) | 15(11, 20) | −1.242 | 0.214 |
| ALP, IU/L | 67(56, 82) | 67(56, 83) | −0.129 | 0.898 |
| TPO, g/L | 73.40(70.80, 76.20) | 73.30(70.70, 76.20) | −0.142 | 0.887 |
| ALB, g/L | 46.30(44.60, 48.20) | 46.30(44.60, 48.18) | −0.455 | 0.649 |
| GLB, g/L | 27(24.70, 29.40) | 26.90(24.70, 29.40) | −0.214 | 0.831 |
| ALB/GLB | 1.70(1.60, 1.90) | 1.70(1.60, 1.90) | −0.034 | 0.973 |
| TBIL, μmol/L | 10.80(8.20, 14.36) | 10.90(8.20, 14.60) | −1.341 | 0.180 |
| DBIL, μmol/L | 3.51(2.79, 4.68) | 3.60(2.79, 4.68) | −1.251 | 0.211 |
| IBIL, μmol/L | 7.13(5.20, 9.76) | 7.24(5.24, 9.99) | −1.397 | 0.162 |
| CG, μg/mL | 0.80(0.50, 1.20) | 0.80(0.50, 1.20) | −1.053 | 0.292 |

**Table 2 Univariate analysis in derivation cohort**

| | Control group (n=5595) | NAFLD group (n=761) | t/$\chi^2$/Z value | P value |
|---|---|---|---|---|
| Male, n(%) | 1860(33.2%) | 371(48.8%) | 70.719 | <0.001 |
| Age, years | 47(34, 57) | 52(44, 59) | −9.391 | <0.001 |
| BMI, kg/m² | 22.02(20.72, 23.03) | 23.04(22.22, 23.53) | −17.559 | <0.001 |
| LDL, mmol/L | 2.88(2.39, 3.45) | 3.16(2.56, 3.82) | −7.586 | <0.001 |
| TG, mmol/L | 0.90(0.68, 1.21) | 1.65(1.20, 2.44) | −27.711 | <0.001 |
| TC, mmol/L | 4.79(4.20, 5.48) | 5.22(4.53, 5.99) | −9.957 | <0.001 |
| Cr, mmol/L | 61(54, 71) | 63(55, 73) | −2.780 | 0.005 |
| UA, mmol/L | 262(223, 307) | 308(263, 356.75) | −15.882 | <0.001 |

| | Control group (n=5595) | NAFLD group (n=761) | t/$\chi^2$/Z value | P value |
|---|---|---|---|---|
| FBG, mmol/L | 4.98(4.71, 5.34) | 5.45(5.03, 6.22) | −18.650 | <0.001 |
| BUN, mmol/L | 4.60(3.90, 5.50) | 4.80(4.00, 5.80) | −3.703 | <0.001 |
| ALT, U/L | 13(10, 18) | 19(14, 27) | −19.440 | <0.001 |
| AST, U/L | 18(15, 21) | 20(16, 24) | −10.989 | <0.001 |
| ALT/AST | 1.33(1.08, 1.62) | 1.00(0.80, 1.31) | −18.032 | <0.001 |
| GGT, U/L | 14(11, 18) | 24(17, 37) | −25.950 | <0.001 |
| TPO, g/L | 73.40(70.70, 76.10) | 74.20(71.10, 76.60) | −3.645 | <0.001 |
| ALP, IU/L | 66(55, 81) | 76(62, 91) | −11.111 | <0.001 |
| ALB, g/L | 46.30(44.50, 48.20) | 46.70(45.10, 48.38) | −3.882 | <0.001 |
| GLB, g/L | 27(24.70, 29.30) | 27.20(24.90, 29.60) | −1.290 | 0.197 |
| ALB/GLB | 1.70(1.60, 1.90) | 1.70(1.60, 1.90) | −0.684 | 0.494 |
| TBIL, μmol/L | 10.70(8.10, 14.30) | 11.20(8.50, 14.68) | −2.312 | 0.021 |
| DBIL, μmol/L | 3.60(2.79, 4.68) | 3.51(2.79, 4.68) | −0.250 | 0.803 |
| IBIL, μmol/L | 7.08(5.18, 9.70) | 7.49(5.44, 10.25) | −3.167 | 0.002 |
| CG, μg/mL | 0.80(0.50, 1.15) | 0.80(0.50, 1.30) | −3.919 | <0.001 |

## 2 Establishment of the NAFLD Diagnostic Model

Binary logistic regression was performed on the derivation cohort, with NAFLD status (yes=1, no=0) as the dependent variable Y. Variables yielding $P<0.05$ in univariate analyses were entered as independent variables; continuous variables used raw measured values; gender was coded as male=1, female=0. Multivariate logistic regression demonstrated that age ($X_1$), BMI ($X_2$), TG ($X_3$), Cr ($X_4$), UA ($X_5$), FBG ($X_6$), ALT ($X_7$), AST ($X_8$), GGT ($X_9$) and ALB ($X_{10}$) were independent risk factors for NAFLD (all $P<0.05$, Table 3). These ten parameters were incorporated to construct the prediction model:

$$\text{Logit}(P) = -16.357 + 0.015X_1 + 0.349X_2 + 1.006X_3 - 0.026X_4 + 0.006X_5 + 0.219X_6 + 0.041X_7 - 0.038X_8 + 0.055X_9 + 0.043X_{10}$$

**Table 3 Multivariate logistic regression analysis in derivation cohort**

| | Regression coefficient | Standard error | Wald | P value | OR | 95% CI |
|---|---|---|---|---|---|---|
| Age, years | 0.015 | 0.004 | 13.125 | <0.001 | 1.015 | 1.007–1.024 |
| BMI, kg/m² | 0.349 | 0.042 | 67.972 | <0.001 | 1.418 | 1.305–1.540 |
| TG, mmol/L | 1.006 | 0.064 | 249.121 | <0.001 | 2.735 | 2.414–3.099 |
| Cr, mmol/L | −0.026 | 0.004 | 36.155 | <0.001 | 0.975 | 0.966–0.983 |
| UA, mmol/L | 0.006 | 0.001 | 57.685 | <0.001 | 1.006 | 1.004–1.008 |
| FBG, mmol/L | 0.219 | 0.029 | 57.991 | <0.001 | 1.245 | 1.177–1.317 |
| ALT, U/L | 0.041 | 0.007 | 35.353 | <0.001 | 1.042 | 1.028–1.056 |
| AST, U/L | −0.038 | 0.011 | 12.254 | <0.001 | 0.962 | 0.942–0.983 |
| GGT, U/L | 0.055 | 0.004 | 182.337 | <0.001 | 1.057 | 1.048–1.065 |
| ALB, g/L | 0.043 | 0.018 | 5.672 | 0.017 | 1.044 | 1.008–1.081 |
| Constant | −16.357 | 1.313 | 155.233 | <0.001 | – | – |

## 3 Model Goodness-of-Fit and Diagnostic Performance

The Hosmer-Lemeshow test yielded $\chi^2$=10.719, $P$=0.218, indicating adequate model fit. ROC curve analysis obtained an AUC of 0.884 (95% CI: 0.876–0.892)(Figure 1, Table 4). At the optimal cut-off corresponding to Youden index=0.621, sensitivity was 81.9% and specificity was 80.2%.

**Table 4 ROC-curve metrics of the diagnostic model across derivation, internal-validation and external-validation cohorts**

| Variable | AUC | Standard error | 95% CI | Z-statistic | Se (%) | Sp (%) | Youden index | P value |
|---|---|---|---|---|---|---|---|---|
| Derivation cohort | 0.884 | 0.0067 | 0.876–0.892 | 56.775 | 81.9 | 80.2 | 0.621 | <0.0001 |
| Internal validation | 0.851 | 0.0106 | 0.837–0.864 | 32.968 | 74.0 | 80.2 | 0.542 | <0.0001 |

| Variable | AUC | Standard error | 95% CI | Z-statistic | Se (%) | Sp (%) | Youden index | P value |
|---|---|---|---|---|---|---|---|---|
| External validation | 0.840 | 0.0115 | 0.826–0.852 | 29.555 | 79.8 | 75.3 | 0.552 | <0.0001 |

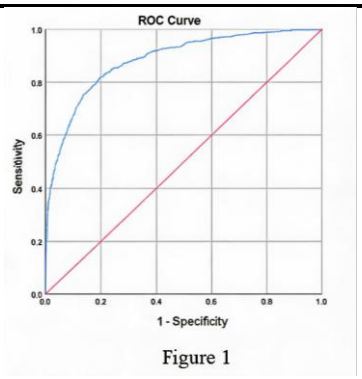



Figure 1 ROC curve for model construction. Receiver operating characteristic curve of the NAFLD diagnostic model in the training cohort.

## 4 Internal Validation of the Diagnostic Model

The internal-validation cohort contained 2817 subjects, among whom 339 had NAFLD (detection rate 12.0%). For internal validation, AUC=0.851 (95% CI: 0.837–0.864)(Figure 2, Table 4), Youden index=0.542, sensitivity=74.0%, specificity=80.2%.

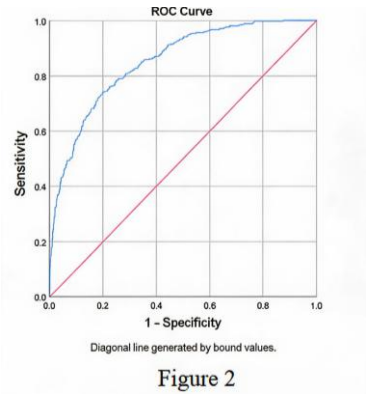


Figure 2 ROC curve for internal validation of the model. Receiver operating characteristic curve for internal validation of the NAFLD diagnostic model.

## 5 External Validation of the Diagnostic Model

The external validation dataset included 3199 participants (1378 males, 1821 females) with BMI<24 kg/m². NAFLD was detected in 342 subjects (10.7%); detection rates were 15.7% (216/1378) for males and 6.9% (126/1821) for females ($\chi^2$=62.981, $P$<0.001). External-validation results: AUC=0.840 (95% CI: 0.826–0.852)(Figure 3, Table 4), Youden index=0.552, sensitivity=79.8%, specificity=75.3%.

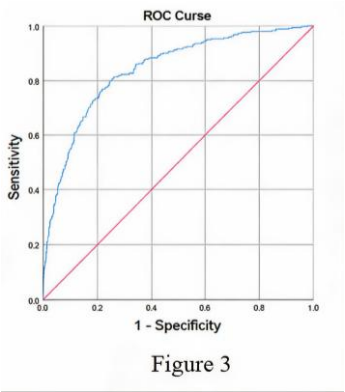


Figure 3

Figure 3 ROC curve for external validation of the model. Receiver operating characteristic curve for external validation of the NAFLD diagnostic model.

## Discussion

The global pooled prevalence of NAFLD is approximately 32.4%[1]. Previous reports noted that 40.8% of NAFLD patients are non-obese and 19.2% are lean; the annual incidence of NAFLD among non-obese individuals reaches 24.6 per 1000 person-years[6]. Prevalence varies geographically from 25% (Malaysia, Pakistan) to 50% (Austria, Mexico, Sweden). NAFLD may progress to non-alcoholic steatohepatitis, liver fibrosis, cirrhosis, hepatocellular carcinoma and death[9]. Non-obese NAFLD is closely associated with steatohepatitis and

advanced hepatic fibrosis[3]. Several studies have demonstrated that lean NAFLD is linked to higher all-cause, cardiovascular-related and liver-related mortality compared with individuals without NAFLD[10]. Therefore, accurate identification of NAFLD in non-overweight populations carries important clinical implications.

Liver biopsy remains the diagnostic gold-standard for NAFLD, yet its invasiveness, high cost and sampling bias limit its widespread clinical application[11]. Ultrasonography is the most commonly-used screening modality, recognising fatty liver by characteristic "bright-liver" appearances and blurry intrahepatic vascular borders; nevertheless, it exhibits low sensitivity for mild steatosis and results are operator-dependent[7]. Controlled-attenuation-parameter-based transient elastography (FibroScan) is widely applied and can detect hepatic steatosis involving ≥5% of liver parenchyma and grade steatosis severity[7]; however, readings are confounded by subcutaneous fat thickness and comorbidities, and the equipment is not universally accessible[12]. CT and magnetic-resonance imaging (MRI) are mainly used for differential diagnosis between diffuse steatosis and focal liver lesions; their diagnostic accuracy for NAFLD is not superior to ultrasound, while costs are relatively high[13]. In many primary-care settings, advanced imaging resources are limited. By contrast, basic liver-kidney biochemistry tests are routine components of health check-ups, offering objectivity, low cost and high patient acceptance. Constructing NAFLD prediction models based on these readily-available laboratory parameters for non-overweight populations can reduce diagnostic costs and improve case-finding rates.

In this study, the NAFLD detection rate among BMI<24 kg/m² participants was 12.0%, comparable to the 13.3% reported by Zhao et al.[14]. Zeng et al.[4] reported a 17.5% prevalence of lean NAFLD among individuals with BMI<23 kg/m² and normal waist circumference. Heterogeneity in lean-NAFLD prevalence across regions and ethnic groups may stem from genetic susceptibility, lifestyle disparities and variations in diagnostic

modalities and criteria[15–16]. Males demonstrated higher NAFLD risk, possibly due to the hepatoprotective effects of oestrogen in females[17]. Multivariate logistic regression identified age, BMI, TG, Cr, UA, FBG, ALT, AST, GGT and ALB as independent risk factors for NAFLD, consistent with existing literature[4,14,18–19]. Even within the BMI<24 kg/m² stratum, higher BMI independently increased NAFLD odds. Hence, weight control and BMI reduction remain important therapeutic targets for metabolic disturbance and liver injury even in lean NAFLD patients[20].

Our fitted model passed the Hosmer-Lemeshow goodness-of-fit test. Internal and external validation yielded AUC values of 0.851 and 0.840 respectively, confirming good overall discriminative performance. Few existing prediction models specifically target NAFLD among BMI-normal subjects. This tool may serve as an adjunct to ultrasonography or FibroScan, and can be deployed in resource-limited primary-care institutions for population-level screening and longitudinal follow-up owing to its non-invasiveness and low cost.

Limitations of this study: (1) NAFLD diagnoses relied on ultrasound and CT rather than FibroScan-CAP or liver biopsy, potentially underestimating true prevalence. (2) This cross-sectional design is susceptible to selection bias. (3) No age-stratified analyses were performed, and only one external validation cohort was recruited. Future research should incorporate age-stratified subgroup analyses and multi-centre external validations to further refine model robustness.

## Conclusion

Age, BMI, TG, Cr, UA, FBG, ALT, AST, GGT and ALB represent key predictors of NAFLD for people with BMI<24 kg/m². The derived logistic-regression-based diagnostic model achieves satisfactory discriminative ability and shows promising clinical utility.

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