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Increased Dietary Inflammatory Index Score Is Associated with Type 2 Diabetes Mellitus in Obese Women: A Case–Control Study

Kadriye Toprak[1], Süleyman Görpelioğlu[2], Şeyda Özdemir[3], Ahmet Özsoy[3], Aylin Ayaz[4]

[1]Department of Nutrition and Dietetics, Ankara Medipol University, Faculty of Health Sciences, Ankara, Turkey
[2]Department of Family Medicine, University of Health Sciences, Dışkapı Yıldırım Beyazıt Training and Research Hospital, Ankara, Turkey
[3]Department of Biochemistry, Ankara Provincial Health Directorate, Etlik City Hospital, Ankara, Turkey
[4]Department of Nutrition and Dietetics, Hacettepe University, Faculty of Health Sciences, Ankara, Turkey

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ABSTRACT

Objective: Recent evidence indicates that diet-induced inflammation is related to chronical diseases including type 2 diabetes mellitus. This study aimed to examine the relationship between the dietary inflammatory index, which quantifies the inflammatory burden of the diet, and type 2 diabetes mellitus risk among obese women.
Methods: This case–control study, including 40 obese cases with type 2 diabetes mellitus and 40 obese controls without type 2 diabetes mellitus, aged between 30 and 50, was conducted from September 2019 to March 2020 in Ankara, Turkey. The Dietary Inflammatory Index was calculated based on the food frequency questionnaire. The logistic regression model was used to estimate the association between Dietary Inflammatory Index and the risk of type 2 diabetes mellitus. Linear regression model was used to estimate beta coefficients for glucose metabolism markers.
Results: Subjects with higher Dietary Inflammatory Index scores (i.e., with a more pro-inflammatory diet) had a higher risk of type 2 diabetes mellitus (odds ratio = 8.57; 95% CI: 1.24, 59.44, $P = .03$). In addition, as a continuous variable, the Dietary Inflammatory Index scores had a significant positive relationship with insulin ($\beta = 0.259$, $P = .026$) and homeostatic model assessment of insulin resistance ($\beta = 0.265$, $P = .015$) after multivariable adjustment.
Conclusion: The present study suggests that higher Dietary Inflammatory Index scores, corresponding to more pro-inflammatory diets, were positively associated with type 2 diabetes mellitus risk among obese women. In addition, the Dietary Inflammatory Index scores and insulin resistance were positively related. As a result of the findings, an anti-inflammatory diet can help prevent insulin resistance and reduce the risk of diabetes.

Keywords: Dietary Inflammatory Index (DII), diabetes, inflammation, obesity

INTRODUCTION

Type 2 diabetes mellitus (T2DM) is a major cause of morbidity and mortality worldwide. An aging population, economic development, urbanization, an increasingly sedentary lifestyle, and unhealthy dietary habits have led to an increase in the T2DM prevalence around the world.[1] According to the International Diabetes Federation (IDF) report, there were 463 million people with diabetes around the world in 2019 and that this number will increase to 700 million by 2045.[2] There has been growing evidence that in addition to the genetic, metabolic, and lifestyle factors that cause T2DM, inflammation also plays an essential role in the pathogenesis of T2DM.[3] Inflammation is characterized by increased levels of pro-inflammatory cytokines such as C-reactive protein (CRP), tumor necrosis factor-alpha (TNF-α), and interleukins (ILs).[4] Especially in chronic low-grade inflammation, pro-inflammatory cytokines that increase in serum levels cause β-cell damage and chronic hyperglycemia, and T2DM occurs as a result.[5] Inflammation can develop due to many environmental and behavioral factors. However, the diet has been accepted as an essential modulator of chronic inflammation in recent years.[6] Indeed, many studies have shown that many nutritional factors and dietary patterns affect the serum levels of inflammatory markers such as IL-6, TNF-α, and CRP.[6,7]

The results of diet on inflammation and of inflammation on diabetes propose that diet may also improve the T2DM

Corresponding author: Kadriye Toprak, e-mail: kadriye.eken.toprak@gmail.com

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risk through inflammation.[8] In this context, previous studies have associated many nutrients that are considered to have an anti-inflammatory effect, such as dietary fiber and carotenoids, with low T2DM risk.[9,10] In contrast, the foods considered to have a pro-inflammatory effect, such as red meat containing high levels of saturated fatty acids, have been associated with high T2DM risk.[11]

Dietary Inflammatory Index (DII) is a literature-derived index developed to evaluate the inflammatory potential of diet. According to the index scoring, the higher DII score indicated a pro-inflammatory diet, whereas the lower score indicated an anti-inflammatory diet. The final score is obtained from not only a certain nutrient or food but from the overall diet.[12] The DII has been validated by inflammatory markers such as CRP, IL-6, or TNF-α in various studies.[13,14] It could be used in any human population with dietary data collected from different assessment methods such as food frequency questionnaire (FFQ), 24-hour dietary recall, and 3- to 7-day food record.[13-15] After the development of the DII, its relationship with various diseases such as cardiovascular diseases,[16] renal diseases,[17] mental health,[18] metabolic syndrome,[19] and, in particular, cancer[20] has been investigated. However, besides only a few studies existing on the relationship between the DII and glycemic markers in the literature,[21,22] to the best of our knowledge, no case–control study has focused on the relationship between the DII and T2DM risk in obese women.

The present study aimed to investigate the association between the inflammatory potential of the diet, as measured by the DII, and T2DM risk among obese adult women. The hypothesis of this study is that a higher DII score (indicating a pro-inflammatory diet) increases the risk of diabetes.

Main Points

- Diet-induced inflammation is related to type 2 diabetes mellitus (T2DM) risk.
- Dietary inflammatory index (DII) is a literature-derived index developed to measure the inflammatory potential of diet.
- It was hypothesized that using the DII in clinical practice may be useful to reduce the risk for diseases related to chronic inflammation including T2DM.
- To the best of our knowledge, this is the first case–control study that investigated the association between the DII and the risk for diabetes among obese women.
- A positive association was found between the dietary inflammatory potential measured by the DII and T2DM.
- In addition, the DII scores and insulin resistance were positively related.

METHODS

Participants

This case–control study was conducted in University of Health Sciences, Dışkapı Yıldırım Beyazıt Training and Research Hospital between September 2019 and March 2020. Voluntary subjects were female, aged 30-50 years with a body mass index (BMI) range of 30-35 kg/m^2. The case group consisted of 40 obese patients with T2DM, whereas the control group consisted of 40 obese patients without T2DM. Type 1 diabetes patients, type 2 diabetes patients receiving insulin treatment, and those with chronical disease were excluded from the study. The graphical abstract of the study is given in the supplementary Figure 1.

Written informed consent was obtained from each participant. The study protocol was approved by the Dışkapı Yıldırım Beyazıt Training and Research Hospital Clinical Research Ethics Committee on August 26, 2019, with the decision number 70/04.

Sociodemographic attributes of the individuals such as age, education level, and employment status and general information regarding their health statuses such as family history of diabetes, smoking, and alcohol intake were obtained by the researcher through face-to-face interviews and were recorded on the questionnaire form.

Anthropometric Measurements

Body weight of the participants was measured using a digital weight scale (Seca 769). The measurements were done with as few and as thin clothes as possible and without shoes in the morning while the participants were hungry. Body height was measured using the height ruler attached to the digital weight scale with the feet placed side by side and head in the Frankfort plane position. Body mass index was calculated after the measurements using the following formula: body weight (kg)/height (m)^2. Considering the World Health Organization (WHO) criteria,[23] individuals between 30 and 35 kg/m^2 were included in the study. Waist and hip circumference were appropriately calculated. The waist-to-hip ratio was calculated by proportioning the participants' waist circumference to their hip circumference. According to the WHO criteria, the participants with a waist/hip ratio of ≥0.85 were accepted to be at risk of developing metabolic complications.[24]

Calculation of Dietary Intake and Dietary Inflammatory Index

A quantitative FFQ was used to collect data on food consumption by the researcher in face-to-face interviews. Consumption frequency of food intake and the portion

size in the last 3 months was queried. To help participants quantify the portions consumed, food photographs were used. Dietary data from the FFQ were converted into the daily intakes (g/day), and using The Nutrient Database (BeBİS, Ebispro for Windows, Germany; Turkish Version/ BeBİS 8.2) total energy, macro, and micronutrient intakes, which were utilized to calculate the DII, were computed. The DII is a valid and reliable tool to measure the dietary inflammatory potential. The development and the calculation steps of the DII have been previously documented in detail.[12,25] Briefly, the DII is based on a literature review of 1943 articles published between 1950 and 2010 linking dietary components to the following inflammatory markers: CRP, IL-1β, IL-4, IL-6, IL-10, and TNF-α. A total of 45 different food parameters including macronutrients and micronutrients, as well as some bioactive components, were identified as linking to inflammation. In the present study, a total of 44 food parameters (except trans fatty acid) used for the DII calculation were available from the FFQ. Higher DII scores indicate a more pro-inflammatory diet; lower DII scores indicate a more anti-inflammatory diet.

Serum Collection and Laboratory Measurements

As laboratory measurements, fasting blood glucose (FBG), glycosylated hemoglobin (HbA1c), and fasting insulin levels were measured from the blood samples. From these data, the homeostatic model assessment of insulin resistance (HOMA-IR) value (the indicator of insulin resistance) was calculated as previously described.[26]

Statistical Analysis

First, tertile cutoff points for the DII score were defined based on the distribution among controls. Then, all the participants were categorized according to these cutoffs. Dietary Inflammatory Index tertiles were defined as $t_1 <$ –0.625; t_2 (–0.625) – (0.046); $t_3 >$0.046. Chi-square tests and Fisher's exact tests were used for categorical variables, and the Student's *t*-test, 1-way analysis of variance test, and Kruskal–Wallis test were used for continuous variables to evaluate differences between different groups. Linear regression was used to evaluate beta coefficients for glucose metabolism markers. Binary logistic regression was used to estimate crude and adjusted association between the DII score (continuous and categorical variable) and T2DM risk. Three regression models were used. The first model was the unadjusted logistic regression model. In the second model, age (continuous), physical activity, and standardized energy intake (kcal/day) were adjusted. In the final model, additionally, BMI was adjusted. Statistical analyses of the study were done using IBM SPSS (Statistical Package for Social Science, SPSS Company, Ill, USA) version 23. Statistical significance was defined as $P < .05$.

RESULTS

The distribution of baseline characteristics and some anthropometric measurements for both the case and control groups and the DII tertiles are shown in Table 1. Compared with controls, T2DM cases were more likely to have higher BMI. In the third tertile of DII score, the BMI measurements were also significantly higher than that observed in any other tertiles. The distribution of food consumption according to the tertiles is presented in Table 2. Compared to the individuals with lower DII scores, the intake of fiber, magnesium, iron, copper, vitamin A, vitamin C, vitamin E, beta carotene, folic acid, thiamine, and vitamin B_6 were significantly lower in the individuals with higher DII scores. Biochemical parameters and the beta coefficients regarding the DII scores are displayed in Table 3. As a continuous variable, the DII scores had a significant positive relationship with all glucose metabolism markers in the crude model. After adjustment for age, physical activity, standardized energy intake, and BMI, a significant positive association between the DII scores and HOMA-IR and insulin was observed. Table 4 shows ORs and 95% CI for T2DM by tertiles. After adjusting for potential confounding factors, the risk of T2DM was found to be 8 times higher in the highest tertile than in the lowest tertile.

DISCUSSION

In this case–control study, the possible role of the DII in diabetes pathogenesis was investigated, and a positive association was found between the dietary inflammatory potential measured by the DII and T2DM. These findings show that a more pro-inflammatory diet indicated by high DII scores may increase the risk for diabetes and that a more anti-inflammatory diet indicated by low DII scores may have a protective effect on the development of T2DM. To the best of our knowledge, this is the first case–control study that investigated the association between the DII and the risk for diabetes among obese women.

The role of increased inflammatory responses on T2DM pathogenesis is known, and dietary components are considered to affect the T2DM risk via inflammation.[8] There have been several studies evaluating the effects of dietary components or dietary models on T2DM risk via inflammation.[6,27] However, only a few studies have investigated the pro- and anti-inflammatory effects of the overall diet on T2DM.[28-30]

The DII was developed to measure the inflammatory potential of an overall diet based on an extensive literature search including studies that subject to dietary components and inflammatory markers.[12] To obtain the DII

Table 1. Baseline Characteristics and Some Anthropometric Measurements According to Case and Control Groups and Tertiles of the DII Score

Variable	Cases (n=40)	Controls (n=40)	*P*†	DII Tertiles			*P*‡
				T_1 (n=16) <-0.625	T_2 (n=27) -0.625 to 0.046	T_3 (n=37) >0.046	
Age (mean) (years)	43.5±4.2	36.5±5.7	**<.001**	38.0 ± 6.2	39.8 ± 6.3	41.0 ± 5.8	.249
Age groups (years)							
30-40	13 (32.5)	31 (77.5)	**<.001**	12 (75.0)	15 (55.6)	17 (45.9)	.148
41-50	27 (67.5)	9 (22.5)		4 (25.0)	12 (44.4)	20 (54.1)	
Occupation							
Housewife	22 (55.0)	19 (47.5)	.087	11 (68.8)	13 (48.2)	17 (45.9)	.168
Employed	18 (45.0)	16 (40.0)		5 (31.2)	10 (37.0)	19 (51.4)	
Unemployed	0 (0.0)	5 (12.5)		0 (0.0)	4 (14.8)	1 (2.7)	
Education level							
Primary school	8 (20.0)	6 (15.0)	.761	4 (25.0)	2 (7.4)	8 (19.2)	.534
High school	19 (47.5)	22 (55.0)		8 (50.0)	16 (59.3)	17 (45.9)	
University	13 (32.5)	12 (30.0)		4 (25.0)	9 (33.3)	12 (32.4)	
Marital status							
Married	29 (72.5)	32 (80.0)	.600	13 (81.2)	20 (74.1)	28 (75.7)	.976
Single	8 (20.0)	7 (17.5)		3 (18.8)	5 (18.5)	7 (18.9)	
Widow/divorced	3 (7.5)	1 (2.5)		0 (0)	2 (7.4)	2 (5.4)	
Smoking status							
Never	32 (80.0)	30 (75.0)	.911	11 (68.8)	23 (85.2)	28 (75.7)	.464
Former smoker	2 (5.0)	2 (5.0)		2 (12.5)	1 (3.7)	1 (2.7)	
Current smoker	6 (15.0)	8 (20.0)		3 (18.8)	3 (11.1)	8 (21.6)	
Family history of DM	27 (67.5)	8 (20.0)	**<.001**	7 (43.8)	8 (29.6)	20 (54.1)	.151
BMI (kg/m^2)	33.4 ± 1.6	32.7 ± 1.7	**.044**	32.3 ± 1.4	32.3 ± 1.4	33.8 ± 1.7	**<.001**
Waist circumference (cm)	101.2 ± 6.8	98.5 ± 6.1	.074	99.5 ± 6.1	96.4 ± 5.5	102.5 ± 6.5	**.001**
Hip circumference (cm)	118.1 ± 6.1	115.6 ± 6.2	.067	115.4 ± 6.2	113.6 ± 4.2	119.9 ± 6.2	**<.001**
Waist-to-hip ratio	0.86 ± 0.04	0.85 ± 0.05	.783	0.86 ± 0.05	0.85 ± 0.05	0.86 ± 0.04	.610
Waist-to-height ratio	0.64 ± 0.04	0.63 ± 0.04	.232	0.63 ± 0.04	0.61 ± 0.03	0.65 ± 0.04	**.002**
Physical activity							
Mild	5 (12.5)	3 (7.5)	.712	2 (12.5)	4 (14.8)	2 (5.4)	.544
Moderate	35 (87.5)	37 (92.5)		14 (87.5)	23 (85.2)	35 (94.6)	

Data were presented with mean ± SD or n (%), where appropriate.
ANOVA, analysis of variance; BMI, body mass index; DII, dietary inflammatory index; DM, diabetes mellitus.
†P values were obtained from Student's t-tests, χ^2 tests, and Fisher's exact test, where appropriate.
‡P values were obtained from ANOVA, χ^2 tests, and Fisher's exact test, where appropriate.

Table 2. Dietary Intakes According to Tertiles of the DII Score (n:80)				
	DII tertiles			
Variables	T_1	T_2	T_3	*P*†
Energy (kcal/day)	2092.2±436.6	2015.0±225.9	1985.9±356.5	0.576
Macronutrients				
Carbohydrates (g/day)	264.5±57.0	248.0±34.6	250.0±58.6	0.563
Fiber (g/day)	38.7±6.2	36.1±4.8	34.7±4.8	**0.041***
Protein (g/day)	80.3±12.2	82.1±10.0	79.0±11.3	0.553
Fat (g)	76.5±19.9	74.6±13.0	72.0±14.4	0.587
Saturated Fat (g)	29.5±6.4	31.1±4.8	29.1±8.6	0.536
Monounsaturated fatty acid (g)	25.7±7.3	23.8±3.8	23.7±5.0	0.411
Polyunsaturated fatty acid (g)	14.5 (10.8-20.4)	11.3 (10.8-15.5)	11.1 (9.6-13.9)	0.129
Omega-3 (g)	1.8 (1.5-2.8)	1.6 (1.3-2.1)	1.5 (1.2-1.9)	0.194
Omega-6 (g)	12.6 (9.5-17.0)	9.7 (9.1-13.3)	9.6 (8.2-11.9)	0.106
Micronutrients				
Magnesium (mg)	396.5±61.1	369.8±48.5	359.0±45.5	0.048
Phosphorus (mg)	1382.8±219.8	1419.9±203.0	1324.9±165.2	0.142
Iron (mg)	14.6±1.9	13.5±1.5	13.1±1.3	**0.006***
Zinc (mg)	11.6±1.8	11.3±1.7	10.8±1.5	0.184
Copper (µg)	2.2±0.4	2.0±0.2	1.9±0.3	**0.026***
Selenium (mg)	13.8 (10.8-14.0)	13.9(11.3-14.1)	13.8 (11.1-14.1)	0.142
Vitamin A (µg)	2004.9±625.9	1843.8±364.8	1547.3±416.4	**0.002***
Vitamin E (mg)	14.4±3.6	12.6±2.0	12.0±2.4	**0.011***
Beta-carotene (µg)	8423.1(6286.5-10118.5)	7449.3(6239.32-8875.2)	6282.9(5626.7-7292.2)	**<0.001***
Thiamine (mg)	1.2±0.2	1.1±0.1	1.1±0.1	**0.020***
Niacin (mg)	1.8±0.3	1.8±0.3	1.7±0.2	0.139
Riboflavin (mg)	29.5±4.1	28.6±3.5	27.7±4.4	0.293
Vitamin B6 (mg)	1.8 (1.5-1.9)	1.5 (1.4-1.6)	1.4 (1.3-1.6)	**0.002***
Vitamin B12 (µg)	3.4 (2.8-4.2)	3.5 (3.0-4.1)	3.4 (2.9-4.2)	0.748
Total folic acid (µg)	404.6 (339.9-443.1)	362.4 (340.4-391.3)	348.7 (323.3-370.1)	**0.005***
Vitamin C (mg)	168.3±29.7	145.8±28.1	138.4±27.9	**0.001***

Data were presented with mean±SD or median (IQR), where appropriate.
†P values were obtained from ANOVA and Kruskal-Wallis test, where appropriate.
*Different lowercase letters in a row indicate a statistically significant difference between group.

scores, the whole diet was taken into account, not just individual nutrients or food. Thus, the DII is considered advantageous in terms of measuring the overall inflammatory potential of the diet.[25] To date, there have been only a few studies investigating the association between the DII and T2DM, and their results are inconsistent.[28-30] A cross-sectional study that evaluated the relationship between the DII and T2DM among adults found that the

Table 3. Beta-coefficient for Glucose Metabolism Markers According to DII Score (n:80)									
	Model 1			Model 2			Model 3		
Dependent Variable	*β*	t	*P*	*β*	t	*P*	*β*	t	*P*
Fasting Blood Glucose	0.283	2.606	**0.011**	0.219	2.233	**0.029**	0.087	0.793	0.430
HbA1c	0.275	2.530	**0.013**	0.201	2.137	**0.036**	0.091	0.851	0.398
Insulin	0.430	4.202	**<0.001**	0.447	4.259	**<0.001**	0.259	2.270	**0.026**
HOMA-IR	0.488	4.933	**<0.001**	0.472	4.716	**<0.001**	0.265	2.492	**0.015**

Linear regression.
Model 1: Crude model.
Model 2: Adjusted for age, physical activity and standardized energy intake.
Model 3: Adjusted for age, physical activity, standardized energy intake and BMI.

Table 4. Odds Ratios (ORs) and 95% Confidence Intervals (95% CIs) for T2DM According to Tertiles of the DII Score (n:80)						
	Model 1		Model 2		Model 3	
DII tertiles	OR (95% CI)	*P*	OR (95% CI)	*P*	OR (95% CI)	*P*
1 (<-0.625)	Reference	-	Reference	-	Reference	-
2 (-0.625-0.046)	4.024 (0.930-17.411)	0.062	4.836 (0.750-31.173)	0.097	5.294 (0.790-35.475)	0.086
3 (>0.046)	8.000 (1.923-33.724)	**0.004**	10.772 (1.646-70.469)	**0.013**	8.566 (1.235-59.437)	**0.030**
DII (as continuous)	2.316 (1.276-4.205)	**0.006**	2.312 (1.094-4.890)	**0.028**	2.043 (0.955-4.372)	0.066

Logistic regression.
Model 1: Crude model.
Model 2: Adjusted for age, physical activity and standardized energy intake.
Model 3: Adjusted for age, physical activity, standardized energy intake and BMI.

subjects in the highest quintile according to the DII scores were at a higher risk of T2DM than those in the lowest quintile.[28] King et al[29] found that the DII was significantly related to both the presence and the severity of diabetes and that with a 1-point increase in the DII score, having >9% HbA1c value increased by 43%. However, in another study conducted in Iran, the DII was not associated with the T2DM incidence.[30] The study mentioned above found that the DII was moderately related to the T2DM risk; however, the results were not statistically significant after multivariable adjustment. The present study indicated that participants with the higher DII score had an 8-fold increased risk of T2DM compared to those with the lowest DII scores.

Regarding the nutrient intake, it was observed that participants who consumed a more pro-inflammatory diet (in the highest tertile) had lower intakes of dietary fiber and some anti-inflammatory vitamins and minerals. Consistent with the findings of the current study, a study conducted in Mexico also reported that the intake of fiber and various vitamins and minerals was lower in the highest quintile than in the lowest quintile.[28] The positive effects of dietary fiber on inflammation have been reported.[9] Indeed, it was shown that dietary fiber and CRP levels are inversely related, and the intake of high dietary fiber both as part of the diet and as a supplement decreased serum CRP concentrations (14% and 18%, respectively).[9] Moreover, previous studies have shown that some vitamins and minerals, particularly antioxidant vitamins, reduce inflammation markers.[31,32] Besides, although it is considered insignificant, the lowest intakes of the fatty acids, including monounsaturated fatty acid (MUFA), polyunsaturated fatty acid (PUFA), omega 3, and omega 6, which are considered to be anti-inflammatory, were in the highest tertile in the present study. Consistent with the findings of the current study, a cross-sectional study that investigated the DII and dietary habits in individuals with T2DM also reported similar results.[33] Additionally, there have also been several studies conducted with different populations that obtained similar results.[28,34]

One of the possible mechanisms of the association between the DII and diabetes risk may be that a pro-inflammatory diet causes insulin resistance by affecting

levels of various inflammatory cytokines. Previous studies have shown positive associations between various inflammatory markers (CRP, TNF, IL-6, etc.) and insulin resistance.[5,35] The DII, which measures the dietary inflammatory potential, may also be positively related to insulin resistance, which is connected with inflammatory processes through inflammation; thus, this association may affect the diabetes risk.[8,28,33] These findings that support this association were also obtained in the present study. It was observed that the DII scores were significantly related to HOMA-IR, the biochemical marker of insulin resistance, even after the multivariable adjustment. There have also been various studies that obtained findings similar to the present study.[8,36] A study investigated the dietary inflammatory potential using the ADII (Adjusted-Dietary Inflammatory Index), which was developed with some modifications in the DII scoring algorithm, found a significant association between the DII scores and HOMA-IR and that the association between diet and insulin resistance was slightly mediated by inflammation.[15] Another study conducted with South African women also found a positive relationship between the DII and all glucose metabolism markers, including HOMA-IR.[36] There have also been some studies that reported different findings than those of the present study. The ORISCAVLUX (Observation of Cardiovascular Risk Factors in Luxemburg) survey found no significant association between the DII scores and any of the glucose metabolism markers.[22] Another study found that high DII scores were related only to postprandial blood glucose among the glucose metabolism markers.[21] These different findings may have been obtained due to the study designs, study populations, and the type and number of parameters used to calculate the DII scores.

The present study has several strengths to be noted. First, this is the first case–control study investigating the association between the DII and the risk for T2DM among obese women. In addition, the study control group was selected attentively. Controls were selected among obese individuals to understand any effect that may be caused by obesity and to see better the effect of diabetes in the association between the DII and diabetes among obese women individuals. Also, the researchers tried to ensure homogeneity in the study by keeping the inclusion criteria of age and BMI within narrow intervals. Since the participants were women, an age limitation was implemented to eliminate the effects of menopause-related physiological changes. Moreover, although FFQ may lead to measurement error, even in healthy individuals, its use in the present study enabled the researchers to reach many of the food parameters required to calculate the DII. In many previous studies, fewer parameters were used.[28,29] Despite these strengths, some limitations should be acknowledged. First, since the study was based on observational data, the cause–effect relationships cannot be inferred. Therefore, the findings need to be further evaluated in future longitudinal studies. Second, its small sample size may have affected the statistical power to determine some effects. Third, although a detailed FFQ was used to determine the intake of food parameters utilized to calculate the DII scores, using FFQ that is based on the individuals' memory, may cause possible misreporting. Finally, the study population consists of exclusively women. This limitation may be considered minor since no gender differences in biological mechanisms have been reported to date.

In conclusion, the current study provided evidence that women consuming a pro-inflammatory diet with higher DII scores had a higher risk of diabetes compared to women with lower DII scores consuming a more anti-inflammatory diet. In addition, the DII scores and insulin resistance were positively related. Given these findings, adopting an anti-inflammatory diet may be an important approach in preventing insulin resistance and reducing the risk of diabetes. Further longitudinal studies are needed to determine the causality between the DII and diabetes. Such studies may test whether the DII would be useful in practice, and especially whether a diet model created considering the DII parameters would reduce inflammation and the risk of diabetes. Finally, the DII may be an essential tool to characterize the diet of populations. It may be expanded further in clinical researches to reduce the risk of diseases related to chronic inflammation.

Ethics Committee Approval: Ethics committee approval was received for this study from the ethics committee of University of Health Sciences, Dışkapı Yıldırım Beyazıt Training and Research Hospital (Date: August 26, 2019, Number: 70/04).

Informed Consent: Written informed consent was obtained from all participants who participated in this study.

Peer-review: Externally peer-reviewed.

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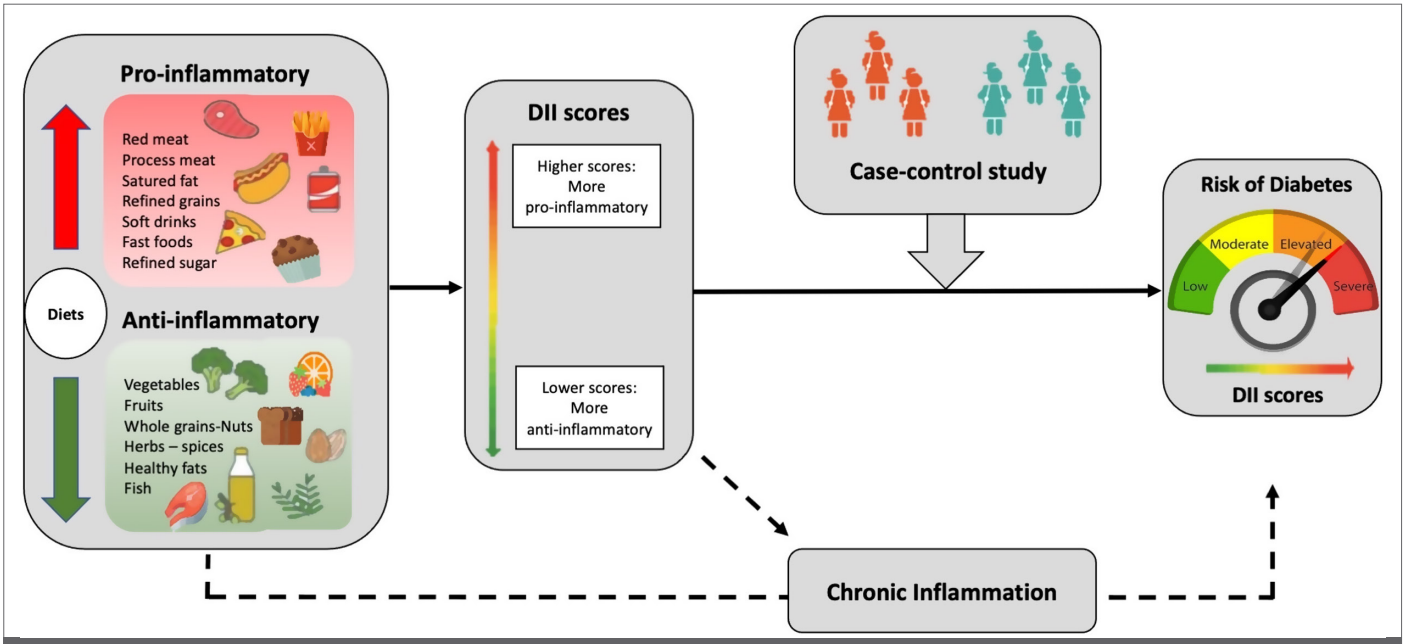

Supplementary Figure 1. Graphical abstract of the study.