Diabetes, Metabolic Syndrome and Obesity

Dovepress
Taylor & Francis Group

Open Access Full Text Article

ORIGINAL RESEARCH

Diagnostic Model for Obesity and Type 2 Diabetes Mellitus Based on Feature Selection from Fatty Acids, Amino Acids, and Clinical Characteristics

Xiaoxia Guo[1], Wenjing Cheng[1], Xuliang Hao[2], Lu Zhang[3], Lei LV[1]

[1]Department of Metabolic Disease, Shanxi Provincial Hospital of Traditional Chinese Medicine, Taiyuan, 030012, People's Republic of China; [2]Institute of Traditional Chinese Medicine Prescriptions, Shanxi Provincial Hospital of Traditional Chinese Medicine, Taiyuan, 030012, People's Republic of China; [3]Department of Pharmaceutical Preparation, Affiliated Hospital of Shanxi University of Traditional Chinese Medicine, Taiyuan, 030024, People's Republic of China

Correspondence: Xuliang Hao, Email gxx1204@outlook.com

Diabetes, Metabolic Syndrome and Obesity downloaded from https://www.dovepress.com/
For personal use only.

Purpose: Given that obesity and type 2 diabetes mellitus (T2DM) share complicated pathophysiological mechanisms, this study aimed to establish a diagnostic model for the two diseases using feature selection from fatty acids, amino acids, and clinical characteristics.

Methods: This prospective study included 81 obese patients, 25 T2DM patients and 33 healthy controls. Amino acids and fatty acids in serum were tested using LC-MS/MS method. Anthropometric and laboratory measurements were recorded. All samples were split into a training set and a test set (7/3 ratio).

Results: Total 54 variables were significantly different between obesity, T2DM and control groups (p-value < 0.05). In uni-variable logistic regression analysis, 44 variables were significantly associated with disease diagnosis. LASSO, RFE and RF algorithms jointly selected 7 optimal variables (Ala, His, Gln, IL-10, age, FBG, and AHR). The support vector machine (SVM) diagnostic model based on the 7 variables showed robust performance in both the training set (AUC = 0.998) and the validation set (AUC = 0.958). Obesity or T2DM patients had significantly increased Ala (p-value < 0.01) but decreased Gln, His and IL-10 (p-value < 0.01) in serum compared to healthy controls. Gln and His levels were positively correlated with IL-10 level (Cor = 0.46, 0.48, p-value < 0.001).

Conclusion: This study developed a 7 feature-based diagnostic model for obesity and T2DM and suggested that Ala, His, Gln, and IL-10 were involved in the common mechanisms and might be potential therapeutic targets.

Keywords: amino acids, fatty acids, diabetes mellitus, obesity, inflammation, diagnostic model

Introduction

Obesity is usually concomitant with diabetes mellitus type 2 (T2DM), both of which are prevalent metabolic afflictions, badly affecting human health and increasing the risks for other disorders, such as cardiovascular diseases and cancers.[1–3] Emerging studies have disclosed common genetic variations and complicated pathophysiological mechanisms shared by obesity and T2DM.[4,5] Moreover, combating the two afflictions exert mutual beneficial effect on each other, holding potential for designing effective intervention strategies and developing novel therapeutics.[6] Therefore, identifying common key indicators of obesity and T2DM will have significance for early diagnosis of the two disorders, thus improving clinical outcome.

T2DM is often accompanied by dyslipidemia, proper management of which is crucial for the clinical outcome.[7,8] Prevalence of diabetes and obesity drives the initiation and progression of non-alcoholic fatty liver disease.[9] A substantial body of evidence has established that amino acids play an important causative role in the pathogenesis of obesity and T2DM.[10,11] Moreover, alterations in parameters of liver function, such as alanine aminotransferase (ALT), aspartate

https://doi.org/10.2147/DMSO.S583656
Received: 4 December 2025
Accepted: 13 February 2026
Published: 3 March 2026

© 2026 Guo et al. This work is published and licensed by Dove Medical Press Limited. The full terms of this license are available at https://www.dovepress.com/terms.php and incorporate the Creative Commons Attribution – Non Commercial (unported, v4.0) License (http://creativecommons.org/licenses/by-nc/4.0/). By accessing the work you hereby accept the Terms. Non-commercial uses of the work are permitted without any further permission from Dove Medical Press Limited, provided the work is properly attributed. For permission for commercial use of this work, please see paragraphs 4.2 and 5 of our Terms (https://www.dovepress.com/terms.php).

aminotransferase (AST), and gamma-glutamyl transferase (GGT), are closely related to the progression of T2DM.[12] There is evidence that thyroid dysfunction is linked to T2DM and obesity clinically.[13] It is well-defined that inflammatory responses, especially driven by macrophages, are a crucial underlying mechanism of T2DM and obesity.[14,15] These findings indicate that fatty acids, amino acids, and clinical factors with reference to liver function, thyroid function and inflammation may hold promise as predictive factors of the two afflictions.

This prospective study enrolled 139 outpatients, including 33 control subjects, 81 obese patients and 25 diabetes patients, 7 of who were also obese patients. As a powerful analytical technique, liquid chromatography-tandem mass spectrometry (LC-MS/MS) characterized by outstanding specificity and sensitivity has been widely adopted in clinical research.[16,17] In this study, we utilized LC-MS/MS for laboratory measurement of amino acids and fatty acids. We integrated fatty acids, amino acids, and clinical characteristics into this research to identify critical features related to diagnosis of T2DM and obesity by using a combination of machine algorithms including uni-variate logistic regression analysis, least absolute shrinkage and selection operator (LASSO), recursive feature elimination (RFE), and random forest (RF). Among the most robust prediction models, support vector machines (SVMs) has been applied to predict risk for several diseases and yielded promising results.[18] The current study established an SVM-based diagnostic model using a training set and tested its diagnostic performance in the validation set. This study would shed light on understanding the molecular underpinnings of the two closely related afflictions and facilitate early effective intervention to delay T2DM onset.

Materials and Methods

Patients and Study Design

This study included 139 outpatients in Clinic of Metabolic Diseases, Shanxi Provincial Hospital of Traditional Chinese Medicine, from September 10, 2024, to December 31, 2024. There were 33 control patients, 81 obese patients and 25 diabetes patients, 7 of who were obese patients as well.

Complete records about symptoms and signs in traditional Chinese medicine (TCM), medical history, and clinical examination information. Diagnostic criteria and staging for simple obesity were established in accordance with the 2022 Update of the Clinical Practice Guidelines for Obesity published by the Korean Society for the Study of Obesity.[19] Obesity was staged as follows: Stage I, $25 \leq \text{BMI} < 30$; Stage II, $30 \leq \text{BMI} < 35$; Stage III, $\text{BMI} \geq 35$. Written informed consent was obtained from each participant. Exclusion criteria: younger than age of 18 years or older than 70 years; type I diabetes; gestational diabetes; secondary hypertension; secondary hyperlipidemia; severe complications of heart, liver, or kidney; mental disorders; regular factor score of dampness syndrome <100 with reference to TCM.

This trial was registered at the International Traditional Medicine Clinical Trial Registry (ITMCTR) under identifier ITMCTR2025002040 (Retrospective registration).

Anthropometric and Laboratory Measurements

At 8 to 9 am, every patient was measured for body weight, height, blood pressure (BP) and waist circumference. Blood samples were collected after an overnight fasting and tested for counts of five categories of blood cells and biochemical parameters related to plasma glucose, blood lipids, and renal function. An aliquot (5 mL) of blood sample was centrifuged at 560 g for 10 min at 4°C. Enzyme-linked immunosorbent assay (ELISA) was used for detecting 5-hydroxytryptamine receptor (HTR)2C, C1q/TNF-related protein (CTRP)9, interleukin (IL)-10, fibroblast growth factor (FGF)-21, monocyte chemoattractant protein (MCP)-1, growth/differentiation factor (GDF)-15 and tumor necrosis factor (TNF)-α in serum.

For quantitative determination of amino acids and fatty acids based on LC-MS/MS method, serum samples were prepared as follows: 400 μL methanol was added to 100 μL of serum sample. After centrifugation at 12,000 RPM at 4°C for 10 mins, all the supernatants were transferred to a new centrifuge tube, and evaporated to dryness. The dried extracts were re-dissolved in 150 μL of 2-chlorophenylalanine (4 ppm) in 80% methanol solution and filtered with a 0.22 μm membrane. Pooling 20 μL from each test sample as quality controls (QCs), the remaining samples were used for LC-MS detection as previously described.[20,21]

Missing Data Imputation

In order to deal with missing data occurred in all datasets, mice package (version 3.17.0, https://cran.r-project.org/web/packages/mice/index.html) in R language (version 4.3.1) was used for data imputation[22] (Table S1). However, for the four variables including impute time in range (TIR), hemoglobin (Hb)A1c, urine microalbumin, and creatinine, the number of missing samples reached 121, which was deemed inappropriate for missing data imputation. Consequently, these four indicators were excluded from the analysis.

Features Selections

Samples in the control group, the obesity group and the T2DM group were split into a training set and a test set using a ratio of 70%/30%. In the training set, uni-variate logistic regression analysis (https://cran.r-project.org/web/packages/rms/index.html) was used to identify the variables significantly associated with disease diagnosis from all variables including amino acids, fatty acids, and clinical characteristics. Subsequently, out of these significant features, key features were selected by LASSO,[23] RFE,[24] and RF[25] approaches, separately, using lars package (version 1.2, https://cran.r-project.org/web/packages/lars/index.html), caret package (version 6.0–76, https://cran.r-project.org/web/packages/caret), and randomForest package (https://cran.r-project.org/web/packages/randomForest/, version 4.6–14) in R language. The overlapped key features selected by all three approaches were then selected as the optimal features.

Construction and Validation of Diagnostic Model

In the training set, a diagnostic model based on the selected optimal features was constructed using SVM[26] with Sigmoid Kernel and 10-fold cross-validation in e1071 package (version 1.6–8) in R language (https://cran.r-project.org/web/packages/e1071). Performance of the model was assessed in both the training set and the validation set using pROC package[27] (https://cran.r-project.org/web/packages/pROC/index.html, version 1.14.0). The area under the receiver operating characteristic curve (AUROC) ranges from 0.5 to 1, with higher value indicating better diagnostic performance.[28]

Differential and Correlation Analysis

Optimal feature variables were compared between different groups in both the training set and the validation set. Correlation of the optimal features variables with clinical factors was analyzed using *Cor* function in R language (version 4.3.1).

Statistical Analysis

Normality distribution analysis of data was conducted using numerical method of Shapiro–Wilk test in R language (version 4.3.1). Continuous variables in normal distribution were expressed as mean ± SD. Differences between groups were measured for significance using Student's unpaired *t*-test and one-way analysis of variance (ANOVA) with Tukey's post hoc test. P-value <0.05 suggests statistical significance.

Results

A Total of 54 Variables Were Significantly Different Between the Control, Obesity and T2DM Groups

In contrast to raw data shown in Figure 1A, the resulting dataset after imputation was displayed in Figure 1B, in which all missing data was imputed by multiple imputations using *mice* package except for TIR, HbA1C, urine microalbumin, and creatinine.

This study tested 63 variables including 22 amino acids, 7 fat acids and 34 clinical characteristics, 53 of which showed non-normal distribution. Therefore, median values were tested for differences between the control, obesity and diabetes groups. As shown in Table 1, 54 variables were significantly different between groups (p-value < 0.05). Noticeably, Ala level was markedly higher in the obese or T2DM patients compared to the healthy controls with the highest p-values (5.1e-14 or 1.3 e-12, Figure 2). Yet, difference in Ala level between the obese and T2DM patients did not reach significance (p-value = 0.67, Figure 2).

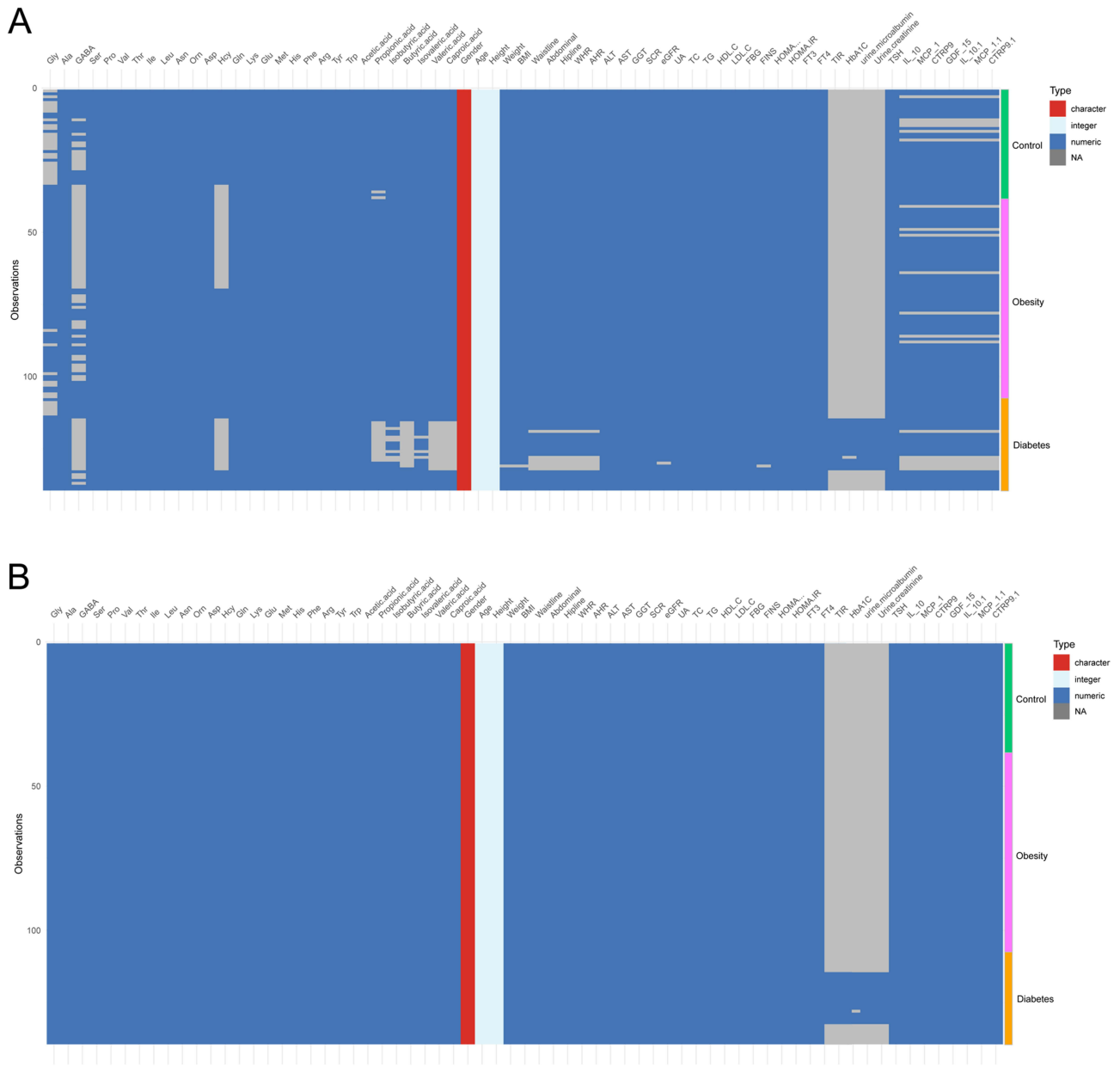

Figure 1 Graphical data display before (**A**) and after (**B**) data imputation.

Seven Optimal Features Were Selected Jointly by Three Approaches

We dichotomized all samples in each group into a training set and a validation set with a 7/3 ratio. The training set (N = 97) included 23 control samples, 57 obesity samples and 17 T2DM samples, while the validation set had 10 control samples, 24 obesity samples and 8 T2DM samples. In the training set, uni-variable logistic regression analysis found that 44 variables were significantly associated with disease diagnosis (p-value < 0.05, Table 2).

We subsequently employed LASSO, RFE and RF algorithms to select the optimal feature variables from the 44 disease diagnosis-related variables, respectively (Figure 3). As a result, 17 optimal feature variables were selected by LASSO, 10 optimal variables by RFE and 16 optimal variables by RF (Figure 3). A total of 7 optimal variables were shared by all three algorithms, including fasting blood glucose (FBG), alanine (Ala), histidine (His), glutamine (Gln), age, IL-10 and abdominaline hipline ratio (AHR) (Figure 4).

Table 1 Comparative Analysis of Amino Acids, Fatty Acids and Clinical Factors in Different Groups

ID	Group			P-value
	Control (N = 33)	Obesity (N = 81)	Diabetes (N = 25)	
Amino acids				
Gly	1.123 ± 3.309	11.772 ± 5.573	11.847 ± 4.642	2.20E-11
Ala	12.660 ± 3.261	25.658 ± 7.861	27.622 ± 9.377	3.00E-14
GABA	0.016 ± 0.018	0.012 ± 0.019	0.038 ± 0.026	7.30E-02
Ser	11.731 ± 2.038	10.226 ± 2.942	9.958 ± 1.962	1.20E-01
Pro	12.676 ± 3.522	13.386 ± 3.653	14.337 ± 3.083	9.20E-02
Val	15.722 ± 2.426	20.645 ± 4.185	22.023 ± 4.624	2.30E-08
Thr	11.095 ± 2.629	12.115 ± 4.623	7.836 ± 4.448	2.90E-02
Ile	10.507 ± 2.492	8.867 ± 3.227	8.023 ± 3.868	9.20E-03
Leu	13.174 ± 2.586	12.877 ± 3.177	13.285 ± 3.655	2.60E-01
Asn	6.727 ± 1.120	6.552 ± 3.020	8.648 ± 2.233	3.70E-04
Orn	6.718 ± 1.978	6.985 ± 4.504	5.989 ± 3.696	6.50E-01
Asp	3.934 ± 1.189	3.384 ± 1.927	1.215 ± 2.103	9.00E-04
Hcy	0.082 ± 0.029	0.068 ± 0.028	0.064 ± 0.032	2.50E-02
Gln	53.459 ± 7.483	36.532 ± 10.063	29.390 ± 8.505	9.00E-14
Lys	13.227 ± 2.976	11.685 ± 3.081	10.546 ± 3.288	4.60E-03
Glu	17.673 ± 6.134	15.664 ± 8.494	10.129 ± 12.631	2.70E-02
Met	6.369 ± 0.994	4.189 ± 1.560	3.319 ± 1.405	1.30E-10
His	6.582 ± 1.162	4.689 ± 1.319	3.816 ± 1.270	1.70E-13
Phe	15.630 ± 2.603	13.075 ± 3.323	10.115 ± 3.905	2.80E-08
Arg	11.460 ± 3.171	9.272 ± 2.652	7.184 ± 2.177	5.50E-07
Tyr	12.139 ± 2.470	9.872 ± 2.816	8.424 ± 3.150	4.00E-06
Trp	9.590 ± 1.390	8.771 ± 2.120	8.265 ± 2.361	6.40E-03
Fatty acids				
Acetic acid (100μL)	2.398 ± 0.754	1.675 ± 0.866	1.147 ± 0.715	8.20E-08
Propionic acid (100μL)	0.443 ± 0.158	0.226 ± 0.200	0.089 ± 0.136	7.90E-11
Isobutyric acid (100μL)	0.040 ± 0.008	0.035 ± 0.014	0.017 ± 0.027	1.80E-02
Butyric acid (100μL)	0.056 ± 0.022	0.072 ± 0.071	0.058 ± 0.036	3.40E-02
Isovaleric acid (100μL)	0.031 ± 0.015	0.027 ± 0.017	0.015 ± 0.023	2.80E-04
Valeric acid (100μL)	0.009 ± 0.006	0.010 ± 0.006	0.008 ± 0.011	7.70E-01
Caproic acid (100μL)	0.024 ± 0.008	0.021 ± 0.010	0.026 ± 0.017	1.10E-02
Clinical characteristics				
Gender (Male/Female)	7/26	12/69	16/9	1.06E-05
Age	27.000 ± 11.767	30.000 ± 8.719	49.000 ± 12.794	2.40E-06
Height	167.000 ± 8.063	163.000 ± 7.349	170.000 ± 6.180	3.00E-03
Weight	60.150 ± 10.440	80.400 ± 20.964	79.000 ± 17.061	4.10E-12
BMI	21.500 ± 2.441	30.400 ± 5.350	27.064 ± 5.475	2.20E-16
Waistline	77.000 ± 7.873	93.000 ± 12.721	94.000 ± 11.998	3.40E-11
Abdominaline	75.000 ± 10.282	98.000 ± 13.205	91.000 ± 11.770	5.90E-12
Hipline	94.000 ± 6.035	109.000 ± 9.890	101.000 ± 13.002	1.60E-03
WHR	0.810 ± 0.053	0.860 ± 0.077	0.878 ± 0.065	1.90E-05
AHR	0.450 ± 0.051	0.600 ± 0.075	0.545 ± 0.075	7.90E-15
ALT	12.300 ± 10.011	23.700 ± 29.096	30.600 ± 23.190	2.90E-08
AST	17.550 ± 4.837	20.500 ± 10.666	24.500 ± 13.521	1.90E-04
GGT	12.000 ± 12.964	24.270 ± 14.886	38.340 ± 32.950	1.80E-11
SCR	68.800 ± 10.790	64.900 ± 11.141	64.900 ± 12.313	2.70E-01

(*Continued*)

https://doi.org/10.2147/DMSO.S583656

Table 1 (Continued).

ID	Group			P-value
	Control (N = 33)	Obesity (N = 81)	Diabetes (N = 25)	
eGFR	109.700 ± 10.468	111.400 ± 13.767	108.240 ± 16.327	5.90E-01
UA	321.000 ± 66.606	393.000 ± 94.391	337.000 ± 101.014	1.70E-03
TC	4.260 ± 0.651	4.760 ± 0.903	4.970 ± 1.540	3.00E-04
TG	0.840 ± 1.210	1.540 ± 0.756	1.850 ± 2.847	2.70E-05
HDL-C	1.260 ± 0.273	1.100 ± 0.435	1.060 ± 0.273	2.30E-03
LDL-C	2.560 ± 0.484	3.170 ± 0.753	3.350 ± 0.714	2.10E-05
FBG	5.150 ± 0.379	5.320 ± 0.540	9.790 ± 4.015	1.00E-14
FINS	4.540 ± 2.318	11.370 ± 9.955	8.670 ± 7.096	4.40E-11
HOMA-β	57.692 ± 30.527	123.410 ± 186.287	26.120 ± 45.479	4.90E-14
HOMA-IR	1.075 ± 0.540	2.990 ± 10.919	1.740 ± 3.800	1.80E-10
FT3	4.880 ± 0.591	5.560 ± 0.566	5.450 ± 0.662	1.60E-04
FT4	10.420 ± 1.442	11.010 ± 1.471	12.130 ± 1.664	9.90E-03
TSH	2.264 ± 2.450	2.177 ± 1.517	1.956 ± 1.597	7.20E-01
5-HTR2C	7.487 ± 2.154	10.838 ± 3.013	9.697 ± 3.383	3.10E-07
FGF-21	129.887 ± 44.896	220.582 ± 69.028	201.032 ± 78.854	2.10E-07
TNF-α	46.077 ± 15.912	72.999 ± 22.701	67.792 ± 23.459	1.20E-07
GDF-15	639.541 ± 214.013	951.453 ± 279.174	1052.381 ± 278.701	2.60E-07
IL-10	34.226 ± 4.940	24.700 ± 6.420	17.164 ± 6.706	1.40E-13
MCP-1	196.762 ± 54.937	293.830 ± 86.263	306.982 ± 85.768	1.10E-07
CTRP9	452.452 ± 143.823	891.756 ± 318.437	707.405 ± 370.999	1.10E-08

Abbreviations: Gly, Glycine; Ala, alanine; GABA, gamma-aminobutyric acid; Ser, Serine; Pro, Proline; Val, Valine; Thr, Threonine; Ile, Isoleucine; Leu, Leucine; Asn, asparagine; Orn, ornithine; Asp, aspartic acid; Hcy, homocysteine; Gln, glutamine; Lys, lysine; Glu, glutamatic acid; Met, methionine; His, histidine; Phe, Phenylalanine; Arg, arginine; Tyr, Tyrosine; Trp, tryptophan; BMI, body mass index; WHR, Waistline Hipline Ratio; AHR, Abdominaline Hipline Ratio; ALT, alanine aminotransferase; AST, aspartate aminotransferase; GGT, gamma-glutamyl transferase; SCR, serum creatinine; eGFR estimated glomerular filtration rate; UA, uric acid; TC, cholesterol; TG, triglycerides; HDL-C, high density lipoprotein cholesterol; LDL-C, high density lipoprotein cholesterol; FBG, fasting blood glucose; FINS, fasting serum insulin; HOMA-β, homeostasis model assessment-β; HOMA-IR, HOMA-insulin resistance; FT3, free triiodothyronine; FT4, free thyroxine; TSH, thyroid stimulating hormone; 5-HTR2C, 5-hydroxytryptamine receptor 2C; FGF-21, fibroblast growth factor 21; TNF-α, tumor necrosis factor-α; GDF-15, growth/differentiation factor-15; IL-10, Interleukin-10; MCP-1, Monocyte chemoattractant protein-1; CTRP9, C1q/TNF-related protein 9.

A Seven-Feature Diagnostic Model Performed Well in Both the Training Set and the Test Set

An SVM model was constructed with FBG, Ala, His, Gln, Age, IL-10 and AHR. In the training set, the seven-feature diagnostic model showed robustness with a combined AUC of 0.998, higher than AUC values of single feature-based models (Figure 5A). Similarly, the model achieved a combined AUC value of 0.958 in the validation set, exceeding single feature-based models (Figure 5B). These results suggest robustness of the seven-feature model in both the training set and the test set.

Ala, Gln, and His Were Closely Related to IL-10 and AHR in Obese or T2DM Patients

Figure 6A and B showed comparative analysis of obesity or T2DM patients with control subjects in both the training set and the validation set for FBG, Ala, His, Gln, age, IL-10 and AHR. Unsurprisingly, diabetes or T2DM patients had obviously elevated Ala level and AHR value than control subjects in either the training set or the validation set (p-value < 0.01). On the contrary, Gln, His and IL-10 levels were much lower in diabetes or T2DM patients compared to control subjects in both sets (p-value < 0.01). Additionally, diabetes patients appeared to be older (p-value < 0.01) and had higher FBG levels (p-value < 0.001).

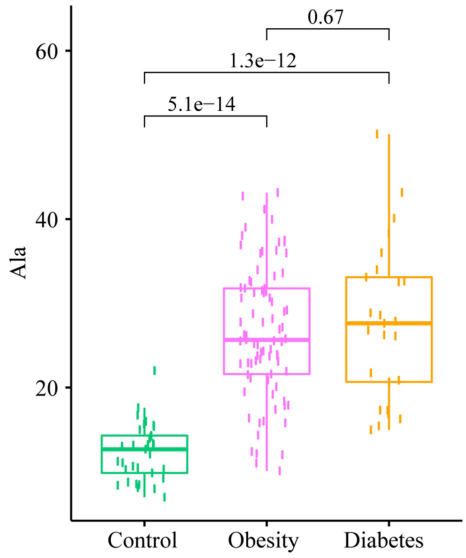

Figure 2 Differences in Ala level between control (green), obesity (purple) and diabetes (yellow) patients. P-values are $5.1e^{-14}$, $1.3e^{-12}$ and 0.67. Ala, alanine.

Correlations of 3 amino acids (Ala, Gln, and His) with 4 clinical factors (IL-10, FBG, AHR, and age) were analyzed to unravel their possible biological roles in the pathophysiology of the two diseases. Specifically, Ala level was positively correlated with AHR value (Cor = 0.602, p-value < 0.001, Table 3), while Gln and His levels were positively correlated with IL-10 level (Cor = 0.46, 0.48, p-value < 0.001, Table 3).

Table 2 Results of Uni-Variable Logistic Regression Analysis

ID	OR (95% CI)	p-value
22 Amino acids		
Gly	1.06 (1.04–1.08)	1.94E-08
Ala	1.04 (1.03–1.05)	9.02E-11
GABA	2.43 (0.01–4.78)	5.35E-01
Ser	0.97 (0.92–1.02)	2.39E-01
Pro	1.03 (0.99–1.07)	1.74E-01
Val	1.09 (1.06–1.12)	8.30E-08
Thr	0.99 (0.96–1.02)	6.60E-01
Ile	0.95 (0.91–0.99)	1.98E-02
Leu	1.01 (0.95–1.04)	8.74E-01
Asn	1.13 (1.06–1.21)	2.43E-04
Orn	1.01 (0.97–1.04)	7.08E-01
Asp	0.89 (0.83–0.95)	8.87E-04
Hcy	0.05 (0.10–0.17)	6.96E-03
Gln	0.96 (0.96–0.97)	2.76E-14
Lys	0.93 (0.90–0.97)	1.64E-03
Glu	0.99 (0.97–1.69)	1.08E-01
Met	0.80 (0.75–0.85)	1.18E-09
His	0.78 (0.74–0.83)	7.48E-13
Phe	0.90 (0.87–0.93)	2.22E-08
Arg	0.89 (0.86–0.92)	1.80E-09
Tyr	0.90 (0.87–0.94)	2.85E-06
Trp	0.93 (0.86–0.99)	3.58E-02
7 Fatty acids		
Acetic acid (100μL)	0.72 (0.64–0.82)	3.85E-06
Propionic acid (100μL)	0.20 (0.12–0.32)	5.10E-09

(*Continued*)

https://doi.org/10.2147/DMSO.S583656

Table 2 (Continued).

ID	OR (95% CI)	p-value
Isobutyric acid (100μL)	0.04 (0.01–0.18)	3.03E-03
Butyric acid (100μL)	2.09 (0.21–3.92)	5.31E-01
Isovaleric acid (100μL)	0.34 (0.03–2.88)	1.01E-01
Valeric acid (100μL)	1.57 (0.16–2.21)	8.53E-02
Caproic acid (100μL)	1.48 (0.22–3.29)	3.48E-01
34 Clinical characteristics		
Gender (Male/Female)	1.42 (1.07–1.88)	1.76E-02
Age	1.03 (1.01–1.12)	5.18E-03
Height	0.99 (0.98–1.09)	7.09E-01
Weight	1.04 (1.01–1.21)	3.65E-03
BMI	1.04 (1.02–1.05)	4.13E-04
Waistline	1.02 (1.01–1.09)	2.03E-04
Abdominaline	1.02 (1.01–1.03)	2.97E-05
Hipline	1.03 (1.01–1.33)	2.37E-02
WHR	1.58 (1.13–2.28)	6.05E-05
AHR	1.69 (1.25–1.83)	1.22E-04
ALT	1.01 (1.01–1.02)	7.94E-04
AST	1.02 (1.01–1.04)	2.11E-02
GGT	1.02 (1.01–1.05)	2.59E-05
SCR	0.99 (0.98–1.01)	4.94E-01
eGFR	0.99 (0.98–1.01)	3.31E-01
UA	0.99 (0.98–1.01)	2.32E-01
TC	1.24 (1.10–1.41)	9.03E-04
TG	1.14 (1.03–1.26)	1.53E-02
HDL-C	0.77 (0.57–1.04)	9.44E-02
LDL-C	1.30 (1.10–1.53)	2.30E-03
FBG	1.15 (1.11–1.18)	9.52E-12
FINS	1.02 (1.01–1.03)	3.28E-02
HOMA-β	0.99 (0.98–1.01)	6.82E-01
HOMA-IR	1.01 (0.99–1.02)	5.28E-01
FT3	1.48 (1.21–1.81)	2.58E-04
FT4	1.10 (1.01–1.19)	2.39E-02
TSH	0.94 (0.88–1.00)	6.70E-02
5-HTR2C	1.07 (1.03–1.11)	4.73E-04
FGF-21	1.02 (1.01–1.08)	5.15E-03
TNF-α	1.02 (1.01–1.12)	2.94E-04
GDF-15	1.09 (1.08–1.11)	2.45E-08
IL-10	0.95 (0.94–0.96)	1.86E-14
MCP-1	0.98 (0.96–0.99)	9.89E-06
CTRP9	0.97 (0.96–0.99)	6.39E-05

Abbreviations: OR, odds ratio; 95% CI, confidence interval; Gly, Glycine; Ala, alanine; GABA, gamma-aminobutyric acid; Ser, Serine; Pro, Proline; Val, Valine; Thr, Threonine; Ile, Isoleucine; Leu, Leucine; Asn, asparagine; Orn, ornithine; Asp, aspartic acid; Hcy, homocysteine; Gln, glutamine; Lys, lysine; Glu, glutamatic acid; Met, methionine; His, histidine; Phe, Phenylalanine; Arg, arginine; Tyr, Tyrosine; Trp, tryptophan; BMI, body mass index; WHR, Waistline Hipline Ratio; AHR, Abdominaline Hipline Ratio; ALT, alanine aminotransferase; AST, aspartate aminotransferase; GGT, gamma-glutamyl transferase; SCR, serum creatinine; eGFR estimated glomerular filtration rate; UA, uric acid; TC, cholesterol; TG, triglycerides; HDL-C, high density lipoprotein cholesterol; LDL-C, high density lipoprotein cholesterol; FBG, fasting blood glucose; FINS, fasting serum insulin; HOMA-β, homeostasis model assessment-β; HOMA-IR, HOMA-insulin resistance; FT3, free triiodothyronine; FT4, free thyroxine; TSH, thyroid stimulating hormone; 5-HTR2C, 5-hydroxytryptamine receptor 2C; FGF-21, fibroblast growth factor 21; TNF-α, tumor necrosis factor-α; GDF-15, growth/differentiation factor-15; IL-10, Interleukin-10; MCP-1, Monocyte chemoattractant protein-1; CTRP9, C1q/TNF-related protein 9.

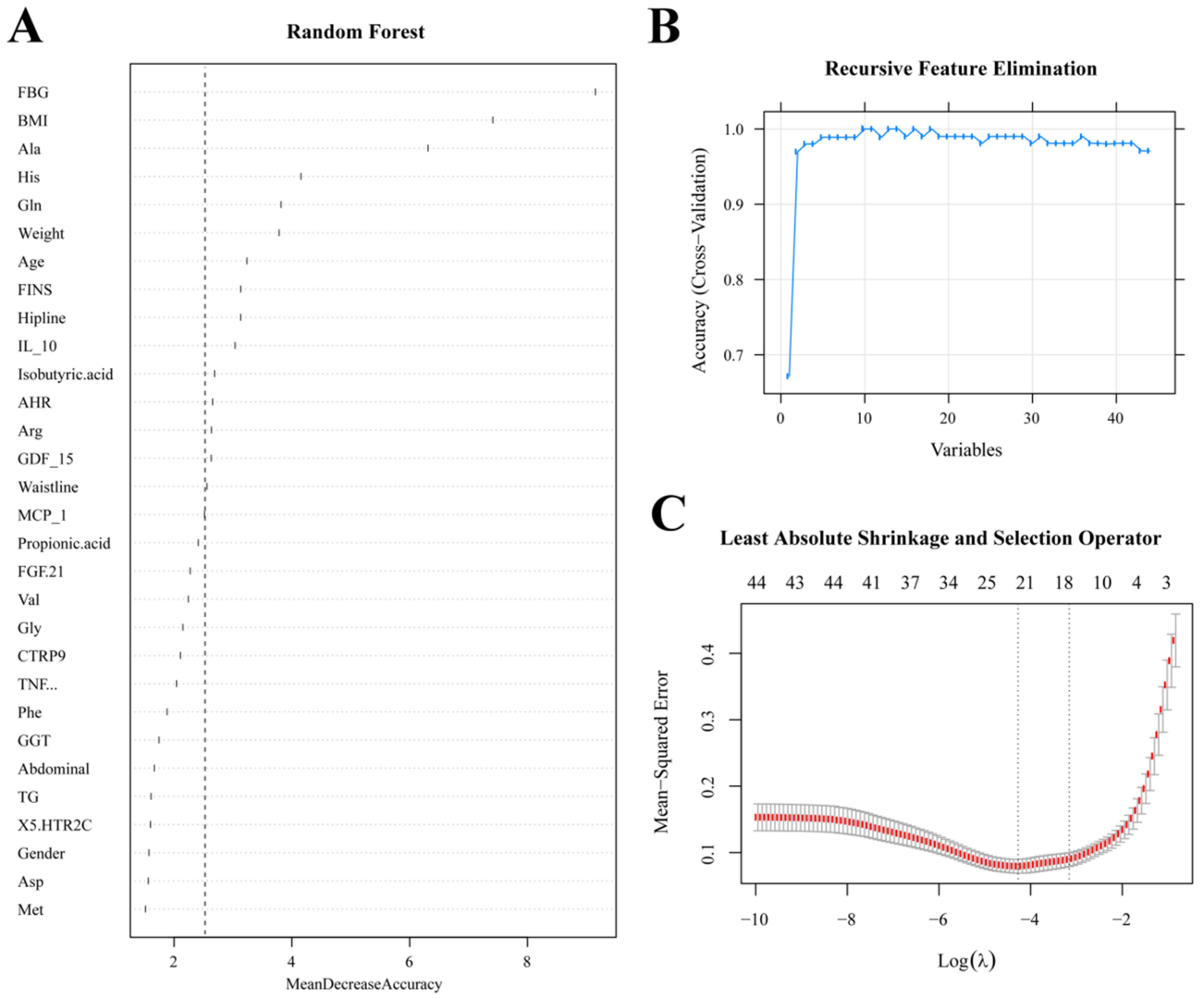

Figure 3 Selection of optimal feature variables by random forest (RF) (**A**), recursive feature elimination (RFE) (**B**), and least absolute shrinkage and selection operator (LASSO) (**C**).

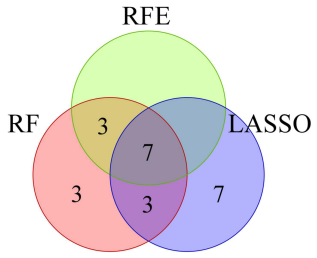

Figure 4 Venn diagrams showed the optimal features shared by RF, RFE, and LASSO.

Discussion

T2DM, a multifactorial disorder, has an increasing incidence worldwide, the accurate diagnosis of which remains a challenging task.[29] Increasing studies have demonstrated that obesity is a well-established risk factor of utmost importance for T2DM.[30,31] The facts that the majority of subjects with pre-diabetes are asymptomatic, and that clinic-based screening is cost-effective, makes machine learning a valuable tool with great potential for early diagnosis in

https://doi.org/10.2147/DMSO.S583656

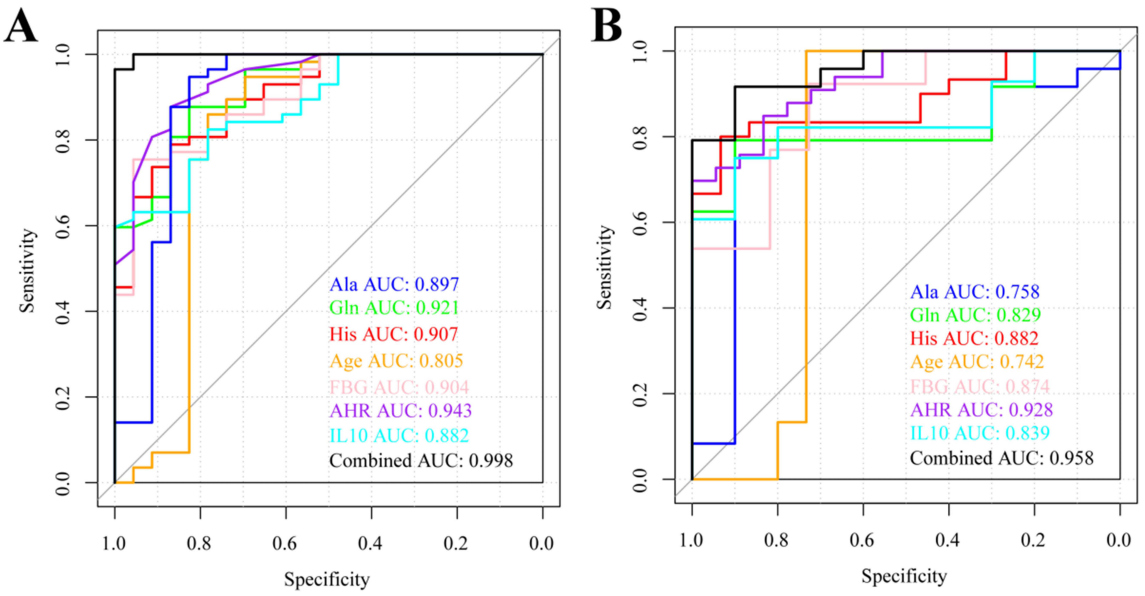

Figure 5 Receiver operating characteristic (ROC) curves for assessing diagnostic power of the model in the training set (**A**) and the validation set (**B**).

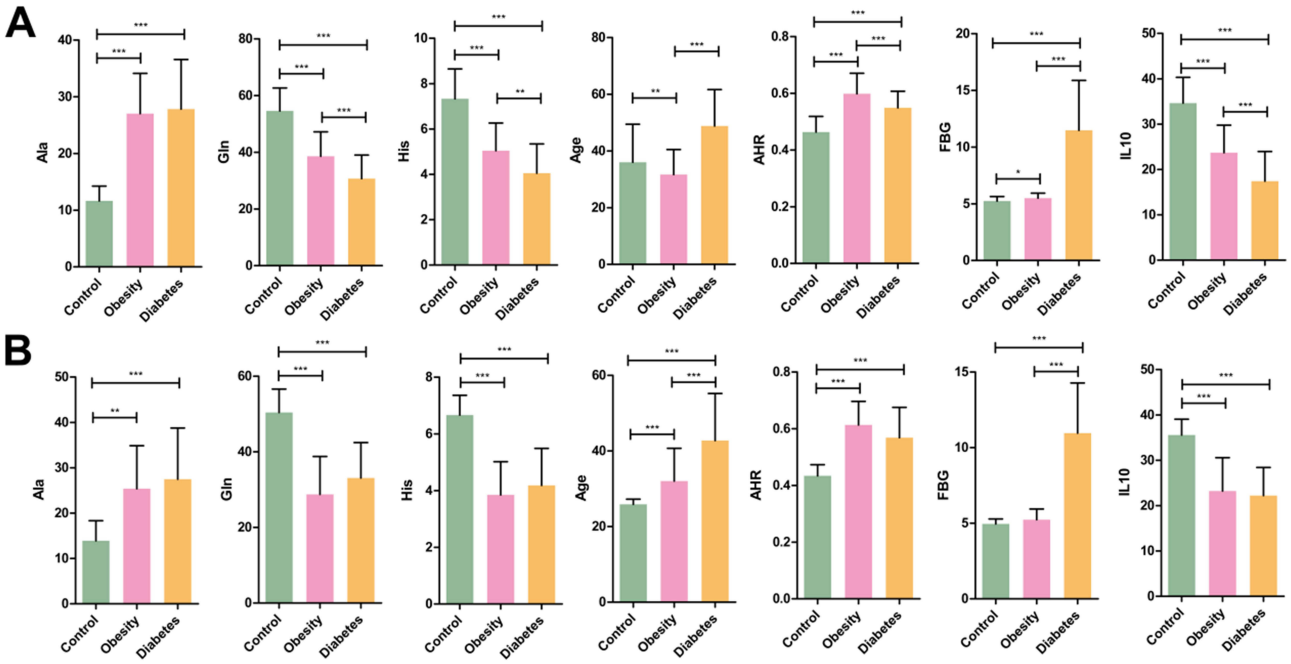

Figure 6 Column diagrams of 7 optimal features in the training set (**A**) and the validation set (**B**). Ala, Gln, His, age, AHR, FBG, and IL-10 are significantly different between healthy controls, obesity and diabetes patients. *p < 0.05; **p < 0.01; ***p < 0.005.
Abbreviations: Gln, glutamine; His, histidine; AHR, Abdominaline Hipline Ratio; FBG, fasting blood glucose; IL-10, Interleukin-10.

T2DM.[32] In this study, fatty acids, amino acids, and clinical features were integrated for the first time to identify key characteristic variables associated with obesity and T2DM, aiming to develop a diagnostic model for both conditions. By employing machine learning algorithms, a diagnostic model based on seven characteristic variables (Ala, His, Gln, IL-10, age, FBG, and AHR) was successfully constructed. The model exhibited excellent discriminatory performance, achieving AUC values of 0.998 and 0.958 in the training and validation sets, respectively. This performance is notably superior to that of several previously reported diagnostic models for T2DM. For instance, Lu et al combined machine learning with network methods for T2DM prediction; using private medical insurance patient records, they constructed a bipartite

Table 3 Correlation Coefficients of the Optimal Feature Variables

ID	Ala	Gln	His
Age	0.0630	−0.0383	−0.0257
AHR	0.6020	−0.2069	−0.2270
FBG	0.1271	−0.2884	−0.2943
IL-10	−0.3902	0.4590	0.4815

Abbreviations: AHR, Abdominaline Hipline Ratio; FBG, fasting blood glucose; IL-10, Interleukin-10.

graph projected into a patient network and trained eight machine learning algorithms with specific features, achieving AUC values ranging from 0.79 to 0.91.[33] A rural cohort study in Henan Province, China, showed that a T2DM prediction model built with laboratory data had good predictive efficacy, with AUC values between 0.811 and 0.872.[34] Abdullah et al fitted a multivariate logistic regression model to predict T2DM risk in Malaysia using environmental and genetic risk factors, yielding AUC values of 0.75–0.83.[35] Xiong et al developed predictive models for T2DM risk by applying five machine learning techniques with tenfold cross-validation, achieving AUC values between 0.86 and 0.87 across all models.[36] In comparison, the SVM-based model in this study achieves higher AUC values in both training and validation sets, which may be attributed to the comprehensive integration of multi-dimensional features (amino acids, inflammatory cytokines, anthropometric parameters, and demographic factors) and the rigorous joint feature selection by LASSO, RFE, and RF algorithms, ensuring that the selected features fully capture the pathophysiological characteristics shared by obesity and T2DM. This study helps decipher the pathophysiological link between obesity and T2DM and may allow for rapid and accurate clinical diagnosis and early interventions.

Missing data has attracted wide-spread interest because of its potential to introduce bias and elicit inaccurate results.[37] Imputation offers a promising solution to existence of missing data occurred often in clinical research.[38,39] This study applied *mice* package for missing data imputation to minimize bias and yield valid results. The identified 7 feature variables with diagnostic value in obesity and T2DM consisted of Ala, His, Gln, IL-10, FBG, age, and AHR. Amino acids are one of the most commonly dys-regulated pathways in gestational diabetes.[40] There is in vivo evidence that ALT is up-regulated in liver of humans with T2DM and that systemic Ala metabolism participates in glycaemic control.[41] Moreover, the raised serum ALT is positively related to higher BMI and poor glycaemic control in T2DM patients.[42] Consistently, in the present study, we observed that Ala and AHR were increased in T2DM and obesity. Moreover, a positive correlation existed between Ala and AHR. Collectively, these findings support that Ala is a contributing factor to the two metabolic disorders. A recent study provides evidence that oral His administration is helpful in improving glycemic control in T2DM, possibly by suppressing inflammation and oxidative stress.[43] In line, this study showed that serum His level was decreased in the obesity or T2DM patients compared to healthy controls, indicating that His plays a inhibitory role in the pathophysiology of the two diseases. However, another study holds a different view that dietary intake of His increases the risk of T2DM in general Chinese residents.[44] These contradictory results call for more in-depth studies in this field.

Previous study finds that Gln is inversely correlated with BMI and insulin resistance index, and alleviates insulin resistance by suppressing inflammatory response of skeletal muscle, suggesting that Gln is a key amino acid in T2DM.[45] Consistently, glutamine/glutamate (Gln/Glu) ratio is reported to be a protective factor for both T2DM and obesity and possesses predictive value for T2DM risk.[46] These results showed that serum Gln level was decreased in the obesity or T2DM patients, which was in agreement with these previously reported findings. Chronic low-grade inflammation elicits insulin resistance and β-cell dysfunction, promoting initiation and development of obesity and T2DM.[47] Deregulation of anti-inflammatory IL-10 plays an important role in the development of the two closely related diseases.[48] Unsurprisingly, this study found that the obese and T2DM patients had significantly decreased serum IL-10 level which was positively correlated with Gln and His levels. It implies that regulating IL-10 is a possible mechanism by which Gln and His suppress the development of obesity and T2DM. Additionally, this study unveiled diagnostic potential of three clinical factors including age, FBG and AHR. Approximately, a half of DM patients are older than 65 years.[49] Monitoring FBG

has critical implications in improving diagnosis and management of T2DM patients.[50] These results suggest that Ala, His, Gln, and IL-10 are involved in the common underlying mechanisms of obesity and T2DM through regulating inflammation, and may be promising therapeutic targets to treat the two diseases.

Some limitations exist regarding the study research. Firstly, this study had limited sample size, especially requiring more T2DM samples. Secondly, it is necessary to confirm diagnostic power of the 7 feature variables-based model in more large validation datasets. Thirdly, we obtained these results from machine learning analysis. Further clinical experiments were warranted to confirm the power of the diagnostic model and uncover more specific downstream mechanisms.

Conclusion

Integrating amino acids, fatty acids with clinical characteristics, with the help of machine learning algorithms, this study established a diagnostic model based on 7 feature variables (Ala, His, Gln, IL-10, FBG, age, and AHR) in obesity and T2DM. These results provide novel insights into the pathophysiological mechanisms linking the two diseases and recommend that Ala, His, Gln, and IL-10 might be appealing therapeutic candidates. Further research is required to improve efficacy and accuracy of the model.

Data Sharing Statement

The datasets generated during and/or analysed during the current study are available from the corresponding author on reasonable request.

Ethics Approval and Informed Consent

This study was performed in line with the principles of the Declaration of Helsinki. Approval was granted by the Ethics Committee of Shanxi Provincial Hospital of Traditional Chinese Medicine (No. 2024KY-07039) and with the patients' informed consent.

Author Contributions

Conceptualization and methodology were primarily handled by X.X.G. (Xiaoxia Guo) and W.J.C. (Wenjing Cheng). X.L. H. (Xuliang Hao) provided supervision, project administration, and funding acquisition. Experimental investigation and data curation were performed by W.J.C., L.Z. (Lu Zhang), and L.L. (Lei Lv). Formal analysis and validation were conducted by L.Z. and L.L. The original draft was prepared by X.X.G. and W.J.C., with review and editing by all authors. All authors gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

Funding

This study was supported by Central Government Guidance Funds for Local Scientific and Technological Development, China (YDZJSX2024D073), and The Provincial Traditional Chinese Medicine Science and Technology Project (2023ZYYA006), and Shanxi Provincial Science and Technology Innovation Talent Team (202204051002028).

Disclosure

The author(s) report no conflicts of interest in this work.

References

1. Boutari C, DeMarsilis A, Mantzoros CS. Obesity and diabetes. *Diabet Res Clin Pract*. 2023;202:110773. doi:10.1016/j.diabres.2023.110773
2. Harborg S, Kjærgaard KA, Thomsen RW, Borgquist S, Cronin-Fenton D, Hjorth CF. New horizons: epidemiology of obesity, diabetes mellitus, and cancer prognosis. *J Clin Endocrinol Metab*. 2024;109(4):924–935. doi:10.1210/clinem/dgad450
3. Salama M, Balagopal B, Fennoy I, Kumar S. Childhood obesity, diabetes and cardiovascular disease risk. *J Clin Endocrinol Metab*. 2023;108 (12):3051–3066. doi:10.1210/clinem/dgad361
4. Ruze R, Liu T, Zou X, et al. Obesity and type 2 diabetes mellitus: connections in epidemiology, pathogenesis, and treatments. *Front Endocrinol*. 2023;14:1–23. doi:10.3389/fendo.2023.1161521
5. Bashir M, Fagier Y, Ahmed B, Konje JC. An overview of diabetes mellitus in pregnant women with obesity. *Best Pract Res Clin Obstet Gynaecol*. 2024;93:1–24. doi:10.1016/j.bpobgyn.2024.102469

6. Shah MA, Haris M, Faheem HI, et al. Cross-talk between obesity and diabetes: introducing polyphenols as an effective phytomedicine to combat the dual sword diabesity. *Curr Pharm Des*. 2022;28(19):1523–1542. doi:10.2174/1381612828666220628123224
7. Goldberg RB. Dyslipidemia in diabetes: when and how to treat? *Endocrinol Metab Clin North Am*. 2022;51(3):603–624. doi:10.1016/j.ecl.2022.02.011
8. Kalra S, Raizada N. Dyslipidemia in diabetes. *Indian Heart J*. 2024;76 Suppl 1(Suppl 1):S80–S82. doi:10.1016/j.ihj.2023.11.002
9. Stefan N, Cusi K. A global view of the interplay between non-alcoholic fatty liver disease and diabetes. *Lancet Diabetes Endocrinol*. 2022;10(4):284–296. doi:10.1016/s2213-8587(22)00003-1
10. Mosley JD, Shi M, Agamasu D, et al. Branched-chain amino acids and type 2 diabetes: a bidirectional Mendelian randomization analysis. *Obesity*. 2024;32(2):423–435. doi:10.1002/oby.23951
11. Vanweert F, Schrauwen P, Phielix E. Role of branched-chain amino acid metabolism in the pathogenesis of obesity and type 2 diabetes-related metabolic disturbances BCAA metabolism in type 2 diabetes. *Nutr Diabetes*. 2022;12(1):1–13. doi:10.1038/s41387-022-00213-3
12. Rafaqat S, Sattar A, Khalid A, Rafaqat S. Role of liver parameters in diabetes mellitus - a narrative review. *Endocr Regul*. 2023;57(1):200–220. doi:10.2478/enr-2023-0024
13. Awad AS, Alshahrni FMM, Alhawyan FS, et al. Inference of link among diabetes, obesity, and thyroid dysfunction in data from a clinic at Saudi Arabia. *Bioinformation*. 2021;17(1):119–125. doi:10.6026/97320630017119
14. Rohm TV, Meier DT, Olefsky JM, Donath MY. Inflammation in obesity, diabetes, and related disorders. *Immunity*. 2022;55(1):31–55. doi:10.1016/j.immuni.2021.12.013
15. Ying W, Fu W, Lee YS, Olefsky JM. The role of macrophages in obesity-associated islet inflammation and β-cell abnormalities. *Nat Rev Endocrinol*. 2020;16(2):81–90. doi:10.1038/s41574-019-0286-3
16. Rappold BA. Review of the use of liquid chromatography-tandem mass spectrometry in clinical laboratories: part II-operations. *Ann Lab Med*. 2022;42(5):531–557. doi:10.3343/alm.2022.42.5.531
17. Ma Y, Wang D, Li H, et al. Liquid chromatography-tandem mass spectrometry in clinical laboratory protein measurement. *Clin Chim Acta*. 2024;562:1–19. doi:10.1016/j.cca.2024.119846
18. Xie S, Ogden RT. Functional support vector machine. *Biostatistics*. 2024;25(4):1178–1194. doi:10.1093/biostatistics/kxae007
19. Haam JH, Kim BT, Kim EM, et al. Diagnosis of obesity: 2022 update of clinical practice guidelines for obesity by the Korean Society for the Study of Obesity. *J Obes Metab Syndr*. 2023;32(2):121–129. doi:10.7570/jomes23031
20. Virág D, Király M, Drahos L, et al. Development, validation and application of LC-MS/MS method for quantification of amino acids, kynurenine and serotonin in human plasma. *J Pharm Biomed Anal*. 2020;180:113018. doi:10.1016/j.jpba.2019.113018
21. Fuertig R, Ceci A, Camus SM, Bezard E, Luippold AH, Hengerer B. LC-MS/MS-based quantification of kynurenine metabolites, tryptophan, monoamines and neopterin in plasma, cerebrospinal fluid and brain. *Bioanalysis*. 2016;8(18):1903–1917. doi:10.4155/bio-2016-0111
22. Tan FES, Jolani S, Verbeek H. Guidelines for multiple imputations in repeated measurements with time-dependent covariates: a case study. *J Clin Epidemiol*. 2018;102:107–114. doi:10.1016/j.jclinepi.2018.06.006
23. Ma J, Couturier DL, Heritier S, Marschner IC. Penalized likelihood estimation of the proportional hazards model for survival data with interval censoring. *Int J Biostat*. 2022;18(2):553–575. doi:10.1515/ijb-2020-0104
24. Karthik KV, Rajalingam A, Shivashankar M, Ganjiwale A. Recursive feature elimination-based biomarker identification for open neural tube defects. *Curr Genomics*. 2022;23(3):195–206. doi:10.2174/1389202923666220511162038
25. Safia NS. Prediction of breast cancer through random forest. *Curr Med Imaging*. 2023;19(10):1144–1155. doi:10.2174/1573405618666220930150625
26. Liang Z, Jiang Z, Wu W, et al. Study and classification of porosity stress sensitivity in shale gas reservoirs based on experiments and optimized support vector machine algorithm for the Silurian Longmaxi Shale in the Southern Sichuan Basin, China. *ACS Omega*. 2022;7(37):33167–33185. doi:10.1021/acsomega.2c03393
27. Robin X, Turck N, Hainard A, et al. pROC: an open-source package for R and S+ to analyze and compare ROC curves. *BMC Bioinf*. 2011;12(1):77. doi:10.1186/1471-2105-12-77
28. Schipper A, Rutten M, van Gammeren A, et al. Machine learning-based prediction of hemoglobinopathies using complete blood count data. *Clin Chem*. 2024;70(8):1064–1075. doi:10.1093/clinchem/hvae081
29. Chang L, Fukuoka Y, Aouizerat BE, Zhang L, Flowers E. Prediction of weight loss to decrease the risk for type 2 diabetes using multidimensional data in Filipino Americans: secondary analysis. *JMIR Diabetes*. 2023;8:e44018. doi:10.2196/44018
30. Han Y, Hu H, Liu Y, Wang Z, Liu D. Nomogram model and risk score to predict 5-year risk of progression from prediabetes to diabetes in Chinese adults: development and validation of a novel model. *Diabetes Obes Metab*. 2023;25(3):675–687. doi:10.1111/dom.14910
31. Chandrasekaran P, Weiskirchen R. The role of obesity in type 2 diabetes mellitus-an overview. *Int J Mol Sci*. 2024;25(3):1–21. doi:10.3390/ijms25031882
32. Duan D, Kengne AP, Echouffo-Tcheugui JB. Screening for diabetes and prediabetes. *Endocrinol Metab Clin North Am*. 2021;50(3):369–385. doi:10.1016/j.ecl.2021.05.002
33. Lu H, Uddin S, Hajati F, Moni MA, Khushi M. A patient network-based machine learning model for disease prediction: the case of type 2 diabetes mellitus. *Appl Intell*. 2022;52(3):2411–2422. doi:10.1007/s10489-021-02533-w
34. Zhang L, Wang Y, Niu M, Wang C, Wang Z. Machine learning for characterizing risk of type 2 diabetes mellitus in a rural Chinese population: the Henan Rural Cohort Study. *Sci Rep*. 2020;10(1):4406. doi:10.1038/s41598-020-61123-x
35. Abdullah N, Abdul Murad NA, Mohd Haniff EA, et al. Predicting type 2 diabetes using genetic and environmental risk factors in a multi-ethnic Malaysian cohort. *Public Health*. 2017;149:31–38. doi:10.1016/j.puhe.2017.04.003
36. Xiong XL, Zhang RX, Bi Y, Zhou WH, Yu Y, Zhu DL. Machine learning models in type 2 diabetes risk prediction: results from a cross-sectional retrospective study in Chinese adults. *Curr Med Sci*. 2019;39(4):582–588. doi:10.1007/s11596-019-2077-4
37. Heymans MW, Twisk JWR. Handling missing data in clinical research. *J Clin Epidemiol*. 2022;151:185–188. doi:10.1016/j.jclinepi.2022.08.016
38. Ryan-Despraz J, Wissler A. Imputation methods for mixed datasets in bioarchaeology. *Archaeol Anthropol Sci*. 2024;16(11):1–21. doi:10.1007/s12520-024-02078-2
39. Pang J, Liu X. Evaluation of missing data imputation methods for human osteometric measurements. *Am J Biol Anthropol*. 2023;181(4):666–676. doi:10.1002/ajpa.24787

40. Alesi S, Ghelani D, Rassie K, Mousa A. Metabolomic biomarkers in gestational diabetes mellitus: a review of the evidence. *Int J Mol Sci*. 2021;22(11):1–15. doi:10.3390/ijms22115512
41. Okun JG, Rusu PM, Chan AY, et al. Liver alanine catabolism promotes skeletal muscle atrophy and hyperglycaemia in type 2 diabetes. *Nat Metab*. 2021;3(3):394–409. doi:10.1038/s42255-021-00369-9
42. Ahamed F, Karim MR, Haque MA, et al. Study on alanine aminotransferase in patients of type 2 diabetes mellitus. *Mymensingh Med J*. 2021;30(2):343–350.
43. Warmbrunn MV, Attaye I, Aron-Wisnewsky J, et al. Oral histidine affects gut microbiota and MAIT cells improving glycemic control in type 2 diabetes patients. *Gut Microbes*. 2024;16(1):1–20. doi:10.1080/19490976.2024.2370616
44. Liu Y, Wang H, Liang Y, et al. Dietary intakes of methionine, threonine, lysine, arginine and histidine increased risk of type 2 diabetes in Chinese population: does the mediation effect of obesity exist? *BMC Public Health*. 2023;23(1):1–13. doi:10.1186/s12889-023-16468-z
45. Dollet L, Kuefner M, Caria E, et al. Glutamine regulates skeletal muscle immunometabolism in type 2 diabetes. *Diabetes*. 2022;71(4):624–636. doi:10.2337/db20-0814
46. Han F, Xu C, Hangfu X, et al. Circulating glutamine/glutamate ratio is closely associated with type 2 diabetes and its associated complications. *Front Endocrinol*. 2024;15:1–10. doi:10.3389/fendo.2024.1422674
47. Prasad M, Chen EW, Toh SA, Gascoigne NRJ. Autoimmune responses and inflammation in type 2 diabetes. *J Leukoc Biol*. 2020;107(5):739–748. doi:10.1002/jlb.3mr0220-243r
48. Ren Y, Zhao H, Yin C, et al. Adipokines, hepatokines and myokines: focus on their role and molecular mechanisms in adipose tissue inflammation. *Front Endocrinol*. 2022;13:1–26. doi:10.3389/fendo.2022.873699
49. Bellary S, Kyrou I, Brown JE, Bailey CJ. Type 2 diabetes mellitus in older adults: clinical considerations and management. *Nat Rev Endocrinol*. 2021;17(9):534–548. doi:10.1038/s41574-021-00512-2
50. Antwi-Baffour S, Mensah BT, Armah DNO, Ali-Mustapha S, Annison L. Comparative analysis of glycated haemoglobin, fasting blood glucose and haematological parameters in Type-2 diabetes patients. *BMC Res Notes*. 2023;16(1):256. doi:10.1186/s13104-023-06520-x

Diabetes, Metabolic Syndrome and Obesity

Dovepress
Taylor & Francis Group

Publish your work in this journal

Diabetes, Metabolic Syndrome and Obesity is an international, peer-reviewed open-access journal committed to the rapid publication of the latest laboratory and clinical findings in the fields of diabetes, metabolic syndrome and obesity research. Original research, review, case reports, hypothesis formation, expert opinion and commentaries are all considered for publication. The manuscript management system is completely online and includes a very quick and fair peer-review system, which is all easy to use. Visit http://www.dovepress.com/testimonials.php to read real quotes from published authors.

Submit your manuscript here: https://www.dovepress.com/diabetes-metabolic-syndrome-and-obesity-journal