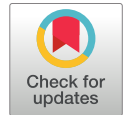


Research Article

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# ASSOCIATION OF DIETARY INFLAMMATORY INDEX WITH SYSTEMIC INFLAMMATION IN WELL-CONTROLLED TYPE 2 DIABETES MELLITUS



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## Abstract

**Objective:** Low-grade chronic inflammation contributes to residual cardiometabolic risk in type 2 diabetes (T2DM), even among patients who achieve glycemic targets. Dietary Inflammatory Index (DII) quantifies the inflammatory potential of habitual diet; however, its association with systemic inflammatory markers in well-controlled T2DM remains unclear. This study aimed to investigate the association between DII scores and circulating inflammatory markers in adults with well-controlled T2DM.

**Material and Methods:** This cross-sectional study included 120 adults with T2DM and 30 healthy controls. Dietary intake was assessed using a validated 7-day food frequency questionnaire, and DII scores were calculated. Patients with T2DM were stratified into DII quartiles. Serum levels of interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), and high-sensitive C-reactive protein (hs-CRP), along with metabolic parameters, were measured. Associations were evaluated using correlation and regression analyses, and effect sizes were estimated using Cohen's d.

**Results:** DII scores were positively correlated with IL-6 ( $r=0.421$ ,  $p<0.001$ ) and hs-CRP ( $r=0.312$ ,  $p=0.002$ ). IL 6 levels increased progressively across DII quartiles ( $p$  for trend  $<0.001$ ), with a large effect size between the lowest and highest quartiles (Cohen's  $d=0.84$ ). Higher DII scores were associated with lower intakes of dietary fiber, polyunsaturated fatty acids, magnesium, and vitamin C (all  $p<0.05$ ). Serum uric acid levels also increased across DII quartiles ( $p=0.044$ ). No significant association was observed between DII and TNF-α.

**Conclusions:** In adults with well-controlled T2DM, a more proinflammatory diet is associated with higher systemic inflammation, particularly elevated IL 6 levels. These findings suggest that dietary inflammatory potential may contribute to residual cardiometabolic risk beyond glycemic control.

## Keywords

Dietary Inflammatory Index • Type 2 diabetes mellitus • Interleukin-6 • High-sensitive C-reactive protein • Dietary inflammation • Low-grade inflammation



## INTRODUCTION

Type 2 diabetes (T2DM), is a complex metabolic disorder characterized by insulin resistance, progressive pancreatic  $\beta$ -cell dysfunction, and chronic low-grade inflammation (1, 2). Inflammation is increasingly recognized not only due to metabolic dysregulation but also as an active contributor to impaired insulin signaling and glycemic control (3). Pro-inflammatory cytokines, such as tumor necrosis factor- $\alpha$  (TNF- $\alpha$ ) and interleukin-6 (IL-6) may exacerbate oxidative stress and accelerate  $\beta$ -cell dysfunction, thereby sustaining a state of “metabolic inflammation” (4-6).

Alongside pharmacologic therapy, lifestyle factors—particularly dietary habits—are key modulators of systemic inflammation in T2DM (7). Diets rich in saturated fats, refined carbohydrates, and added sugars tend to promote inflammatory pathways, whereas nutrient-dense dietary patterns emphasizing fiber, antioxidants, and omega-3 fatty acids are associated with anti-inflammatory effects (8, 9). The Dietary Inflammatory Index (DII) was developed to quantify the inflammatory potential of habitual diet based on evidence from the literature (10, 11). Higher DII scores have been linked to increased risks of several chronic outcomes, including cardiovascular disease, cancer, and mortality (12–14, 18).

However, the relationship between DII and circulating inflammatory biomarkers in patients with well-controlled T2DM remains insufficiently characterized. This is clinically relevant because low-grade inflammation may persist despite achieving glycemic targets, potentially contributing to residual cardiometabolic risk. Therefore, we aimed to examine the association between DII scores and inflammatory markers (TNF- $\alpha$  and IL-6) in adults with well-controlled T2DM and, to explore whether dietary inflammatory load is related to anthropometric measures, biochemical profiles, and nutrient intake patterns. We hypothesized that a more proinflammatory diet would be associated with higher levels of systemic inflammatory markers even under adequate glycemic control.

## MATERIAL AND METHODS

### Study design and participants

This cross-sectional study was conducted between november 2019 and april 2020 in a tertiary care endocrinology setting. A total of 120 adults (aged 30-74 years) diagnosed with T2DM who had achieved glycemic targets (Hemoglobin A1c; (HbA1c)  $\leq 7\%$ ) under oral or insulin therapy were enrolled. An additional control group of 30 age- and sex-matched healthy individuals was included for comparison. Exclusion criteria included type 1 diabetes, HbA1c  $> 7\%$ , active infection, renal dysfunction, or a history of malignant tumor. This study was approved by İstanbul Faculty of Medicine Clinical Research Ethics Committee (Date: 24.05.2019, No: 10).

### Dietary assessment and DII calculation

Dietary intake was assessed using a 7-day recall-based food frequency questionnaire. Portion sizes were standardized using the Food and Nutrition Photo Catalogue and national reference recipes. Nutrient intakes were analyzed using the Nutrition Information System (BEBIS). Dietary Inflammatory Index scores were computed from 31 food parameters using the method described by Shivappa et al., and linked to the global mean database (11). Participants were classified into DII quartiles (Q1–Q4), reflecting increasing dietary inflammatory potential.

### Anthropometry and physical activity

Height, weight, body mass index (BMI), waist circumference, and body composition (via bioelectrical impedance analysis, Tanita BC-420MA) were assessed. Abdominal obesity was defined as WC  $\geq 88$  cm for women and  $\geq 102$  cm for men. Physical activity was evaluated using the short-form International Physical Activity Questionnaire (s-IPAQ) and categorized as inactive, moderately active, or highly active.

### Biochemical and inflammatory markers

Fasting venous blood samples were collected after  $\geq 8$  hours of fasting. Standard biochemical parameters (glucose, lipid parameters, and uric acid (UA)) were analyzed in the hospital laboratory. Inflammatory markers (IL-6, TNF- $\alpha$ ) (Elabscience® Biotechnology Co., Ltd, China) and stored at  $-80$  °C until analysis. High-sensitive C-reactive protein (hs-CRP) levels were measured using immunoturbidimetric methods in the central laboratory.

### Statistical analysis

Normality was assessed using the Shapiro–Wilk test. Group comparisons were made using ANOVA or Welch’s test as appropriate. Post hoc tests (Tukey or Games–Howell) identified between-group differences. Correlation (Pearson’s  $r$ ), linear regression, and effect size (Cohen’s  $d$ ) were used to assess associations between DII and inflammatory markers. Analyses were performed in SPSS v23.0 with a significance threshold of  $p < 0.05$ .

## RESULTS

### Characteristics of the participants

A total of 120 people with T2DM and 30 healthy controls were included. The baseline characteristics of the T2DM participants stratified by DII quartiles are shown in Table 1. There were no significant differences in age, and sex in all quartiles ( $p > 0.05$  for all). Waist circumference increased significantly from Q1 to Q4 ( $p < 0.01$ ), indicating greater central adiposity among participants who ate more proinflammatory diets.



**Table 1.** Characteristics of the study population

| Variable                 | T2DM Group (n=120) | Control Group (n=30) | p-value |
|--------------------------|--------------------|----------------------|---------|
| Age (years)              | 53.1±8.4           | 52.7±7.9             | 0.740   |
| Gender (Male/Female)     | 48 / 72            | 12 / 18              | 0.990   |
| BMI (kg/m²)              | 28.3±3.1           | 25.6±2.8             | <0.001  |
| Waist Circumference (cm) | 91.5±6.2           | 87.4±5.9             | <0.01   |
| Fasting Glucose (mg/dL)  | 142.2±21.4         | 91.6±10.2            | <0.001  |
| HbA1c (%)                | 6.8±0.4            | 5.3±0.3              | <0.001  |
| IL-6 (pg/mL)             | 3.5±1.3            | 1.8±0.7              | <0.001  |
| TNF-α (pg/mL)            | 5.4±1.2            | 3.1±0.9              | <0.001  |
| hs-CRP (mg/L)            | 3.2±1.6            | 2.1±1.1              | <0.010  |

T2DM: Type 2 diabetes mellitus, BMI: Body Mass Index, HbA1c: Glycated hemoglobin, IL-6: Interleukin-6, TNF-α: Tumor necrosis factor-alpha, hs-CRP: High-sensitivity C-reactive protein

Inflammatory biomarkers

The distribution of IL-6, TNF-α, and hs-CRP in the DII quartiles is presented in Table 2. IL-6 levels increased significantly in all quartiles (p<0.001), as did hs-CRP (p=0.011), while TNF-α showed a non-significant upward trend (p=0.072). These findings are visualized in Figure 1, which indicates progressively increasing concentrations of IL 6 and hs-CRP from Q1 to Q4.

**Table 2.** Participant and inflammatory characteristics in DII quartiles (Group T2DM)

| Variable                 | Q1 (n=30) | Q2 (n=30) | Q3 (n=30) | Q4 (n=30) | p-value |
|--------------------------|-----------|-----------|-----------|-----------|---------|
| Age (years)              | 52.1±8.3  | 53.3±7.9  | 53.5±8.1  | 53.8±7.9  | 0.840   |
| Gender (Male/Female)     | 12 / 18   | 13 / 17   | 11 / 19   | 12 / 18   | 0.930   |
| BMI (kg/m²)              | 26.4±2.5  | 27.8±2.8  | 28.6±2.9  | 30.1±2.9  | <0.01   |
| Waist circumference (cm) | 88.2±5.4  | 90.1±6.0  | 92.0±6.6  | 94.7±6.3  | <0.01   |
| HbA1c (%)                | 6.7±0.3   | 6.8±0.4   | 6.9±0.4   | 6.9±0.5   | 0.13    |
| Duration of T2DM (years) | 6.1±2.9   | 6.4±3.2   | 6.5±3.4   | 6.8±3.6   | 0.62    |
| IL-6 (pg/mL)             | 2.4±1.2   | 2.9±1.3   | 3.5±1.4   | 4.1±1.5   | <0.001  |
| TNF-α (pg/mL)            | 4.6±1.0   | 5.0±1.1   | 5.5±1.2   | 6.2±1.3   | 0.072   |
| hs-CRP (mg/L)            | 2.9±1.6   | 3.1±1.5   | 3.3±1.7   | 3.5±1.7   | 0.011   |

Q1, 2, 3, 4: quartile 1,2,3,4, T2DM: Type 2 diabetes mellitus, BMI: Body Mass Index, HbA1c: Glycated Hemoglobin, IL-6: Interleukin-6, TNF-α: Tumor Necrosis Factor-alpha, hs-CRP: High-sensitivity C-reactive Protein

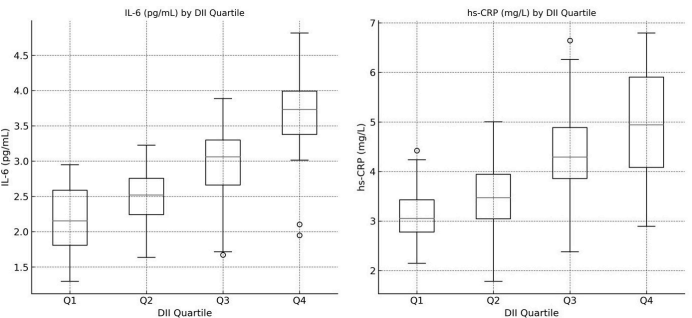


**Figure 1.** Inflammatory marker profiles across DII Quartiles. The box plots show IL-6 and hs-CRP concentrations across the quartiles of the Dietary Inflammatory Index (DII) in individuals with T2DM. Both markers increased progressively from the most anti-inflammatory (Q1) to the most pro-inflammatory (Q4) quartiles.

A moderate positive correlation between DII and IL-6 was found (r=0.421, p<0.001) and a weaker correlation with hs-CRP (r=0.312, p=0.002), as shown in Table 3. These associations are further illustrated in Figure 2, which shows a linear regression model between DII and IL-6.

**Table 3.** Correlation between the dietary inflammatory index and inflammatory markers

| Variable      | r     | p-value |
|---------------|-------|---------|
| IL-6 (pg/mL)  | 0.421 | <0.001  |
| TNF-α (pg/mL) | 0.198 | 0.053   |
| hs-CRP (mg/L) | 0.312 | 0.002   |

IL-6: Interleukin-6, TNF-α: Tumor necrosis factor-alpha, hs-CRP: High-sensitivity C-reactive Protein

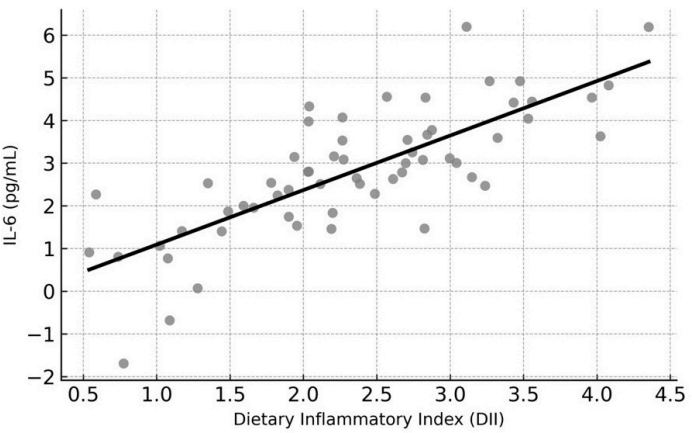


**Figure 2.** Correlation between the Dietary Inflammatory Index (DII) scores and IL-6 levels among patients with type 2 diabetes. A significant positive correlation was observed between DII and IL-6 (r=0.421, p < 0.001), indicating that more pro-inflammatory diets were associated with higher systemic inflammatory activity.

The magnitude of the difference in IL-6 levels between the most anti-inflammatory (Q1) and most pro-inflammatory (Q4) quartiles yielded a Cohen's d effect size of 0.84, indicating a large effect (Table 4).

**Table 4.** Effect size and correlation summary for DII and IL-6

| Comparison              | Effect Size / Correlation | p-value |
|-------------------------|---------------------------|---------|
| Q1 vs Q4 (Cohen's d)    | 0.840                     | <0.001  |
| DII vs IL-6 (Pearson r) | 0.421                     | <0.001  |

Q1 and Q4: Quartile 1 and 4, respectively, DII: Dietary Inflammatory Index, IL-6: Interleukin-6

Nutrient intake patterns

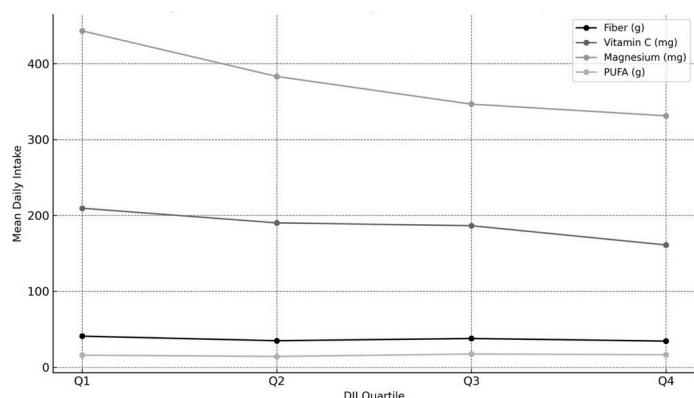
Nutrient intake across DII quartiles is presented in Table 5. Individuals in Q1 reported significantly higher consumption of fiber, vitamin C, magnesium, polyunsaturated fatty acids (PUFA), and other antiinflammatory nutrients. These trends are depicted in Figure 3, which shows a consistent decline in antiinflammatory nutrient intake from Q1 to Q4.



**Table 5.** Comprehensive nutrient intake in DII quartiles (T2DM and control groups)

| Nutrient                   | Q1 (n=30)      | Q2 (n=30)      | Q3 (n=30)      | Q4 (n=30)      | Control (n=30) | p-value |
|----------------------------|----------------|----------------|----------------|----------------|----------------|---------|
| <b>MUFA (g)</b>            | 26.96±5.93     | 26.30±4.61     | 30.63±6.03     | 32.83±7.78     | 25.38±5.45     | <0.001  |
| <b>PUFA (g)</b>            | 15.92±5.05     | 14.25±4.45     | 17.39±4.34     | 16.47±6.61     | 13.02±4.79     | 0.009   |
| <b>n-3 fatty acid (g)</b>  | 1.94±0.77      | 1.84±0.79      | 1.82±0.45      | 1.86±0.83      | 1.57±0.50      | 0.311   |
| <b>n-6 fatty acid (g)</b>  | 13.23±4.49     | 11.78±4.00     | 14.79±4.26     | 13.79±5.79     | 10.89±4.41     | 0.011   |
| <b>Cholesterol (mg)</b>    | 335.78±93.37   | 336.62±125.10  | 309.71±111.21  | 365.79±131.99  | 333.92±109.69  | 0.468   |
| <b>Fiber (g)</b>           | 40.95±7.82     | 34.97±6.02     | 37.90±7.10     | 34.47±6.01     | 38.48±7.22     | 0.002   |
| <b>Vitamin A (mcg)</b>     | 1069.26±554.37 | 1130.52±723.86 | 1047.21±470.78 | 1002.96±257.21 | 870.83±179.85  | 0.009   |
| <b>Vitamin E (mg)</b>      | 16.96±5.03     | 15.96±5.01     | 19.04±6.53     | 17.41±5.78     | 14.47±4.90     | 0.025   |
| <b>Vitamin B1 (mg)</b>     | 1.37±0.21      | 1.21±0.26      | 1.28±0.22      | 1.25±0.18      | 1.28±0.23      | 0.091   |
| <b>Vitamin B2 (mg)</b>     | 1.78±0.27      | 1.74±0.38      | 1.58±0.29      | 1.47±0.29      | 1.73±0.32      | 0.001   |
| <b>Vitamin B3 (mg)</b>     | 21.25±4.10     | 18.64±3.16     | 21.37±4.02     | 19.95±3.09     | 20.32±4.58     | 0.045   |
| <b>Vitamin B6 (mg)</b>     | 1.80±0.50      | 1.64±0.67      | 1.61±0.65      | 1.43±0.52      | 1.77±0.41      | 0.095   |
| <b>Folate (mcg)</b>        | 250.27±37.70   | 233.93±25.07   | 227.04±24.33   | 222.92±18.02   | 251.52±33.41   | 0.010   |
| <b>Vitamin B12 (mcg)</b>   | 5.34±2.61      | 5.08±2.01      | 5.09±1.92      | 5.51±3.26      | 4.97±1.68      | 0.902   |
| <b>Vitamin C (mg)</b>      | 209.44±51.40   | 190.19±54.63   | 186.46±43.65   | 161.06±54.55   | 179.82±76.54   | 0.015   |
| <b>Magnesium (mg)</b>      | 443.04±80.42   | 383.04±75.77   | 346.55±41.31   | 331.21±36.40   | 414.12±66.37   | <0.001  |
| <b>Zinc (mg)</b>           | 12.80±2.32     | 11.63±2.50     | 13.12±2.74     | 11.86±1.91     | 12.43±2.40     | 0.007   |
| <b>Selenium (mcg)</b>      | 20.75±12.63    | 18.09±9.23     | 16.68±6.52     | 18.34±6.52     | 19.64±17.64    | 0.015   |
| <b>Iron (mg)</b>           | 19.05±3.81     | 16.81±2.30     | 18.97±3.55     | 17.61±2.37     | 18.46±2.73     | 0.007   |
| <b>Total energy (kcal)</b> | 1820±210       | 1885±230       | 1955±245       | 2050±240       | -              | <0.05   |

Q1, 2, 3, 4: Quartile 1, 2, 3, 4, MUFA: Monounsaturated fatty acids, PUFA: Polyunsaturated fatty acids

**Figure 3.** Trends in Anti-Inflammatory Nutrient Intake in DII Quartiles. Q1, 2, 3, 4: Quartile 1, 2, 3, 4, DII: Dietary Inflammatory Index, PUFA: Polyunsaturated fatty acids

## Biochemical parameters

Routine biochemical markers are summarized in Table 6. Uric acid levels increased significantly in the DII quartiles ( $p=0.044$ ), whereas changes in total cholesterol, triglycerides, and liver enzymes did not reach statistical significance.

**Table 6.** Biochemical markers across DII Quartiles (T2DM Group)

| Parameter                        | Q1 (n=30)  | Q2 (n=30)  | Q3 (n=30)  | Q4 (n=30)  | p-value |
|----------------------------------|------------|------------|------------|------------|---------|
| <b>Total cholesterol (mg/dL)</b> | 192.3±34.2 | 195.6±36.1 | 198.8±33.9 | 202.1±32.7 | 0.184   |
| <b>HDL (mg/dL)</b>               | 48.6±10.4  | 46.8±10.2  | 45.3±9.9   | 44.1±10.1  | 0.093   |
| <b>LDL (mg/dL)</b>               | 115.2±28.6 | 117.5±29.4 | 119.8±27.5 | 123.4±26.8 | 0.171   |
| <b>Triglycerides (mg/dL)</b>     | 151.7±45.9 | 158.3±42.7 | 165.2±47.3 | 172.6±49.5 | 0.065   |
| <b>AST (U/L)</b>                 | 23.6±7.1   | 24.4±6.8   | 25.1±7.4   | 26.3±6.9   | 0.079   |
| <b>Urea (mg/dL)</b>              | 34.9±8.7   | 36.2±8.1   | 37.1±9.0   | 38.5±9.4   | 0.092   |
| <b>Creatinine (mg/dL)</b>        | 0.88±0.16  | 0.90±0.18  | 0.93±0.20  | 0.95±0.21  | 0.118   |
| <b>Uric acid (mg/dL)</b>         | 5.4±1.2    | 5.6±1.3    | 5.8±1.4    | 6.0±1.5    | 0.044   |

Q1, 2, 3, 4: Quartile 1, 2, 3, 4, HDL: High-density lipoprotein cholesterol, LDL: Low-density lipoprotein cholesterol, AST: Aspartate aminotransferase.

## DISCUSSION

This study demonstrates that dietary inflammatory potential, as assessed by the DII, is significantly associated with elevated systemic inflammation in patients with T2DM despite achieving glycemic targets. Individuals consuming more pro-inflammatory diets exhibited higher serum interleukin-6 (IL-6) and high-sensitivity C-reactive protein (hs-CRP) levels, indicating that glycemic control alone may be insufficient to fully attenuate metabolic inflammation.



Consistent with previous reports, DII scores in people with diabetes typically span a wide range, reflecting substantial interindividual variability in dietary inflammatory burden (10, 15). In our cohort, IL-6 increased linearly across DII quartiles, with a moderate-to-strong correlation ( $r=0.421$ ) and a large effect size between the lowest and highest quartiles (Cohen's  $d=0.84$ ), suggesting a clinically meaningful shift in inflammatory status. These findings align with observations by Denova-Gutiérrez et al. in Mexican adults with T2DM, who similarly reported higher inflammatory marker levels with increasing DII scores (20).

The biological plausibility of these associations is well established. Diets rich in saturated fat and poor in fiber and micronutrients promote oxidative stress, gut dysbiosis, and metabolic endotoxemia, activating inflammatory pathways mediated by nuclear factor-kappa B (NF- $\kappa$ B) and toll-like receptors (3, 4, 8). In line with this, participants with higher DII scores in our study reported lower intakes of fiber, PUFA, vitamin C, and magnesium—nutrients known to modulate inflammatory responses and improve insulin sensitivity (9, 15, 16).

Although TNF- $\alpha$  levels showed an upward trend across DII quartiles, the association did not reach statistical significance. Similar patterns have been observed in metabolically stable T2DM populations, where IL 6 and hs-CRP appear to be more sensitive indicators of diet-related inflammation (5, 17). Serum UA was also positively associated with DII, supporting broader evidence linking proinflammatory dietary patterns to metabolic and oxidative stress pathways (19). The lack of statistical significance for TNF- $\alpha$  may reflect limited sample size or low variability in glycemic control, which could reduce the power to detect subtler cytokine changes.

An intriguing observation was that healthy controls exhibited lower inflammatory marker levels despite higher mean DII scores than the lowest DII quartile of patients with T2DM. This apparent paradox likely reflects disease-specific factors, such as underlying insulin resistance, metabolic memory, and chronic immune activation, which may sustain inflammation independently of dietary exposure in T2DM (1, 2). These findings underscore the multifactorial nature of

metabolic inflammation and suggest that dietary inflammatory potential alone does not fully explain systemic inflammatory burden in diabetes.

Taken together, our results support the clinical relevance of the DII as a complementary tool in diabetes nutrition practice. While current management strategies primarily emphasize glycemic targets and macronutrient distribution, incorporating dietary inflammatory potential into nutritional assessment may improve risk stratification and facilitate more personalized dietary counseling in T2DM.

This study has several limitations. Its cross-sectional design precludes causal inference, dietary intake assessment relied on 7-day recall, and inflammatory biomarkers were measured at a single time point. In addition, the inclusion of patients with well-controlled T2DM may limit generalizability to populations with poorer glycemic control or greater comorbidity burden. Furthermore, although BMI and waist circumference increased across DII quartiles, additional multivariable adjustment analyses could not be performed. Therefore, residual confounding related to adiposity and other metabolic variables cannot be excluded.

## CONCLUSION

In adults with well-controlled type 2 diabetes, a more proinflammatory dietary profile is associated with higher systemic inflammatory activity, particularly elevated IL 6 and hs-CRP levels, despite adequate glycemic control. These findings suggest that dietary inflammatory load may contribute to persistent low-grade inflammation and residual cardiometabolic risk beyond glucose regulation.

Incorporating the inflammatory potential of diet into nutritional assessment and counseling may enhance risk stratification and support comprehensive metabolic management in type 2 diabetes. Prospective and intervention studies are warranted to determine whether reducing dietary inflammatory burden can improve inflammatory status and long-term cardiometabolic outcomes.



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**Ethical Committee Approval** This study was approved by Istanbul Faculty of Medicine Clinical Research Ethics Committee (Date: 24.05.2019 No: 10).

**Informed Consent** Written informed consent was obtained from all participants before study enrollment.

**Peer Review** Externally peer-reviewed.

**Author Contributions** Conception/Design of Study- F.Y.T., K.K., A.K.U.; Data Acquisition- F.Y.T., K.K., A.K.U., F.C.; Data Analysis/ Interpretation- F.Y.T., F.C.; Drafting Manuscript- F.Y.T., A.K.U.; Critical Revision of Manuscript- F.Y.T., F.C., A.K.U., H.H., O.S.S., K.K.; Final Approval and Accountability- F.Y.T., B.F.C., H.H., O.S.S., A.K.U., K.K..

**Conflict of Interest** The authors have no conflict of interest to declare.  
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