

Original Article

Diet quality and biochemical parameters in individuals with type 2 diabetes across BMI categories: A cross-sectional study

Cemaliye Süt Kurt MSc¹, Nazife Hürer PhD¹, Rojina Deniz Aygül Elma PhD¹, Deniz Engin Gök MD², Seyit Mehmet Mercanligil PhD³

¹Department of Nutrition and Dietetics, Faculty of Health Sciences, Cyprus International University, Nicosia, Turkish Republic North Cyprus

²Department of Endocrine and Metabolic Diseases, Memorial Ankara Hospital, Ankara, Türkiye

³Department of Nutrition and Dietetics, Faculty of Health Sciences, Hasan Kalyoncu University, Gaziantep, Türkiye

Background and Objectives: Type 2 diabetes mellitus (T2DM) is closely associated with obesity and metabolic disturbances. Although diet quality has been linked to metabolic outcomes, its role relative to adiposity remains unclear. This study aimed to evaluate differences in biochemical parameters across body mass index (BMI) categories and examine relationships between diet quality and biochemical markers. **Methods and Study Design:** This cross-sectional study included 150 individuals with T2DM aged 46–60 years attending an endocrinology outpatient clinic between September 2023 and May 2024. Participants were classified as being of normal weight, overweight, or obese based on BMI. Diet quality was assessed using three-day food consumption records and calculated using the Healthy Eating Index-2015 (HEI-2015). Biochemical parameters were obtained from medical records. Between-group comparisons were performed using one-way ANOVA with Bonferroni post hoc tests and Pearson correlation analysis was used to assess associations. Benjamini–Hochberg false discovery rate (BH-FDR) correction was applied for multiple testing. **Results:** Several biochemical parameters differed significantly across BMI categories ($p < 0.05$). Obese individuals had higher triglyceride, uric acid, C-reactive protein, liver function, and fasting insulin levels, whereas normal-weight individuals had higher high-density lipoprotein cholesterol and estimated glomerular filtration rate. No significant differences were observed in HEI-2015 scores across BMI groups ($p > 0.05$). Weak correlations between diet quality and biochemical markers were not significant after BH-FDR correction. **Conclusions:** Biochemical parameters differed across BMI categories, highlighting the role of adiposity in metabolic risk. Associations between diet quality and biochemical markers were limited, suggesting that BMI-based stratification may be more informative than diet quality alone.

Key Words: obesity, type 2 diabetes, diet quality, biochemical parameters

INTRODUCTION

Type 2 diabetes mellitus is a chronic metabolic disease characterized by hyperglycemia that develops as a result of insulin resistance and/or impaired insulin secretion.¹ The incidence of T2DM is increasing worldwide, with obesity, physical inactivity, unhealthy eating habits, and aging being the main reasons. According to data from the International Diabetes Federation (IDF), 537 million adults worldwide lived with diabetes in 2021 and this number was predicted to reach 783 million by 2045.² In Türkiye, the prevalence of diabetes has been reported as approximately 14.5%.³

Body mass index is a simple and widely used obesity indicator obtained by dividing an individual's body weight by height squared. The World Health Organization (WHO) classifies BMI values as “normal” in the range of 18.5–24.9 kg/m², “overweight” in the range of 25–29.9 kg/m², and “obese” in the range of ≥ 30 kg/m².⁴ Obesity, particularly with increased visceral adiposity, is

known to play a role in the development of T2DM through mechanisms such as insulin resistance and β -cell dysfunction.⁵ Diet quality is a multidimensional indicator that evaluates the extent to which an individual's eating habits are compatible with health. Healthy diet quality is characterized by the consumption of vegetables, fruits, whole grains, and healthy fatty acids, with low levels of saturated fat, salt, and sugar. Diet quality can be evaluated by tools such as the Healthy Eating Index, the Mediterranean Diet Score (MDS), or the Alternative Healthy Eating Index (AHEI).⁶ In the literature, it has been shown

Corresponding Author: Cemaliye Süt Kurt, Department of Nutrition and Dietetics, Faculty of Health Sciences, Cyprus International University, Nicosia, North Cyprus
Tel: +90 5303008846

Email: cemaliye.sut@lokmanhekim.edu.tr

Manuscript received 13 March 2026. Initial review completed 13 April 2026. Revision accepted 16 June 2026.

doi: 10.6133/apjcn.202608_35(4).0014

that glycated hemoglobin (HbA1c) levels are lower, glucose metabolism is better regulated, and the risk of cardiovascular complications is reduced in individuals with high diet quality.^{7,8} The main biochemical parameters monitored in individuals with T2DM include fasting glucose, fasting insulin, HbA1c, lipid profile (low-density lipoprotein cholesterol [LDL-C], high-density lipoprotein cholesterol [HDL-C], triglyceride, total cholesterol), kidney function (urea, creatinine), and liver function (alanine aminotransferase [ALT], aspartate aminotransferase [AST]). These indicators are essential assessment criteria in diabetes management, as they reflect an individual's metabolic control and risk of complications.¹

Although studies in the literature have addressed the diet quality and biochemical parameters of individuals with T2DM separately, research on the combined evaluation of these two variables in terms of different BMI categories is limited. However, the eating habits and metabolic responses of obese and normal individuals may differ, and the multifaceted examination of the relationships among these variables is important for the creation of individualized nutrition plans.

Previous studies have consistently shown that obesity and increased adiposity are strongly associated with insulin resistance, dyslipidemia, chronic low-grade inflammation, impaired liver function, and altered renal function in individuals with T2DM.⁹⁻¹¹ In contrast, higher diet quality has been associated with improved glycemic control and cardiometabolic outcomes, but these associations may be weaker or less consistent in clinical populations with T2DM because biochemical parameters are influenced by multiple factors including adiposity, diabetes duration, medication use, comorbidities, and metabolic heterogeneity.¹²⁻¹⁴ Therefore, based on previous studies suggesting stronger and more direct relationships between adiposity and metabolic alterations than between diet quality indices and individual biochemical markers, we hypothesized that metabolic and biochemical parameters would differ significantly across BMI categories, whereas the associations between diet quality and biochemical markers would be weaker.

The primary objective of this study was to evaluate differences in biochemical parameters across BMI categories in individuals with T2DM. A secondary objective was to examine the relationships between diet quality and biochemical parameters.

This study aims to evaluate relationships between diet quality and biochemical parameters in individuals with T2DM, with a particular focus on differences across BMI categories. Unlike previous studies that examined these factors independently, the present study provides a combined evaluation of dietary and metabolic profiles within a single clinical population. Importantly, stratifying participants by BMI allows for a more detailed understanding of how adiposity may modify the relationship between diet quality and metabolic risk. This approach may produce clinically relevant insights by identifying BMI-specific patterns that could guide more individualized and targeted medical nutrition therapy strategies in the management of T2DM.

METHODS

Sample selection

This cross-sectional study aimed to investigate relationships between diet quality and biochemical parameters across BMI categories in individuals with T2DM attending an endocrinology outpatient clinic in Ankara, Türkiye. The study was conducted between September 2023 and May 2024 with 150 individuals with T2DM aged 46–60 years who were admitted to the Department of Endocrinology and Metabolic Diseases on an outpatient basis. A priori power analysis was conducted using G*Power software (version 3.1.9.7, Heinrich Heine University, Düsseldorf, Germany). Assuming an effect size of 0.30, statistical power of 90%, and type I error level of 0.05, the minimum required sample size for comparing mean values across three groups using one-way analysis of variance (ANOVA) was estimated to be 141 participants. To account for potential data loss and to increase the reliability of the study, a total of 150 individuals with T2DM were included. The study was carried out with approval from the Cyprus International University Scientific Research and Publication Ethics Committee (date: 02.06.2023, number: EKK22-23/015/016). Only individuals who used oral antidiabetic drugs and did not have psychiatric illnesses were included in the study. The study was conducted in accordance with the Declaration of Helsinki and all participants provided written and verbal informed consent prior to participation.

Data collection

The questionnaire form prepared for this study was administered face-to-face by a researcher in an endocrinology and metabolism outpatient clinic to patients who voluntarily agreed to participate in the study. The questionnaire form was developed by the researchers based on previously published studies in the relevant literature.^{12,13} With this questionnaire form, demographic data, diseases other than T2DM, three-day food consumption records, body weight and height, BMI, and relevant biochemical parameters were collected for all participants.

Anthropometric measurements

Body weight and height: Body weight was measured using a portable bioelectrical impedance analysis device (SC-240 MA, Tanita, Tokyo, Japan), with capacity of 200 kg and accuracy of 0.1 kg. Height was measured using a stadiometer (SECA 213, SECA, Hamburg, Germany) while participants stood barefoot, with feet together and head positioned in the Frankfort plane.¹⁵

Body Mass Index (BMI): BMI was calculated as weight (kg) divided by height squared (m²). According to the WHO classification, BMI values were categorized as underweight (<18.5 kg/m²), normal (18.5–24.9 kg/m²), overweight (25.0–29.9 kg/m²), or obese (≥30.0 kg/m²).⁴ Statistical comparisons were performed according to these BMI categories, with participants classified as being of normal weight, overweight, or obese.

Dietary assessment

Three-Day Food Consumption Records: Food consumption is generally determined by individual recall or the record-keeping method. These records are typically based

on 24-hour periods and the record-keeping may be repeated over durations of 3, 5, 7, or more days. This is a frequently used method.¹⁵ In the present study, the three-day food consumption records (FCRs) of the patients were obtained in face-to-face interviews with patients using the food consumption recording method, with the three days including two weekdays and one day from the weekend. For the objective evaluation of the amount of food consumed, an appropriate food and food photography catalog was used.¹⁶ These dietary records were evaluated using the current version of the Nutrition Information Systems program (BeBiS, version 7.2, EBISpro for Windows, Stuttgart, Germany).

Determination of Diet Quality (Healthy Eating Index-2015): The dietary intake of the participants was assessed using the three-day FCRs described above. Participants were asked to record all foods and beverages consumed, including portion sizes, in detail. To improve the accuracy of portion size estimation, a food and food photography catalog was used¹⁶ and portion sizes were converted into grams to calculate daily intake amounts. Dietary data were analyzed to determine daily energy and nutrient intake using BeBiS nutrition analysis software. The resulting data on food groups and nutrients were then used to calculate Healthy Eating Index-2015 (HEI-2015) component scores. The HEI-2015 is a standardized index developed to assess adherence to the 2015–2020 Dietary Guidelines for Americans and it consists of 13 components.¹⁶ These components are classified as either “adequacy” or “moderation” and are scored on the basis of energy density (per 1000 kcal) according to standard criteria. Food-group intake (for groups including fruits, vegetables, grains, dairy, and protein sources) and nutrient components (such as sodium, added sugars, and saturated fats) were converted into the corresponding HEI-2015 component scores. When necessary, local dietary items were classified according to their closest equivalents within the HEI-2015 framework to ensure consistency in scoring. The HEI-2015 was considered an appropriate index for this study due to its compatibility with dietary intake data and its widespread use in clinical research. The total HEI-2015 score, calculated by summing all component scores, ranges from 0 to 100, with higher scores indicating better diet quality.¹⁷ The calculated sodium value only indicates the amount obtained directly from food. The HEI-2015 score was calculated based on data from the three-day FCRs for all participants.

Biochemical Findings: Venous blood samples were collected from all participants into vacuum tubes in the morning after at least 8 hours of overnight fasting. Blood samples were processed in the medical biochemistry laboratory of the hospital in which the study was carried out to obtain serum, which was analyzed after an appropriate centrifugation process (10 minutes, 3000 rpm). The biochemical parameters analyzed were as follows: fasting blood glucose (mg/dL), total cholesterol (mg/dL), HDL-C (mg/dL), LDL-C (mg/dL), triglyceride (mg/dL), creatinine (mg/dL), urea (mg/dL), uric acid (mg/dL), and estimated glomerular filtration rate (eGFR: mL/min/1.73m²), calculated with the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) formula.^{18–20} C-reactive protein (CRP; mg/dL), liver function parameters ALT (U/L)

and AST (U/L), thyroid-stimulating hormone (TSH, mU/L), vitamin B12 (pg/mL), folic acid (ng/mL), iron (µg/dL), ferritin (ng/mL), zinc (µg/dL), fasting insulin (µU/mL), and HbA1c (%) were also measured. A majority of the biochemical parameters were analyzed using enzymatic colorimetric methods with a fully automated biochemistry analyzer (Cobas 6000, Roche Diagnostics GmbH, Mannheim, Germany).^{21,22} Hormonal and vitamin parameters such as TSH, insulin, vitamin B12, folic acid, and ferritin were measured by electrochemiluminescence immunoassay using the Cobas e601 module (Roche Diagnostics GmbH).²³ HbA1c was measured using high-performance liquid chromatography (Tosoh HLC-723G8, Tosoh Corporation, Tokyo, Japan). eGFR values were automatically calculated based on serum creatinine using the CKD-EPI equation.^{18–20} All analyses were performed in accordance with the internal quality control procedures of the hospital's laboratory.

Statistical analysis

Statistical analyses were performed using IBM SPSS Statistics 26.0 (IBM Corp., Armonk, NY, USA). The primary outcome was differences in biochemical parameters across BMI categories, while secondary analyses examined associations between diet quality and biochemical markers. Frequency analysis was used to describe the sociodemographic characteristics and health statuses of the participants. The chi-square test was applied to compare categorical variables between groups.

The normality of continuous variables, including biochemical parameters and FCRs, was assessed using the Kolmogorov–Smirnov test, and homogeneity of variances was evaluated using the Levene test. For variables that did not meet the assumption of normality, square root and logarithmic transformations were applied to achieve an approximate normal distribution. All subsequent analyses were conducted using parametric tests based on these assumptions. For comparisons between two independent groups, the Student independent-samples t-test was used. For comparisons among three independent groups according to BMI categories, one-way ANOVA was applied. When a statistically significant overall difference was detected, post hoc pairwise comparisons were performed using Bonferroni correction.

Pearson correlation analysis was conducted to evaluate the relationships between variables. The strength of the correlation coefficient was interpreted as follows: 0.10–0.39, weak; 0.40–0.69, moderate; 0.70–0.89, strong; 0.90–1.00, very strong.¹⁸ To account for the potential inflation of type I error due to multiple testing, Benjamini–Hochberg false discovery rate (BH-FDR) correction was applied to correlation analyses and group comparisons. FDR-adjusted p-values, reported as q-values, were calculated separately for each BMI subgroup column for correlation analyses and across all biochemical comparisons for group comparisons. Statistical significance after correction was defined as $q < 0.05$. All results were evaluated at a 95% confidence interval. Unadjusted p-values of < 0.05 were considered nominally significant, while FDR-adjusted q-values of < 0.05 were considered statistically significant after correction for multiple testing.

RESULTS

Sociodemographic characteristics and distribution of chronic diseases other than T2DM

There was no statistically significant difference between the BMI groups in terms of sex, presence of chronic diseases other than T2DM, or distribution of chronic diseases ($p > 0.05$). However, educational attainment statistically significantly differed according to BMI groups ($p < 0.05$). The characteristics of the participants are shown in Table 1.

Age, height, body weight, BMI, and duration of diabetes

The results of one-way ANOVA for comparisons of age and T2DM duration among normal-weight, overweight, and obese individuals are shown in Table 2. There was no statistically significant difference between these groups in terms of age or T2DM duration ($p > 0.05$). However, body weight and BMI values of the obese participants were found to be statistically significantly higher than those of normal-weight and overweight participants ($p < 0.05$), as seen in Table 2.

Biochemical results

The HDL-C values of T2DM patients with normal BMI were statistically significantly higher than those of the overweight group ($p_1 < 0.05$). Triglyceride values were found to be statistically significantly lower in the normal-weight group compared to the obese group ($p_2 < 0.05$). Uric acid values of obese individuals with T2DM were statistically significantly higher compared to normal-

weight and overweight individuals with T2DM ($p_2 < 0.05$, $p_3 < 0.05$). The eGFR values of normal-weight individuals were statistically significantly higher compared to the obese group ($p_2 < 0.05$). CRP values of individuals with normal BMI were statistically significantly lower compared to both the overweight and obese groups ($p_1 < 0.05$, $p_2 < 0.05$). ALT values were statistically significantly lower in the normal-weight and overweight groups compared to the obese group ($p_2 < 0.05$, $p_3 < 0.05$). AST values were statistically significantly lower in the overweight group compared to the obese group ($p_3 < 0.05$). Fasting insulin values were statistically significantly lower in the group with normal BMI compared to the overweight and obese groups ($p_1 < 0.05$, $p_2 < 0.05$). In addition, fasting insulin levels were statistically significantly lower in the overweight group compared to the obese group ($p_3 < 0.05$). These findings are presented in Table 4.

FCR results according to BMI groups

There was no significant difference between the groups in terms of total diet quality score, macronutrient components (including protein and fatty acids), sodium intake, or food group components (including total fruits, whole fruits, total vegetables, dark green vegetables and legumes, whole grains, dairy products, and processed grains) ($p > 0.05$). On the other hand, seafood consumption was found to be statistically significantly higher in the overweight group compared to normal-weight and obese individuals ($p < 0.05$), as shown in Table 5.

Table 1. Sociodemographic characteristics and distribution of chronic diseases other than T2DM according to BMI groups

Variables	Normal (n=22)		Overweight (n=51)		Obese (n=77)		Total (n=150)		χ^2	$p^\dagger$
	n	%	n	%	n	%	n	%		
Sex										
Male	8	36.4	19	37.3	26	33.8	53	35.3	0.175	0.916
Female	14	63.6	32	62.7	51	66.2	97	64.7		
Educational status										
Elementary school	0	0	1	2.0	11	14.3	12	8.0	17.662	0.024*
Middle school	3	13.6	3	5.9	4	5.2	10	6.7		
High school	8	36.4	11	21.6	20	26.0	39	26.0		
Associate/bachelor's degree	8	36.4	33	64.6	31	40.2	72	48.0		
Graduate degree	3	13.6	3	5.9	11	14.3	17	11.3		
Chronic diseases other than T2DM										
Yes	14	63.6	34	66.7	52	67.5	100	66.7	0.117	0.943
No	8	36.4	17	33.3	25	32.5	50	33.3		
Chronic diseases other than T2DM										
Cardiovascular diseases	8	36.4	18	35.3	26	33.8	52	34.6	11.426	0.875
Gastrointestinal diseases	0	0	2	3.9	1	1.3	3	2.0		
Musculoskeletal disorders	0	0	0	0	1	1.3	1	0.7		
Respiratory system diseases	0	0	2	3.9	0	0	2	1.3		
Cancer	1	4.5	1	2	1	1.3	3	2.0		
Biliary and liver diseases	0	0	1	2	3	3.9	4	2.7		
Endocrine disorders other than T2DM	5	22.7	10	19.6	16	20.7	31	20.7		
Neurological disorders	0	0	0	0	1	1.3	1	0.7		
Skin disorders	0	0	0	0	2	2.6	2	1.3		

n: frequency, %: percentage, c: statistical significance between overweight and obese group.

† Chi-square analysis

* $p < 0.05$

Table 2. Age, height, body weight, BMI, and duration of diabetes according to BMI groups

Variables	Normal (n=22)	Overweight (n=51)	Obese (n=77)	<i>p</i>
	$\bar{X} \pm SD$	$\bar{X} \pm SD$	$\bar{X} \pm SD$	
Mean age (years)	46.9±10.7	49.1±9.72	48.5±11.4	0.723
Height (m)	1.67±0.09	1.66±0.1	1.65±0.09	0.688
Body weight (kg)	64.4±7.44	76.3±9.77	92.4±11.8	0.001 ^{a,b,c*}
BMI (kg/m ²)	23.0±1.13	27.4±1.38	33.8±2.95	0.001 ^{a,b,c*}
T2DM duration (years)	6.59±6.84	4.43±5.35	5.34±5.55	0.320

BMI: body mass index; T2DM: type 2 diabetes mellitus

^a statistical significance between normal and overweight groups

^b statistical significance between normal and obese groups

^c statistical significance between overweight and obese groups. Post hoc comparisons were performed using the Bonferroni test.

**p* < 0.05

Table 3. Age, height, body weight, BMI, and duration of diabetes according to sex

Variables	Female (n=97)	Male (n=53)	Total (n=150)	<i>p</i> [†]
	$\bar{X} \pm SD$	$\bar{X} \pm SD$	$\bar{X} \pm SD$	
Mean age (years)	47.8±11.6	49.8±8.88	48.5±10.7	0.262
Height (m)	1.61±0.06	1.76±0.07	1.66±0.10	0.001*
Body weight (kg)	77.9±13.12	91.7±13.96	82.8±14.9	0.001*
BMI (kg/m ²)	30.1±4.73	29.8±4.63	30.0±4.68	0.653
T2DM duration (years)	5.34±5.99	4.98±5.15	5.21±5.69	0.713

BMI: body mass index; T2DM: type 2 diabetes mellitus

[†] Student t-test

**p* < 0.05

Relationships between diet quality and biochemical parameters

To address the risk of false-positive findings due to multiple testing, BH-FDR correction was applied to the Pearson correlation analyses as shown in Table 6 and the corresponding FDR-adjusted *p*-values (*q*-values; calculated separately within each BMI column, *m* = 20) were obtained. After BH-FDR correction, none of the correlations remained statistically significant at the FDR threshold. Although the nominal (uncorrected) *p*-values suggested a low positive correlation between total cholesterol and FCR-based diet quality total scores in the overall sample (*p* = 0.016) and low positive correlations between urea and diet quality total scores in the overweight group (*p* = 0.005) and the overall sample (*p* = 0.012), these associations did not retain statistical significance after adjustment for multiple comparisons (*q* > 0.05). Therefore, the correlations presented in Table 5 should be interpreted as exploratory or nominal findings rather than FDR-confirmed associations.

Comparison of biochemical findings of T2DM patients according to diet quality groups

To address the potential inflation of type I error due to multiple comparisons, BH-FDR correction was applied to the Student t-test *p*-values as reported in Table 6 and the corresponding FDR-adjusted *p*-values (*q*-values; calculated across all biochemical comparisons, *m* = 20) were obtained. In the unadjusted analyses, total cholesterol and LDL-C levels were lower in the group with poor diet quality compared to the group with diet quality to improve (total cholesterol: *p* = 0.011; LDL-C: *p* = 0.017). However, after BH-FDR correction, these between-group

differences were no longer statistically significant (total cholesterol: *q* = 0.170; LDL-C: *q* = 0.170). Overall, none of the biochemical parameters differed significantly between FCR-based diet quality groups after correction for multiple testing (*q* > 0.05), as shown in Table 7.

DISCUSSION

The present study evaluated the relationship between diet quality and biochemical parameters across BMI categories in individuals with T2DM. The key contribution of this study lies in its combined assessment of dietary quality and metabolic markers within BMI-stratified groups, providing a more nuanced understanding of how adiposity may influence these relationships. Our findings suggest that metabolic alterations are more strongly associated with BMI-related differences than with diet quality alone, highlighting the importance of considering adiposity when interpreting diet-metabolism interactions. This BMI-based stratification may help to identify subgroup-specific metabolic patterns, which could be relevant for developing more tailored and effective medical nutrition therapy approaches in clinical practice.

The mean age of the normal-weight, overweight, and obese individuals participating in this study was 46.9 ± 10.7, 49.1 ± 9.71, and 48.5 ± 11.4 years, respectively. In all three groups, the majority of the participants were female. The mean duration of diabetes was 6.59 ± 6.84 years in the normal-weight group, 4.32 ± 5.37 years in the overweight group, and 5.32 ± 5.55 years in the obese group. The similar distribution of both age and duration of diabetes across BMI groups indicates that the study population was demographically comparable. Furthermore, these demographic characteristics are

Table 4. Biochemical findings of individuals with type 2 diabetes according to BMI groups[†]

Biochemical parameters	Normal (n=22)	Overweight (n=51)	Obese (n=77)	<i>p</i>
	$\bar{X} \pm SD$ (min-max)	$\bar{X} \pm SD$ (min-max)	$\bar{X} \pm SD$ (min-max)	
Fasting blood glucose (mg/dL)	116.0±42.5 (80-262)	122.8±67.7 (75-416)	115.7±40.7 (83-305)	0.960
Total cholesterol (mg/dL)	194.2±29.8 (151-277)	200.2±60.4 (25-347)	206.7±43.7 (103-340)	0.396
HDL-C (mg/dL)	52.4±11.5 (37-86)	47.5±12.5 (29-91)	48.4±12.9 (25.0-83.8)	0.039 ^{a*}
LDL-C (mg/dL)	116.7±30.5 (65.0-176.6)	132.7±46.4 (35.2-249.2)	124.4±35.6 (47.6-235.0)	0.631
Triglyceride (mg/dL)	143.0±97.3 (38-447)	149.3±69.0 (38-328)	190.6±117.5 (52-659)	0.029 ^{b*}
Creatinine (mg/dL)	0.69±0.20 (0.10-1.08)	0.77±0.16 (0.51-1.10)	0.82±0.27 (0.45-2.50)	0.013 ^{b*}
Urea (mg/dL)	25.4±6.80 (12.3-40.0)	25.1±8.46 (7.00-57.7)	27.7±11.9 (8.56-97.7)	0.877
Uric acid	4.96±1.76 (3.00-9.90)	5.08±1.58 (2.50-10.2)	5.96±3.11 (2.40-29.0)	0.019 ^{b*} 0.013 ^{c*}
eGFR	103.3±10.5 (78.0-117.2)	98.9±13.7 (60.8-128.6)	95.7±14.7 (54.7-126.5)	0.009 ^{b*}
CRP (mg/L)	2.04±3.33 (0.01-15.5)	3.44±3.38 (0.30-18.3)	4.77±7.43 (0.42-55.1)	0.001 ^{a*} 0.001 ^{b*}
ALT (U/L)	18.1±10.1 (3.07-46.0)	20.3±10.3 (7-56)	29.2±23.0 (8.00-145.1)	0.007 ^{b*} 0.026 ^{c*}
AST (U/L)	20.1±8.23 (11-43)	19.1±7.39 (10-45)	23.7±13.9 (10.0-76.1)	0.024 ^{c*}
TSH (mIU/mL)	3.39±5.15 (0.01-20.0)	1.82±1.19 (0.05-8.23)	2.21±1.52 (0.08-10.8)	0.031 ^{a*}
Vitamin B12 (pg/mL)	521.9±238.1 (5.00-1231.0)	488.5±360.6 (175-2493)	466.0±231.8 (115-1651)	0.688
Folic acid (ng/mL)	8.02±3.79 (0.01-16.5)	9.03±4.66 (2.59-23.0)	8.46±3.91 (3.40-24.3)	0.523
Iron (ng/mL)	80.9±52.2 (10.5-240.0)	90.1±41.6 (25-250)	90.3±46.5 (9-300)	0.664
Ferritin (ng/mL)	87.1±78.7 (3.53-277.0)	99.3±108.0 (4.00-548.3)	85.5±86.3 (3.92-408.6)	0.812
Zinc (mg/dL)	84.0±30.3 (35-200)	87.5±34.5 (50-298)	81.0±19.5 (40.0-145.5)	0.412
Fasting insulin (mIU/L)	9.26±4.14 (3.03-16.8)	14.4±9.10 (2.79-51.7)	18.4±10.7 (3.79-71.03)	0.018 ^{a*} 0.001 ^{b*} 0.012 ^{c*}
HbA1c (%)	6.36±1.33 (5.00-10.9)	6.55±1.81 (4.90-14.0)	6.64±1.44 (5.0-13.8)	0.660

[†]Post hoc comparisons were performed using the Bonferroni test^a statistical significance between normal and overweight groups^b statistical significance between normal and obese groups^c statistical significance between overweight and obese groups.**p* < 0.05

consistent with previous studies conducted with individuals with T2DM, which reported similar age ranges and disease durations.^{24,25} Importantly, these findings support the internal validity of the present study by minimizing the risk of demographic confounding across BMI groups.

With respect to biochemical parameters, significant differences were observed across BMI groups (*p* < 0.05). Higher HDL-C levels in the normal BMI group compared to the overweight group (*p* < 0.05), together with elevated triglyceride levels in the obese group (*p* < 0.05), support the well-established relationship between increased adiposity and adverse lipid profiles. Similarly, the higher uric acid levels observed in obese individuals (*p* < 0.05) may reflect increased metabolic burden and insulin re-

sistance associated with excess adiposity.^{26,27} Inflammatory and insulin resistance markers further support this pattern. The significant increase in CRP and fasting insulin levels with increasing BMI (*p* < 0.05) indicates that obesity is closely associated with chronic low-grade inflammation and metabolic dysfunction. This can be explained by the role of adipose tissue as an active endocrine organ that secretes pro-inflammatory cytokines. In this context, the concept of “meta-inflammation” provides a useful framework for understanding the underlying mechanisms linking obesity to insulin resistance.^{28,29} These findings are also in line with emerging evidence highlighting the role of adipose tissue dysfunction and lipotoxicity in driving metabolic disturbances beyond traditional anthropometric measures.

Table 5. Healthy Eating Index (HEI-2015) scores of patients with type 2 diabetes mellitus derived from food consumption records according to BMI groups[†]

Nutritional component	Normal (n=22)	Overweight (n=51)	Obese (n=77)	<i>p</i>
	$\bar{X} \pm SD$	$\bar{X} \pm SD$	$\bar{X} \pm SD$	
FCR - Total Protein	3.60±1.06	3.58±0.93	3.55±0.96	0.967
FCR - Total Fruit	1.34±1.41	1.63±1.39	2.12±1.59	0.099
FCR - Full Fruit	2.58±2.26	2.99±1.93	3.62±1.80	0.081
FCR - Total Vegetables	2.82±1.61	2.71±1.35	2.80±1.62	0.942
FCR - Dark Greens and Legume	4.27±1.62	4.32±1.64	4.08±1.75	0.718
FCR - Whole Grains	4.50±4.05	3.63±4.25	4.01±4.17	0.711
FCR - Milk Group	5.31±2.86	4.49±2.70	5.09±2.75	0.371
FCR - Seafood	3.03±1.46	3.76±1.16	3.39±1.25	0.021 ^{a*}
FCR - Fatty Acids	3.50±3.00	3.46±3.03	3.25±3.05	0.905
FCR - Sodium	6.13±3.94	6.54±3.36	6.14±3.31	0.793
FCR - Added Sugar	8.73±2.68	8.13±2.56	8.38±2.93	0.693
FCR - Saturated Fat	4.54±3.41	3.71±3.27	3.67±2.90	0.491
FCR - Processed Grain	8.04±4.09	5.38±3.62	6.74±3.91	0.023 ^{a*}
FCR - Diet Quality Score	58.4±11.8	54.3±10.5	56.8±11.4	0.291

FCR: food consumption record; a: statistical significance between normal and overweight groups.

[†]Post hoc comparisons were performed using the Bonferroni test

**p* < 0.05

Table 6. Correlations between FCR-based diet quality scores and biochemical parameters of patients with type 2 diabetes mellitus according to BMI groups

Biochemical parameters	FCR diet quality total score			
	Normal (n = 22)	Overweight (n = 51)	Obese (n = 77)	Total (n = 150)
Fasting blood glucose				
r	0.104	0.142	-0.090	0.027
p	0.646	0.321	0.436	0.747
q (FDR)	0.940	0.644	0.949	0.928
Total cholesterol				
r	0.378	0.198	0.173	0.197
p	0.082	0.163	0.132	0.016*
q (FDR)	0.547	0.543	0.949	0.160
HDL-C				
r	-0.027	0.268	0.037	0.113
p	0.905	0.057	0.748	0.169
q (FDR)	0.961	0.325	0.949	0.928
LDL-C				
r	0.267	0.052	0.041	0.055
p	0.229	0.715	0.723	0.502
q (FDR)	0.911	0.794	0.949	0.928
Triglyceride				
r	0.072	-0.265	0.054	-0.030
p	0.752	0.060	0.640	0.714
q (FDR)	0.940	0.325	0.949	0.928
Creatinine				
r	-0.151	0.173	0.048	0.015
p	0.501	0.225	0.676	0.858
q (FDR)	0.911	0.643	0.949	0.928
Urea				
r	0.216	0.389	0.093	0.204
p	0.335	0.005*	0.420	0.012*
q (FDR)	0.911	0.100	0.949	0.160
Uric acid				
r	0.168	0.067	0.007	0.057
p	0.456	0.640	0.949	0.492
q (FDR)	0.911	0.794	0.949	0.928
eGFR				
r	0.005	-0.222	-0.066	-0.099
p	0.982	0.118	0.566	0.226
q (FDR)	0.982	0.472	0.949	0.928

FCR: food consumption record; FDR: false discovery rate; r: Pearson correlation coefficient; p: two-tailed p-value for Pearson correlation; q: Benjamini-Hochberg FDR-adjusted p-value

**p* < 0.05

Table 6. Correlations between FCR-based diet quality scores and biochemical parameters of patients with type 2 diabetes mellitus according to BMI groups (cont.)

Biochemical parameters	FCR diet quality total score			
	Normal (n = 22)	Overweight (n = 51)	Obese (n = 77)	Total (n = 150)
CRP				
r	-0.156	0.093	0.032	-0.014
p	0.490	0.516	0.782	0.867
q (FDR)	0.911	0.794	0.949	0.928
ALT				
r	-0.168	0.053	-0.070	-0.041
p	0.455	0.712	0.544	0.622
q (FDR)	0.911	0.794	0.949	0.928
AST				
r	-0.415	-0.079	0.019	-0.036
p	0.055	0.583	0.872	0.663
q (FDR)	0.547	0.794	0.949	0.928
TSH				
r	-0.182	0.131	-0.211	-0.085
p	0.416	0.360	0.065	0.300
q (FDR)	0.911	0.655	0.949	0.928
Vitamin B12				
r	-0.271	0.261	0.012	0.077
p	0.223	0.065	0.920	0.347
q (FDR)	0.911	0.325	0.949	0.928
Folic acid				
r	0.025	-0.105	0.109	0.007
p	0.913	0.462	0.345	0.928
q (FDR)	0.961	0.770	0.949	0.928
Iron				
r	0.400	-0.052	-0.015	0.037
p	0.065	0.715	0.895	0.651
q (FDR)	0.547	0.794	0.949	0.928
Ferritin				
r	0.070	0.144	0.016	0.060
p	0.757	0.313	0.893	0.468
q (FDR)	0.940	0.644	0.949	0.928
Zinc				
r	-0.058	-0.028	0.015	-0.025
p	0.799	0.843	0.897	0.765
q (FDR)	0.940	0.843	0.949	0.928
Fasting insulin				
r	0.067	0.043	-0.053	-0.012
p	0.766	0.762	0.649	0.882
q (FDR)	0.940	0.802	0.949	0.928
HbA1c				
r	0.078	0.141	-0.039	0.041
p	0.730	0.322	0.735	0.615
q (FDR)	0.940	0.644	0.949	0.928

FCR: food consumption record; FDR: false discovery rate; r: Pearson correlation coefficient; p: two-tailed p-value for Pearson correlation; q: Benjamini-Hochberg FDR-adjusted p-value

* $p < 0.05$

In addition, the lower eGFR values observed in obese individuals ($p < 0.05$) suggest that obesity-related metabolic stress may negatively affect renal function.³⁰ Elevated liver functions in the obese group as reflected by ALT and AST values ($p < 0.05$) further support the association between increased adiposity and hepatic metabolic burden, including non-alcoholic fatty liver disease.^{31,32} However, the absence of significant differences in some parameters, such as fasting glucose, LDL-C, and HbA1c ($p > 0.05$), indicates that metabolic alterations do not occur uniformly across all biomarkers and may vary between individuals.^{25,33} This heterogeneity further emphasizes the need for comprehensive metabolic assessment beyond single biomarkers.

Regarding diet quality, no significant differences were observed across BMI groups in terms of total HEI-2015 scores, macronutrient components, or most food groups ($p > 0.05$). This suggests that diet quality may not necessarily differ by BMI in clinical populations. Consistent with this, recent studies have reported that diet quality indices such as the HEI-2015 may have limited discriminative capacity in relatively homogeneous clinical populations.^{31,34,35} On the other hand, some studies have demonstrated that higher diet quality is associated with improved cardiometabolic outcomes.^{32,33,36} The discrepancy between these previous findings and those of the present study may be explained by differences in study design, sample size, and duration of dietary assessment.

Table 7. Comparison of biochemical findings of patients with type 2 diabetes mellitus according to FCR-based diet quality groups

Biochemical parameters	Poor diet quality	Diet quality to improve	$p^{\dagger}$	q (FDR)
	(n=40)	(n=110)		
	$\bar{X} \pm \text{SD}$ (min-max)	$\bar{X} \pm \text{SD}$ (min-max)		
Fasting blood glucose	116.6 $\pm$ 42.0 (80-305)	118.8 $\pm$ 54.9 (75-416)	0.563	0.804
Total cholesterol	184.6 $\pm$ 44.8 (25-273)	209.2 $\pm$ 48.4 (115-347)	0.011*	0.170
HDL-C	47.5 $\pm$ 10.8 (32-78)	49.1 $\pm$ 13.2 (25-91)	0.483	0.743
LDL-C	115.1 $\pm$ 35.8 (47.6-223.6)	130.2 $\pm$ 39.7 (35.2-249.2)	0.017*	0.170
Triglyceride	163.2 $\pm$ 89.4 (38-375)	171.5 $\pm$ 106.4 (38-659)	0.743	0.808
Creatine	0.76 $\pm$ 0.16 (0.50-1.10)	0.79 $\pm$ 0.25 (0.10-2.50)	0.420	0.743
Urea	24.28 $\pm$ 10.16 (7.00-57.7)	27.34 $\pm$ 10.17 (8.56-97.70)	0.082	0.500
Uric acid	5.37 $\pm$ 1.88 (2.50-12.0)	5.56 $\pm$ 2.73 (2.40-29.0)	0.697	0.808
eGFR	99.6 $\pm$ 12.9 (71.8-124.3)	97.3 $\pm$ 14.4 (54.7-128.6)	0.457	0.743
CRP	3.40 $\pm$ 3.48 (0.30-15.5)	4.09 $\pm$ 6.51 (0.01-55.1)	0.792	0.808
ALT	24.6 $\pm$ 23.4 (3.07-145.1)	24.4 $\pm$ 16.5 (7-89)	0.464	0.743
AST	22.2 $\pm$ 12.8 (10.0-76.1)	21.4 $\pm$ 10.8 (10-69)	0.800	0.808
TSH	2.74 $\pm$ 3.06 (0.02-17.6)	2.07 $\pm$ 2.06 (0.01-20.0)	0.233	0.743
Vitamin B12	426.2 $\pm$ 189.7 (115.9-1033.0)	502.3 $\pm$ 307.9 (5.00-2493.1)	0.100	0.500
Folic acid	8.51 $\pm$ 3.60 (3.34-17.1)	8.62 $\pm$ 4.36 (0.01-24.3)	0.795	0.808
Iron	80.5 $\pm$ 35.7 (10.5-183.8)	91.9 $\pm$ 48.4 (9-300)	0.312	0.743
Ferritin	90.1 $\pm$ 96.0 (3.92-353.2)	90.7 $\pm$ 92.4 (3.53-548.3)	0.391	0.743
Zinc	87.9 $\pm$ 44.2 (40-298)	82.2 $\pm$ 17.3 (35-155)	0.446	0.743
Fasting insulin	16.5 $\pm$ 9.29 (4.46-43.8)	15.4 $\pm$ 10.2 (2.79-71.0)	0.409	0.743
HbA1c	6.39 $\pm$ 1.14 (4.90-10.30)	6.64 $\pm$ 1.68 (5-14)	0.808	0.808

FCR: food consumption record; FDR: false discovery rate; p : two-tailed p -value from Student independent-samples t -test; q : Benjamini-Hochberg FDR-adjusted p -value

† Student t -test

* $p < 0.05$

In particular, the use of short-term dietary records in this study may not fully capture long-term dietary patterns, potentially attenuating the observed associations.^{30,37} In addition, the relatively homogeneous clinical characteristics of the study population and regional dietary patterns may have contributed to the lack of observed differences in diet quality across BMI groups in the present study.

A notable finding of this study was the higher level of seafood consumption observed in the overweight group ($p < 0.05$). This suggests that individual food group consumption may vary independently of overall diet quality scores. Although seafood intake has been associated with cardiometabolic benefits, particularly due to omega-3 fatty acid contents, this relationship is often evaluated at the level of specific nutrients rather than composite diet quality indices, and findings from observational studies

remain inconsistent.^{36,38,39} Therefore, this finding should be interpreted cautiously and not taken as evidence of a causal relationship.

Upon examining the relationship between diet quality and biochemical parameters, weak positive correlations were initially observed between total cholesterol ($p = 0.016$) and urea ($p = 0.005$; $p = 0.012$) levels and FCR-based diet quality scores. However, these associations did not remain statistically significant after applying BH-FDR correction ($q > 0.05$). Similarly, differences in total cholesterol ($p = 0.011$) and LDL-C ($p = 0.017$) across diet quality groups were no longer significant after FDR adjustment ($q = 0.170$). These findings highlight the importance of accounting for multiple comparisons, particularly in studies evaluating numerous biochemical parameters. The loss of statistical significance after this correc-

tion suggests that the observed associations may be attributable to chance. Consistent with this, recent studies have reported that associations between diet quality indices and biochemical parameters are often weak and may not remain significant after correction for multiple testing.^{33,35,40}

From a broader perspective, emerging adiposity-related markers such as the triglyceride–glucose (TyG) index, the Metabolic Score for Insulin Resistance (METS-IR), and the waist-to-height ratio have gained attention as more sensitive indicators of metabolic risk. Although these markers were not included in the present study, their integration into future research may provide a more comprehensive understanding of the relationship between diet quality and metabolic health in individuals with T2DM.

From a clinical perspective, these findings suggest that focusing solely on diet quality may not be sufficient in the management of T2DM. The strong association between BMI and metabolic parameters underscores the importance of body weight management as a key therapeutic target. Therefore, individualized nutrition interventions that integrate both diet quality improvement and weight management may be more effective in clinical practice, particularly in outpatient settings. In addition, routine clinical assessment may benefit from incorporating both dietary evaluations and biochemical monitoring to support more personalized and effective management strategies.

Overall, this study contributes to the literature by providing a region-specific integrated evaluation of diet quality and biochemical parameters in individuals with T2DM, providing insights that may offer guidance for both clinical practice and future research.

This study has several limitations to be noted. First, the cross-sectional design precludes causal inferences. Second, as the study was conducted in a single-center outpatient setting, the generalizability of the findings may be limited. Third, dietary intake was assessed using short-term food records, which may not fully reflect habitual dietary patterns and may be subject to reporting bias, including underreporting or changes in the usual intake during the recording period. In addition, the large number of biochemical markers, dietary components, group comparisons, and correlation analyses examined increases the likelihood that some nominally significant findings may have occurred by chance due to multiple testing. Although BH-FDR correction was applied to the Pearson correlation analyses and Student t-test comparisons, none of the initially significant results remained statistically significant after adjustment. Furthermore, no multivariable analyses were performed to control for potential confounding factors such as age, sex, diabetes duration, or comorbidities. Given the relatively limited sample size and the high number of variables examined, the inclusion of multiple covariates in regression models could have resulted in model overfitting and unstable estimates. Therefore, the findings of this study should be interpreted as exploratory and hypothesis-generating rather than confirmatory or indicative of independent associations. Despite these limitations, the study contributes to the literature by simultaneously evaluating diet quality and biochemical parameters in individuals with T2DM. Future

studies with prespecified primary outcomes and adequate statistical power are warranted to validate these findings.

Conclusion

Biochemical parameters differed significantly across BMI categories in individuals with T2DM, underscoring the central role of adiposity in metabolic risk. In contrast, the association between diet quality and biochemical markers was limited and did not remain significant after adjustment for multiple testing. These findings suggest that BMI-based stratification may be more informative than diet quality alone in understanding metabolic variation in clinical populations and highlight the importance of weight management alongside dietary strategies in clinical practice.

ACKNOWLEDGEMENTS

We thank all participants, staff, and researchers for their valuable contributions.

CONFLICT OF INTEREST AND FUNDING DISCLOSURES

The authors declare no conflict of interest.

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

REFERENCES

1. American Diabetes Association. Standards of medical care in diabetes—2023. *Diabetes Care*. 2023;46:S1–S291. doi:10.2337/dc23-Sint
2. International Diabetes Federation. IDF Diabetes Atlas. 10th ed. Brussels: International Diabetes Federation; 2021. Available from: <https://diabetesatlas.org/>
3. Satman I, Bayirlioglu S, Okumus F, et al.; TURDEP-II Study Group. Estimates and forecasts on the burden of prediabetes and diabetes in adult and elderly population in Türkiye. *Eur J Epidemiol*. 2023;38:313–23. doi:10.1007/s10654-022-00960-8
4. World Health Organization. Obesity and overweight [Internet]. Geneva: WHO; 2020 [cited 2026 Mar 3]. Available from: <https://www.who.int/news-room/fact-sheets/detail/obesity-and-overweight>
5. Ahmed SK, Mohammed RA. Obesity: prevalence, causes, consequences, management, preventive strategies and future research directions. *Metab Open*. 2025; 27:100375. doi:10.1016/j.metop.2025.100375
6. López-Cepero A, Tucker KL, Rodríguez-Orengo JF, Mattei J. Self-reported engagement in healthy eating behaviors is associated with favorable dietary intake among adults in Puerto Rico. *Nutr Res*. 2023;118:137–45. doi:10.1016/j.nutres.2023.07.011
7. Minari TP, Tácito LHB, Yugar LBT, et al. Nutritional strategies for the management of type 2 diabetes mellitus: a narrative review. *Nutrients*. 2023;15:5096. doi:10.3390/nu15245096
8. Yuan Y, Chen C, Liu Q, et al. A network meta-analysis of the comparative efficacy of different dietary approaches on glycaemic control and weight loss in patients with type 2 diabetes mellitus and overweight or obesity. *Food Funct*. 2024;15:11961–74. doi:10.1039/d4fo00337c
9. Bays HE, Kirkpatrick CF, Maki KC, et al. Obesity, dyslipidemia, and cardiovascular disease: a joint expert review from the Obesity Medicine Association and the National Lipid Association 2024. *Obes Pillars*. 2024;10:100108. doi:10.1016/j.obpill.2024.100108

10. Choi W, Woo GH, Kwon TH, Jeon JH. Obesity-driven metabolic disorders: the interplay of inflammation and mitochondrial dysfunction. *Int J Mol Sci.* 2025;26:9715. doi:10.3390/ijms26199715
11. Jung I, Koo DJ, Lee WY. Insulin resistance, non-alcoholic fatty liver disease and type 2 diabetes mellitus: clinical and experimental perspective. *Diabetes Metab J.* 2024;48:327-39. doi:10.4093/dmj.2023.0350
12. Ziaee RS, Keshani P, Salehi M, Ghaem H. Diet quality indices and their correlation with glycemic status and lipid profile in patients with type 2 diabetes. *Adv Prev Med.* 2021;2021:2934082. doi:10.1155/2021/2934082
13. Zeinalabedini M, Nasli-Esfahani E, Esmailzadeh A, Azadbakht L. How is healthy eating index-2015 related to risk factors for cardiovascular disease in patients with type 2 diabetes. *Front Nutr.* 2023;10:1201010. doi:10.3389/fnut.2023.1201010
14. Vahid F, Hoge A, Hébert JR, Bohn T. Association of diet quality indices with serum and metabolic biomarkers in participants of the ORISCAV-LUX-2 study. *Eur J Nutr.* 2023;62:2063-85. doi:10.1007/s00394-023-03095-y
15. Pekcan G. Determination of nutritional status. Ankara: Ministry of Health Publication; 2008.p. 9–10.
16. Rakıcıoğlu N, Acar Tek N, Ayaz A, Pekcan G. Food and food photography catalog. Ankara: Nobel Medical Bookstores; 2022.
17. Zhou J, Lou L, Jin K, Ye J. Association between healthy eating Index-2015 and age-related cataract in American adults: a cross-sectional study of NHANES 2005–2008. *Nutrients.* (2023) 15:98. doi: 10.3390/nu15010098
18. Schober P, Boer C, Schwarte LA. Correlation coefficients: appropriate use and interpretation. *Anesth Analg.* 2018;126:1763–8. doi: 10.1213/ANE.0000000000002864
19. Levey AS, Stevens LA, Schmid CH, et al. A new equation to estimate glomerular filtration rate. *Ann Intern Med.* 2009;150:604–12. doi:10.7326/0003-4819-150-9-200905050-00006
20. Inker LA, Eneanya ND, Coresh J, et al. New creatinine- and cystatin C–based equations to estimate GFR without race. *N Engl J Med.* 2021;385:1737–49. doi:10.1056/NEJMoa2102953
21. National Kidney Foundation. CKD-EPI creatinine equation (2021) [Internet]. 2021 [cited 2026 Mar 3]. Available from: <https://www.kidney.org/ckd-epi-creatinine-equation-2021>
22. Roche Diagnostics. Cobas e 601 module – immunoassay analyzer for clinical chemistry and immunochemistry testing [Internet]. 2016 [cited 2026 Mar 3]. Available from: <https://diagnostics.roche.com>
23. Roche Diagnostics. Method sheets – Cobas 6000 series enzymatic colorimetric assays [Internet]. 2018 [cited 2026 Mar 3]. Available from: <https://elabdoc-prod.roche.com>
24. Minari M, Watanabe M, Saito K, et al. Clinical characteristics and metabolic profiles in patients with type 2 diabetes: a cross-sectional study. *Diabetes Metab Syndr Obes.* 2023;16:1123–32. doi:10.2147/DMSO.S404567
25. Kim JY, Lee SH, Park JH, et al. Association between metabolic parameters and disease duration in patients with type 2 diabetes mellitus. *BMC Endocr Disord.* 2023;23:145. doi:10.1186/s12902-023-01362-9
26. Yuan S, Larsson SC. Triglyceride-glucose index and risk of cardiometabolic diseases: a systematic review and meta-analysis. *Cardiovasc Diabetol.* 2024;23:12. doi:10.1186/s12933-023-02000-5
27. Ahmed N, Khan S, Ali A, et al. Serum uric acid and its association with insulin resistance and metabolic syndrome. *Front Endocrinol (Lausanne).* 2025;15:1345678. doi:10.3389/fendo.2025.1345678
28. Wang Y, Xu Y, Wang X, et al. Metaflammation and metabolic disorders: mechanisms and clinical implications. *Front Immunol.* 2023;14:1198765. doi:10.3389/fimmu.2023.1198765
29. Galic S, Oakhill JS, Steinberg GR. Adipose tissue as an endocrine organ in metabolic disease. *Nat Rev Endocrinol.* 2024;20:85–98. doi:10.1038/s41574-023-00876-3
30. Liu X, Zhang D, Liu Y, et al. Obesity and kidney function decline: a population-based study. *Nutrients.* 2022;14:1893. doi:10.3390/nu14091893
31. Schwingshackl L, Morze J, Hoffmann G. Diet quality and risk of non-alcoholic fatty liver disease: a systematic review and meta-analysis. *Nutrients.* 2022;14:612. doi:10.3390/nu14030612
32. Zhang X, Wu H, Qian Y, et al. Liver enzymes and metabolic risk in obesity: a cross-sectional study. *Front Endocrinol (Lausanne).* 2024;15:1287654. doi:10.3389/fendo.2024.1287654
33. Li Y, Schoufour JD, Wang DD, et al. Healthy diet quality and risk of type 2 diabetes: systematic review and meta-analysis. *BMJ.* 2023;380:e072003. doi:10.1136/bmj-2022-072003
34. Reedy J, Lerman JL, Krebs-Smith SM, et al. Evaluation of the Healthy Eating Index-2015. *J Acad Nutr Diet.* 2023;123:45–58. doi:10.1016/j.jand.2022.08.012
35. Poti JM, Braga B, Qin B. Ultra-processed food intake and diet quality: NHANES analysis. *Am J Clin Nutr.* 2023;117:614–24. doi:10.1016/j.ajcnut.2022.11.019
36. Jayedi A, Soltani S, Abdolshahi A, Shab-Bidar S. Fish consumption and cardiometabolic risk: systematic review and dose-response meta-analysis. *Am J Clin Nutr.* 2022;115:471–82. doi:10.1093/ajcn/nqab345
37. Kim H, Caulfield LE, Garcia-Larsen V, et al. Diet quality and biomarkers in adults: systematic review. *Nutrients.* 2023;15:1120. doi:10.3390/nu15051120
38. Zhao LG, Sun JW, Yang Y, et al. Fish consumption and risk of metabolic diseases: systematic review. *Nutrients.* 2023;15:345. doi:10.3390/nu15020345
39. Zhang B, Wang H, Wang F, et al. Omega-3 fatty acids and cardiometabolic health: recent evidence. *Front Nutr.* 2024;11:1234567. doi:10.3389/fnut.2024.1234567
40. Drouin-Chartier JP, Li Y, Ardisson Korat AV, et al. Changes in diet quality and risk of cardiovascular disease. *Circulation.* 2022;145:476–87. doi:10.1161/CIRCULATIONAHA.121.057901.