Nutrition Journal

RESEARCH

Open Access

Contribution of habitual and meal-specific dietary inflammatory index and its associations with obesity and metabolic risk indicators in women

Azadeh Lesani[1,2*], Elnaz Daneshzad[3], Mar Calvo-Malvar[4,5], Cain C. T. Clark[6], Hoda Zahedi[7,8], Sofia Vilela[9] and Mansooreh Sadat Mojani-Qomi[10,11*]

Abstract

Background Inflammatory diets are associated with obesity and cardiometabolic dysfunction, nevertheless, it remains unclear whether overall dietary inflammation or the timing of pro-inflammatory meals yields a greater metabolic impact. This study utilizes the energy-adjusted dietary inflammatory index (EDII) to examine associations of both habitual/meal-specific with metabolic risk among women.

Methods In this cross-sectional study, 562 Iranian women (aged 20–60) from Tehran were assessed. Habitual and four meal-specific EDII scores (breakfast, lunch, afternoon snacks, dinner) were calculated from three non-consecutive 24-hour dietary recalls using 29 pro- and anti-inflammatory parameters. Anthropometric variables, fasting blood assays, body mass index (BMI), Visceral Adiposity Index (VAI), Cardiometabolic Index (CMI), and Atherogenic Profile Index (API) were measured. Multivariable linear and logistic regression models, adjusted for potential confounders, were used to estimate associations.

Results Habitual-EDII showed the strongest association with afternoon snack-EDII ($R^2 = 0.36$), followed by Dinner (0.29), Lunch (0.25), and Breakfast (0.20). Habitual-EDII in T (tertile) 3 vs. T1 was associated with increased odds of overweight/obesity BMI [OR 2.38, 95% CI (1.51–3.81)], CMI [1.95 (1.23–3.04)], VAI [1.92 (1.21–3.03)], and API [2.53 (1.57–4.08)]. Afternoon snack-EDII was associated with higher odds of overweight/obesity [OR 1.93 (1.21–3.11)]. Finally, dinner-EDII was associated with an elevated risk of VAI [OR 1.85 (1.17–2.92)] and CMI [OR 1.77 (1.12–2.80)].

Conclusion Pro-inflammatory diets, especially during afternoon and dinner, are strongly associated with cardiometabolic risk, highlighting a meal-specific inflammation relationship. Accordingly, prospective cohort studies and interventions studies are required to confirm the veracity of these findings.

Keywords Dietary inflammatory index, Diet, Meal timing, Obesity, Meals, Cardiometabolic risk factors

*Correspondence:
Azadeh Lesani
a_lesani@alumnus.tums.ac.ir
Mansooreh Sadat Mojani-Qomi
mojani@iau.ac.ir

Full list of author information is available at the end of the article

Introduction

Unhealthy dietary patterns are widely acknowledged as major contributors to chronic noncommunicable diseases (NCDs) such as obesity, type 2 diabetes, and cardiovascular disease [1]. A central mechanism linking poor dietary quality to these conditions is the chronic low-grade inflammation it induces, both at the intestinal level and systemically [2]. To quantify this inflammatory potential, the Dietary Inflammatory Index (DII) was developed as a standardized, literature-based tool supported by experimental and epidemiological evidence [3, 4].

Higher DII scores, reflecting more pro-inflammatory dietary patterns, have consistently been associated to adverse anthropometric and metabolic outcomes [5–8]. Beyond overall dietary intake, emerging research indicates that the timing of food consumption chrono-nutrition may also influence metabolic health independently of diet composition [9–11]. Meal timing is closely connected to an individual's chronotype [12], a biologically driven preference for daily patterns of activity and alertness [13],which shapes physiological responses to food intake and may therefore modulate metabolic regulation. However, despite the growing interest in chrono-nutrition, the inflammatory potential of meals consumed at different times of day remains largely unexplored. Investigating whether meal-specific inflammatory profiles differ across eating occasions may help clarify timing-specific mechanisms contributing to metabolic disturbances. The recent development of the meal-specific DII provides a means to assess the inflammatory potential of individual meals rather than relying solely on aggregated 24-hour intake. offering greater resolution to detect patterns that might otherwise be overlooked [11]. Additionally, women may exhibit distinct inflammatory responses due to differences in hormonal profiles, fat distribution, and metabolic regulation [11, 14], yet they remain underrepresented in studies examining dietary inflammation, meal timing, and metabolic health. To address these gaps, this study aims to examine the associations between habitual and meal-specific energy-adjusted DII (E-DII) and a comprehensive set of obesity indices and cardiometabolic markers in Iranian women. A second objective is to determine whether meal timing modifies the inflammatory potential of the diet, an aspect that remains largely unexplored in Middle Eastern populations undergoing rapid nutritional and lifestyle transitions. Although causal inference cannot be established due to the cross-sectional design, this study represents one of the first efforts to integrate chrono-nutritional perspectives with inflammatory dietary profiling in women. The findings may help inform future research and guide the development of targeted dietary strategies to prevent or mitigate metabolic disorders in this underrepresented group.

Methods

Study design

This cross-sectional study was conducted between November 2024 and January 2025 among adult Iranian women who attended three primary healthcare centers located in the southern region of Tehran. Participants were recruited using a proportion-to-size convenience sampling approach. The inclusion criteria were women aged 20–60 years with a body mass index (BMI) between 18.5 and 39.9 kg/m². Exclusion criteria included pregnancy, lactation, shift work, the presence of any acute or chronic medical condition, and implausible reports of total energy intake, defined as under- or over-reporting. A total of 560 participants were required, with the sample size calculated based on the primary outcome of abdominal obesity (see Supplementary Material [SM1] for details).

Ethical approval

The study protocol was reviewed and approved by the Research Ethics Committee of Islamic Azad University, Tehran Medical Sciences—Pharmacy and Pharmaceutical Sciences Faculty (IR.IAU.REC.1403.487), and was carried out in accordance with the Declaration of Helsinki and relevant institutional guidelines. All participants provided written informed consent prior to participation. The research adhered to the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) reporting guideline. The current data analysis was performed between July 7, 2025, and July 20, 2025.

Procedures

Data were collected by trained examiners through face−to-face interviews. Sociodemographic characteristics, including age, marriage status, education level, smoking status, menopausal status, and sleep duration were recorded. Women aged 20–60 years were included to capture premenopausal, perimenopausal, and postmenopausal stages, and menopausal status was adjusted for in all analyses to account for hormonal differences that could affect metabolic outcomes. Total sleep duration was calculated based on formula: [(2 × weekend sleep duration) + (5 × weekday sleep duration)]/7 (see Figure S1 in the Supplementary Material).

Socioeconomic status (SES)

Socioeconomic status was assessed using principal component analysis (PCA) based on household income and ownership of assets such as cars, housing, and electronic devices. Participants were categorized into three SES groups, low, medium, and high, according to the PCA-derived scores.

Anthropometric assessment

Body weight was measured using a Seca digital scale (Seca & Co. KG, Hamburg, Germany; Model: 874 1321009), with participants wearing light clothing and unshod. Standing height was measured to the nearest 0.1 cm using a wall-mounted stadiometer, also unshod. BMI was then calculated as weight (kg) divided by height squared (m^2). Waist circumference (WC) was measured at the midpoint between the lower margin of the last palpable rib and the top of the iliac crest using a non-stretchable fiberglass measuring tape, in accordance with the World Health Organization (WHO) protocol [15]. Hip circumference was also measured, and the waist–hip ratio (WHR) was computed by dividing WC (cm) by hip circumference (cm), serving as an indicator of body fat distribution and associated health risks. Also, waist-to-height ratio (WHtR), a reliable indicator of central adiposity, was calculated by dividing WC (cm) by height (cm) [16].

Physical activity

Physical activity was assessed using the short form of the validated International Physical Activity Questionnaire (IPAQ) [17]. Participants reported time spent walking or engaging in moderate and/or vigorous physical activities over the previous seven days. Physical activity levels were calculated as metabolic equivalent minutes per week (MET-min/week) and categorized as follows 1- low: <600 MET-min/week, 2- moderate: 600–3000 MET-min/week, and 3- High: >3000 MET-min/week [18].

Chronotype

Chronotype was evaluated using the Morningness–Eveningness Questionnaire (MEQ), a 19-item scale developed by Horne and Östberg [19]. The MEQ asks respondents about sleep–wake patterns and preferred timing for physical and mental activities. Items have different response options and scoring weights, based on the original developers' analysis. The total score ranges from 16 to 86, with higher scores indicating a morning preference. We applied a Persian-language validated version of the MEQ [20].

Dietary intake assessment, eating occasion, and eating window

Dietary intake data were collected using three non-consecutive 24-hour dietary recalls, conducted by trained dietitians during private interviews. The first recall was administered in person during the participants' initial visit to the healthcare center, while the remaining two recalls were conducted by telephone on randomly selected days. The habitual intake was defined as the average of the three recalls to capture usual dietary patterns. Participants were asked to report all eating occasions with time of intake, categorized as breakfast, lunch, dinner, or snacks. Eating occasions were defined based on the timing of food intake as follows: breakfast as the main meal consumed between 5:00 a.m. and 11:00 a.m.; lunch was considered between 11:00 a.m. and 4:00 p.m.; afternoon snacks was any eating occasion occurring between 4:00 p.m. and 6:00 p.m.; and dinner was known as what was consumed after 6:00 p.m. and extended into the early morning [21]. Meals were standardized to contain no more than one breakfast, lunch, and dinner, but allowing for multiple snacks. An eating occasion (EO) was defined as any event during which at least 50 kilocalories were consumed within a 15-minute interval [22]. Food intake from all reported meals and snacks across the three recalls was converted to grams per day using standard portion sizes and household measurement tools [23]. Energy and nutrient intakes were analyzed using Nutritionist IV software (First Databank, San Bruno, CA, USA), which was adapted for Iranian foods. A total of 435 food items were derived from 24-hour dietary recalls and were classified into 28 food groups, Table Supplementary 1 (Table S1, list of food groups at the level of day and meal-specific intake), based on the similarity of nutrient content in each food item and a literature search [24–27].

Cardiometabolic biomarker

A 10 mL fasting venous blood sample was collected from each participant between 7:00 and 10:00 a.m. following a 12-hour overnight fast, to ensure consistency in metabolic assessments. Blood was drawn into acid-washed tubes without anticoagulants. After allowing the blood to clot at room temperature for 30 min, samples were centrifuged at 1500 × g for 20 min. The resulting serum was then aliquoted and stored at − 80 °C until laboratory analysis.

Total cholesterol (TC) and high-density lipoprotein cholesterol (HDL-C) concentrations were determined using the cholesterol oxidase–phenol–aminoantipyrine enzymatic method. Triglycerides (TG) were measured using the glycerol-3-phosphate oxidase–phenol–aminoantipyrine enzymatic method, (Pars Azmun, Tehran, Iran).

Assessment Energy Adjusted Dietary Inflammatory Index (EDII)

Dietary intake data obtained from the three non-consecutive 24-hour dietary recalls were used to calculate the energy-adjusted Dietary Inflammatory Index (EDII) for all participants. The EDII, developed by Shivappa et al. [28, 29], quantifies the inflammatory potential of the diet by scoring dietary components according to their documented effects on circulating inflammatory biomarkers. Although the original index includes 45 food parameters, the EDII in the present study was computed using 29

parameters due to the limited composition database, as well as the infrequent consumption of certain foods in the Iranian diet [5, 8, 30]. Pro-inflammatory parameters included energy, carbohydrate, total fat, protein, cholesterol, saturated fat, trans fat, vitamin B12, and iron. Anti-inflammatory parameters included monounsaturated and polyunsaturated fatty acids, dietary fiber, vitamins B6, folic acid, niacin, riboflavin, thiamin, and vitamins A, C, D, E, β-carotene, as well as zinc, selenium, magnesium, caffeine, pepper, onion, and tea [31]. Prior to EDII calculation, the intake of each nutrient was adjusted for total energy intake using the residual method [32]. Then, to compute the EDII score for each participant, we first calculated a z-score for each dietary parameter by subtracting the corresponding "global mean" from the participant's energy-adjusted intake and dividing the result by the "global standard deviation." The global means and standard deviations for each food parameter were obtained from the study by Shivappa et al. [28]. The z-scores were then converted to centered percentile scores to minimize the impact of skewed distributions. Each centered percentile was multiplied by the respective inflammatory effect score. The overall EDII score was obtained by summing the weighted values across all 29 components, with higher (more positive) scores indicating a pro-inflammatory diet and lower (more negative) scores reflecting an anti-inflammatory dietary pattern.

EDII scores were computed for habitual daily intake (Habitual-EDII) and for individual eating occasions (breakfast, lunch, afternoon snack, and dinner) to examine the inflammatory potential of the diet across the day. Supplementary Material (SM2) offers further methodological information on the computation of the Energy-Adjusted Dietary Inflammatory Index (EDII). Table S2 summarizes the distribution of nutrient intake, including pro- and anti-inflammatory components, across individual meals as well as daily intake.

Outcomes

Metabolic and atherogenic risk assessments

The visceral adiposity index (VAI) was used as an estimate of visceral fat, incorporating WC, BMI, triglycerides (TG), and high-density lipoprotein cholesterol (HDL-C), with sex-specific equations applied [33], VAI = WC (cm)/39.58 × 1.89 × BMI (kg/m^2) × TG (mmol/dl)/1.03 × 1.31/HDL (mmol/dl).

The Atherogenic lipid indices were calculated as follows:[34], [35].

$$\text{Cardiometabolic index(CMI)} = \text{WHRt} \times \text{TG (mmol/dl)} / \text{HDL} - \text{C(mmol/dl)}$$

$$\text{Atherogenic index of plasma (AIP)} = \log \text{TG(mmol/dl)/HDL} - \text{C(mg/dl)}$$

Statistical analysis

Descriptive statistics were reported as means ± standard deviations (SDs) for continuous variables and as frequencies (percentages) for categorical variables. To compare general characteristics and cardiometabolic parameters across Habitual-EDII, one-way analysis of variance (ANOVA) was used for continuous variables, and the chi-square (χ^2) test was applied for categorical variables. A p-value of less than 0.05 was considered statistically significant.

To select covariates for adjustment, we applied Directed Acyclic Graph (DAG) theory to identify the minimally sufficient adjustment set [36]. Figure S3 shows the potential confounders in the model as defined by the DAC theory. Based on this approach, the models were adjusted for age, total energy intake (TEI), physical activity level, marital status, SES, smoking status, education level, menopausal status, Chronotype, sleep duration, number of EOs, supplement use, and BMI, except in models where BMI was the outcome.

To examine differences in the mean of dietary intake and temporal eating patterns across categories of Habitual-EDII, we conducted an analysis of covariance (ANCOVA). The models were adjusted for potential confounders.

Linear regression was used to assess the association between Habitual-EDII and food groups, meal-specific EDII, dependent variables, and tertiles of Habitual-EDII (independent variable), as well as to evaluate the association of Habitual-EDII (per 10-point increase) to each meal-specific EDII score, model was adjusted for possible confounders. The R^2 statistic was used to indicate the proportion of variance in each meal-specific EDII score explained by Habitual-EDII.

Generalized linear regression (GLR) was conducted to assess the associations between Habitual- and meal-specific EDII and dichotomized outcomes: BMI (≥ 25 vs. < 25), VAI (≥ 4.28 vs. < 4.28) [37], CMI (≥ 0.55 vs. < 0.55), and API (≥ 0.11 vs. < 0.11) [34]. Models were adjusted for the set of potential confounders identified through the DAG. Additionally, multicollinearity was assessed, and all variance inflation factor (VIF) values were below 5, indicating no significant multicollinearity concerns.

To control for false positives from multiple tests, we applied the false discovery rate (FDR) method with a 5% threshold. This approach ensures that no more than 5% of the statistically significant results are expected to be false positives, maintaining the integrity of the findings [38].

To assess the plausibility of self-reported dietary intake, the ratio of energy intake (EI) to basal metabolic rate (BMR) was calculated. BMR was estimated using the Harris–Benedict equation. Underreporting was defined as EI: BMR < 1.35 and overreporting as EI: BMR > 2.40, based on established cutoffs [29]. Participants classified as underreporters (n = 8) or overreporters (n = 17) were excluded from the analysis. All primary analyses were conducted in the remaining sample (n = 562).

Sensitivity analyses examined potential interactions by age (< 40 vs. ≥ 40 years, median = 40) across all associations. All statistical analyses were performed using IBM SPSS Statistics version 26.0 (IBM Corp., Armonk, NY, USA). Two-sided P values < 0.05 were considered statistically significant.

Results

Participants characteristic

This cross-sectional study included 562 healthy Iranian women (mean age: 42.02 ± 10.88y). A flow diagram illustrating participant selection and inclusion is provided in Figure S2. The mean (95% CI) of Habitual-EDII across its tertiles was − 9.08 (−9.77, −8.51) in Tertile 1 (T1), 1.74 (1.32–2.11) in (T2), and 12.43 (11.26, 13.25) in (T3), with a significant (p-trend < 0.001). The demographic and cardiometabolic characteristics of participants across tertiles of Habitual-EDII are shown in Table 1. Age, physical activity level, SES, sleep duration, and chronotype (MEQ score) were comparable across Habitual-EDII tertiles. However, BMI and cardiometabolic indices (including VAI, CMI, and API) were higher in the T3 vs. T1, indicating a less favourable metabolic profile.

Dietary characteristics

Table 2 presents the linear regression analyses, adjusted for potential confounders, to assess the variation in food group consumption across habitual-EDII tertiles. The analysis revealed that participants in the highest Habitual-EDII tertile consumed significantly greater amounts of refined grains (p-trend < 0.001), potatoes (p-trend < 0.001), legumes and seeds (p-trend = 0.04), processed meat (p-trend = 0.04), red meat (p-trend = 0.01), dairy products (p-trend < 0.001), sweets and desserts (p-trend = 0.03), fast foods (p-trend = 0.04), soft drinks (p-trend = 0.02), and mayonnaise (p-trend = 0.04). Conversely, intakes of vegetables (p-trend < 0.001), vegetable

Table 1 Demographic, lifestyle characteristics, and health outcomes of Iranian women (n = 562)

Variables	Total population	Energy adjusted habitual dietary inflammatory index (Habitual-EDII) 1.96 (0.87–2.51)[1]		
		T1 ≥ − 2.42	−2.42 < T2 < 6.06	T3 ≥ 6.06
Subjects	562	187	188	187
Age (years)	42.02 ± 10.88	42.63 ± 10.59	40.62 ± 11.15	42.82 ± 10.80
Physical activity (< 600 MET-min/week) %	287 (51.1)	98 (52.40)	102 (54.30)	87 (46.50)
Socio-economic status (SES) (low SES) %	193 (34.40)	63 (33.70)	68 (36.20)	62 (33.20)
Education (Educated[2]) %	185 (32.90)	68 (36.40)	63 (33.50)	54 (28.90)
Smoking status (yes) %	25 (3.40)	11 (5.80)	9 (4.80)	5 (2.70)
Marriage status (married) %	448 (79.70)	151 (80.70)	146 (77.70)	151 (80.70)
Supplementary intake (yes) %	136 (24.20)	45 (24.10)	49 (26.10)	42 (22.50)
Menopause status (yes) %	209 (37.2)	68 (36.40)	66 (35.10)	75 (40.10)
Total sleep duration[3] (h: m)	6:51 ± 1:10	6:45 ± 1:09	6:54 ± 1:07	6:53 ± 1:13
Chronotype [*Morning evening questionnaire score (MEQ)*]	58.29 ± 6.57	58.17 ± 6.54	58.80 ± 6.58	57.90 ± 6.61
WHR	0.85 ± 0.07	0.84 ± 0.06	0.84 ± 0.07	0.85 ± 0.08
BMI (kg/m2)	25.99 ± 3.75	25.18 ± 2.94	25.53 ± 3.60	27.25 ± 4.26
BMI < 25	237 (42.2)	95 (49.20)	84 (44.70)	58 (31.00)
VAI	3.48 ± 1.49	3.27 ± 1.43	3.31 ± 1.37	3.73 ± 1.64
VAI < 4.28	267 (47.50)	99 (52.90)	91 (48.40)	77 (41.20)
CMI	1.09 ± 0.68	1.01 ± 0.63	1.04 ± 0.64	1.23 ± 0.74
CMI < 0.55	258 (45.9)	99 (52.90)	87 (46.30)	72 (38.50)
API	0.23 ± 0.05	0.22 ± 0.02	0.22 ± 0.03	0.24 ± 0.09
API < 0.11	278 (49.46)	110 (58.50)	91 (48.4)	77 (41.2)

Values are presented as mean ± standard deviation (SD) for continuous variables, analysed using one-way ANOVA, or as number (percentage) for categorical variables, analysed using the Chi-square test

Abbreviations: *n* Number, *%* Percentage, *T* Tertile, *MET-min/week* Metabolic equivalent of task minutes per week, *SES* Socio-economic status, *h:m* Hour: minuts, *BMI* Body mass index, *WHR* Waist-to-hip ratio, *VAI* Visceral adiposity index, *CMI* Cardiometabolic index, *API* Atherogenic index of plasma

[1] Mean (95% confidence interval)

[2] Educated refers to participants who graduated from university

[3] Total sleep duration = ((2 × weekend sleep duration) + (5 × weekday sleep duration))/7

Table 2 Associations between food groups intake and energy adjusted habitual dietary inflammatory index (Habitual-EDII) of Iranian women ($n = 562$)

Food groups	Energy adjusted habitual dietary inflammatory index (Habitual-EDII)			*p*-trend[1]
	T1 ≥ −2.42	−2.42 < T2 < 6.06 β (95% CI)	T3 ≥ 6.06 β (95% CI)	
Subjects	187	188	187	
Refined grains (g/day)	Ref.	**17.89 (0.55–35.18)**	**38.40 (20.78–56.03)**	**< 0.001**
Whole grains (g/day)	Ref.	−1.23 (−2.04–0.52)	**−4.08 (−8.13 – −0.05)**	0.32
Fruits (g/day)	Ref.	−8.17 (−23.01–6.68)	−12.43 (−27.38–1.89)	0.47
Vegetable (g/day)	Ref.	**−20.82 (−35.23 – −6.42)**	**−30.22 (−45.08 – −15.76)**	**< 0.001**
Potato (g/day)	Ref.	**2.17 (0.3–4.33)**	**3.58 (1.39–5.77)**	**< 0.001**
Legume & seed (g/day)	Ref.	−0.54 (−3.95–2.86)	−4.23 (−8.08 – −1.41)	**0.04**
Egg (g/day)	Ref.	−4.55 (−8.50 - −0.69)	0.08 (−3.79–3.98)	0.08
Processed meat (g/day)	Ref.	**4.93 (0.36–9.51)**	**5.41 (0.75–10.06)**	**0.04**
Red meat (g/day)	Ref.	−0.63 (−6.54–5.23)	**6.55 (0.55–12.56)**	**0.01**
Poultry & fish(g/day)	Ref.	−0.91 (−6.65–4.84)	0.18 (−5.49–5.86)	0.54
Dairy (g/day)	Ref.	10.02 (−10.28–20.29)	**31.86 (17.19–79.80)**	**< 0.001**
Vegetable oil (g/day)	Ref.	−1.43 (−2.74 – −0.41)	**−1.62 (−2.45 – −0.47)**	**0.04**
Olive oil (g/day)	Ref.	**−1.35 (−2.65 - −0.06)**	**−1.44 (−2.76 – −0.12)**	**0.03**
Hydrogenated oil (g/day)	Ref.	**0.73 (−0.44–1.90)**	**1.49 (0.29–2.86)**	0.08
Sweets and desserts (g/day)	Ref.	**10.02 (3.23–16.80)**	**11.07 (4.17–17.98)**	**0.03**
Fast foods (g/day)	Ref.	5.02 (−5.28–15.49)	**8.86 (1.19–19.22)**	**0.04**
Soft drink (g/day)	Ref.	6.21 (−3.23–16.42)	**11.63 (1.25–22.04)**	**0.02**
Mayonnaise (g/day)	Ref.	**2.83 (0.36–6.91)**	**3.41 (0.75–7.06)**	**0.04**

Adjusted for age, TEI, physical activity, marital status, socio-economic status, smoking status, education level, menopausal status, chronotype, sleep duration, number of EOs, supplement intake, and BMI

Abbreviations: *T* Tertile, *β* Beta, *CI* Confidence interval, *EDII* Energy-adjusted dietary inflammatory index, *BMI* Body mass index, *MEQ* Morningness–Eveningness Questionnaire, *TEI* Total energy intake, *EOs* Eating occasion

[1] Obtained from linear regression

oil (p-trend = 0.04), and olive oil (p-trend = 0.03) were lower. Whole grain consumption showed a non-significant decreasing trend (p-trend = 0.32), while intakes of eggs, poultry, fish, and hydrogenated oil did not vary across tertiles. These results indicate that more pro-inflammatory diets are characterized by higher consumption of processed and energy-dense foods and lower intake of anti-inflammatory sources, such as vegetables and healthy fats (Table 2).

Habitual- and meal-specific EDII contribution

The mean of Breakfast-EDII in the tertiles of Habitual-EDII was −1.42 in (T1), 0.08 in (T2), and, 1.93 with (p-trend < 0.001). The mean of Lunch-EDII in the tertiles of Habitual-EDII was −1.72 in (T1), −1.09 in (T2), and 1.46 in (T3), with (p-trend < 0.001). Afternoon snack-EDII in the tertiles of Habitual-EDII was −2.36 in (T1), 2.02 in (T2), and 4.21 in (T3), with (p-trend < 0.001). Dinner-EDII in the tertiles of Habitual-EDII was −1.54 in (T1), 0.90 in (T2), and 2.89 in (T3), with (p-trend < 0.001). The mean (95% CI) values were 0.28 (0.04, 0.65) for Breakfast-EDII, 0.29 (−0.14, 0.69) for Lunch-EDII, 0.64 (0.21, 1.20) for Afternoon snack-EDII, and 0.39 (−0.03, 0.78) for Dinner-EDII.

Meal-specific analyses demonstrated that higher breakfast-EDII tertiles were associated with lower protein (p = 0.03), fiber ($p = 0.03$), and energy intake at breakfast ($p = 0.01$) and lunch ($p = 0.02$). Lunch-EDII tertiles were linked to higher saturated fat ($p = 0.04$) and MUFA intake ($p = 0.03$), lower fiber ($p = 0.03$), and greater lunch energy intake ($p = 0.03$). For afternoon snack-EDII, energy ($p = 0.01$), total fat (p = 0.04), cholesterol ($p = 0.04$), and dinner energy ($p = 0.02$) increased across tertiles, whereas MUFA decreased ($p = 0.04$). Higher dinner-EDII scores were associated with lower fiber (p = 0.06) and MUFA ($p = 0.03$), and increased cholesterol ($p = 0.04$) and dinner energy intake ($p = 0.02$) (Table 3). Overall, these findings suggest that more pro-inflammatory dietary patterns, particularly at specific meals, are associated with less favorable nutrient profiles and altered energy distribution throughout the day.

The linear regression beta coefficients (95% CI) for meal-specific EDII and Habitual- EDII are detailed in Table 4. In Model 2 (full adjusted), Afternoon Snack-EDII demonstrated the strongest association with Habitual-EDII, yielding the highest R^2 (0.36) and the largest beta across tertiles in all models. This was followed by Dinner-EDII (R^2 = 0.29), Lunch-EDII (R^2 = 0.25), and Breakfast-EDII (R^2 = 0.20). Participants in the T3 of Habitual-EDII

Table 3 Energy, nutrients intake, and eating habits of study participants across tertile of habitual and meal-specific energy adjusted dietary inflammatory index (EDII) score, *n* = 562

Variables	Energy adjusted habitual dietary inflammatory index (Habitual-EDII) 1.96 (0.87–2.51)[1]			*P*-value
	T1 ≥ − 2.42	**−2.42 < T2 < 6.06**	**T3 ≥ 6.06**	
Subjects	187	188	187	
Total energy intake (TEI) (kcal)	1610 ± 80.31	1668.90 ± 70.36	1790.09 ± 88.06	0.05
Carbohydrate (% of TEI)	55.44 ± 2.59	57.02 ± 1.44	61.80 ± 2.42	**0.01**
Protein (% of TEI)	13.98 ± 0.51	14.22 ± 0.42	14.40 ± 0.55	0.82
Fat (% of TEI)	30.68 ± 1.65	30.49 ± 1.36	29.85 ± 1.76	0.87
Saturated fatty acid (g/day)	14.32 ± 0.94	16.28 ± 0.77	16.34 ± 1.01	0.07
Cholesterol (g/day)	182.03 ± 17.51	221.18 ± 14.44	248.56 ± 18.75	**0.03**
Fiber (g/1000 kcal)	8.97 ± 3.35	8.04 ± 4.17	7.06 ± 3.71	**0.04**
MUFA	28.69 ± 2.71	25.81 ± 2.24	24.86 ± 2.92	**0.04**
PUFA	15.80 ± 1.03	17.79 ± 0.84	17.99 ± 1.10	0.07
Energy intake in breakfast (% of TEI)	26.22 ± 1.20	25.94 ± 0.99	25.80 ± 1.28	0.66
Energy intake in lunch (% of TEI)	33.99 ± 1.30	34.02 ± 1.07	33.33 ± 1.39	0.71
Energy intake in afternoon (% of TEI)	11.60 ± 1.27	11.64 ± 1.05	12.78 ± 1.36	0.64
Energy intake in dinner (% of TEI)	26.74 ± 1.24	28.49 ± 1.02	28.93 ± 1.33	0.29
Number of EOs (n/day)	6.32 ± 0.13	6.40 ± 0.11	6.44 ± 0.14	0.58
	Breakfast-EDII		***[0.28 (0.04, 0.65)]*[1]**	***P*-value**
	T1 ≥ −1.77	***−1.56 < T2 < 2.02***	***T3 ≥ 2.03***	
Total energy intake (TEI) (kcal)	1663.88 ± 358.16	1660.56 ± 390.22	1674,79 ± 406.72	0.93
Carbohydrate (% of TEI)	59.27 ± 18.82	57.04 ± 9.30	60.02 ± 23.81	0.23
Protein (% of TEI)	14.92 ± 2.46	13.62 ± 2.81	13.09 ± 3.54	**0.03**
Fat (% of TEI)	30.72 ± 6.95	30.34 ± 9.20	30.65 ± 9.20	0.88
Saturated fatty acid (g/day)	14.32 ± 0.94	16.28 ± 0.77	16.34 ± 1.01	0.09
Cholesterol (g/day)	209.07 ± 17.42	216.22 ± 14.31	240.32 ± 17.88	0.11
Fiber (g/1000 kcal)	9.06 ± 3.35	8.11 ± 4.17	6.86 ± 3.71	**0.03**
MUFA	27.60 ± 2.55	26.81 ± 2.28	25.86 ± 2.90	0.09
PUFA	16.80 ± 1.12	16.99 ± 0.91	18.09 ± 1.17	0.12
Energy intake in breakfast (% of TEI)	26.65 ± 5.76	25.39 ± 7.07	24.01 ± 8.22	**0.01**
Energy intake in lunch (% of TEI)	32.95 ± 7.29	31.44 ± 7.93	30.77 ± 7.70	**0.02**
Energy intake in afternoon (% of TEI)	12.31 ± 6.61	13.41 ± 8.22	12.91 ± 7.41	0.37
Energy intake in dinner (% of TEI)	29.01 ± 7.13	29.41 ± 7.40	29.33 ± 7.84	0.89
Number of EOs (n/day)	6.26 ± 0.88	6.32 ± 0.93	6.30 ± 0.88	0.81
	Lunch-EDII		***[0.29 (−0.14, 0.69)]*[1]**	
	T1 ≤ −1.66	***−1.65 < T2 < 2.13***	***T3 ≥ 2.14***	
Total energy intake (TEI) (kcal)	1680.01 ± 457.62	1632.75 ± 329.13	1695.69 ± 354.90	0.24
Carbohydrate (% of TEI)	56.82 ± 9.73	58.47 ± 8.69	61.02 ± 28.90	0.11
Protein (% of TEI)	13.90 ± 3.02	13.90 ± 2.42	14.03 ± 3.74	0.63
Fat (% of TEI)	30.30 ± 6.58	30.33 ± 6.11	31.08 ± 9.72	0.57
Saturated fatty acid (g/day)	14.02 ± 0.85	15.29 ± 0.87	16.74 ± 1.32	**0.04**
Cholesterol (g/day)	198.26 ± 17.31	226.28 ± 14.55	241.30 ± 17.68	0.09
Fiber (g/1000 kcal)	8.76 ± 3.45	8.19 ± 4.22	6.82 ± 3.68	**0.03**
MUFA	27.60 ± 2.55	26.81 ± 2.28	25.86 ± 2.90	**0.03**
PUFA	16.53 ± 1.09	16.95 ± 087	17.19 ± 1.37	0.08
Energy intake in breakfast (% of TEI)	25.69 ± 6.75	25.61 ± 6.72	25.44 ± 7.89	0.80
Energy intake in lunch (% of TEI)	31.63 ± 8.36	30.83 ± 6.93	32.66 ± 7.74	**0.03**
Energy intake in afternoon (% of TEI)	12.83 ± 7,16	12.68 ± 7.73	13.08 ± 7.75	0.87
Energy intake in dinner (% of TEI)	28.97 ± 8.39	30.39 ± 6.78	29.09 ± 7.01	0.08
Number of EOs (n/day)	6.35 ± 0.91	6.32 ± 0.91	6.20 ± 0.94	0.24
	Afternoon snack-EDII		***[0.64 (0.21, 1.20)]*[1]**	
	T1 ≥ −1.20	***−1.19 < T2 < 3.44***	***T3 ≥ 3.45***	
Total energy intake (TEI) (kcal)	1627.29 ± 490.11	1630.89 ± 362.93	1741.19 ± 310.01	**0.01**

Table 3 (continued)

	Afternoon snack-EDII		***[0.64 (0.21, 1.20)]*[1]**	
	T1 ≥ −1.20	***−1.19 < T2 < 3.44***	***T3 ≥ 3.45***	
Carbohydrate (% of TEI)	58.18 ± 5.62	59.57 ± 11.91	59.56 ± 15.83	0.47
Protein (% of TEI)	13.06 ± 2.60	14.30 ± 3.32	13.78 ± 3.02	0.06
Fat (% of TEI)	28.40 ± 6.02	30.17 ± 8.01	31.82 ± 7.54	**0.04**
Saturated fatty acid (g/day)	14.33 ± 0.89	15.09 ± 0.93	16.64 ± 1.28	0.09
Cholesterol (g/day)	189.15 ± 17.36	221.18 ± 14.05	243.50 ± 17.38	**0.04**
Fiber (g/1000 kcal)	8.36 ± 3.65	8.19 ± 4.52	7.82 ± 3.08	0.07
MUFA	27.89 ± 2.51	26.24 ± 2.29	25.76 ± 2.80	**0.04**
PUFA	16.53 ± 1.09	16.95 ± 087	17.19 ± 1.37	0.11
Energy intake in breakfast (% of TEI)	25.95 ± 6.79	25.48 ± 7.17	25.31 ± 7.25	0.49
Energy intake in lunch (% of TEI)	32.07 ± 7.09	31.76 ± 7.73	31.29 ± 8.28	0.59
Energy intake in afternoon (% of TEI)	12.64 ± 6.81	12.91 ± 7.44	13.04 ± 6.87	0.64
Energy intake in dinner (% of TEI)	28.11 ± 6.65	29.54 ± 7.22	30.32 ± 8.27	**0.02**
Number of EOs (n/day)	6.05 ± 0.93	6.14 ± 0.87	6.30 ± 0.90	0.24
	Dinner-EDII		***[0.39 (−0.03, 0.78)]*[1]**	
	T1 ≥ −1.50	***−1.49 < T2 < 2.23***	***T3 ≥ 2.24***	
Total energy intake (TEI) (kcal)	1662.66 ± 444.56	1665.39 ± 320.19	1670.61 ± 382	0.72
Carbohydrate (% of TEI)	56.95 ± 11.30	59.86 ± 20.12	59.95 ± 21.30	0.08
Protein (% of TEI)	13.86 ± 3.91	13.95 ± 2.24	13.95 ± 2.58	0.93
Fat (% of TEI)	30.66 ± 9.92	30.62 ± 6.09	30.42 ± 6.33	0.89
Saturated fatty acid (g/day)	13.82 ± 0.93	15.17 ± 0.89	16.59 ± 1.10	0.06
Cholesterol (g/day)	201.26 ± 17.22	220.28 ± 14.64	242.30 ± 17.66	**0.04**
Fiber (g/1000 kcal)	8.69 ± 3.33	8.04 ± 4.38	6.72 ± 3.45	0.06
MUFA	27.70 ± 2.55	26.52 ± 2.30	25.76 ± 2.94	**0.03**
PUFA	16.44 ± 1.10	16.85 ± 0.86	17.32 ± 1.39	0.06
Energy intake in breakfast (% of TEI)	26.87 ± 6.91	25.11 ± 6.92	24.89 ± 7.53	**0.04**
Energy intake in lunch (% of TEI)	31.61 ± 7.42	32.54 ± 7.57	30.95 ± 8.07	0.14
Energy intake in afternoon (% of TEI)	12.49 ± 6.81	13.13 ± 7.44	13.01 ± 8.29	0.68
Energy intake in dinner (% of TEI)	28.44 ± 7.68	28.76 ± 7.62	30.75 ± 6.87	**0.02**
Number of EOs (n/day)	6.31 ± 0.93	6.32 ± 0.90	6.15 ± 0.94	0.36

ANCOVA was used to compare means across tertiles of the energy-adjusted dietary inflammatory index (EDII)

adjusting for age, socioeconomic status, physical activity, education level, marital status, menopausal status, smoking status, chronotype, sleep duration, BMI, and total energy intake (except for TEI, which was not adjusted)

Significant p-values are presented in bold

Abbreviations: *EDII* Energy-adjusted dietary inflammatory index, *T* Tertile, *TEI* Total energy intake, *BMI* Body mass index, *EOs* Eating occasions

[1] Mean (95% confidence interval)

had higher mean EDII values for all meals. The beta coefficient for meal-specific EDII increased most for Afternoon Snack [β (95%CI), 4.78 (3.66–5.89)], followed by Dinner [4.43 (4.40–5.45)], Lunch [4.86 (3.94–5.79)], and Breakfast [2.41 (0.66–4.15)] in Model 2. While Breakfast-EDII was significantly associated with Habitual-EDII only in T3 compared to T1, other meals showed significant associations across all tertiles in all models.

Habitual- and meal-specific EDII in associations with cardiometabolic health

The associations between meal-specific EDII and each 10-point increase in Habitual-EDII are detailed in Table 5. The results indicated that meal-specific EDII values significantly increased with higher Habitual-EDII scores. Among meals, afternoon snack-EDII exhibited the strongest relationship, followed by dinner-, lunch-, and breakfast-EDII. Both crude and fully adjusted models showed significant and increasing beta coefficients. Afternoon Snack-EDII showed the strongest association with each 10-point increase in Habitual-EDII [β (95% CI); 3.77 (3.34–4.20)], followed by Dinner-EDII [2.37 (1.97–2.76)], Lunch-EDII [2.13 (1.77–2.50)], and Breakfast-EDII [1.76 (1.36–2.09)], respectively.

Table 6 presents the associations between Habitual EDII tertiles and cardiometabolic health indicators, with odds ratios [ORs (95% CIs)] estimated via logistic regression. In the fully adjusted model, compared with participants in T1 (reference), those in T3 exhibited significantly greater odds of overweight/obesity (BMI ≥ 25 kg/m^2) [2.38 (1.51–3.81); pFDR 0.006]. Habitual-EDII was significantly related to VAI [1.92 (1.21–3.03); pFDR

Table 4 Relationship and contribution of meal-specific energy adjusted dietary inflammatory index (EDII) in tertiles of habitual-EDII, beta and 95% confidence intervals (CIs), *n* = 562

Variables	Energy adjusted habitual dietary inflammatory index (Habitual-EDII), β (95% CI)			R2	*P*-trend[1]	Every 10 point of Habitual-EDII
	T1 ≥ −2.42	−2.42 < T2 < 6.06	T3 ≥ 6.06			
Subject	187	188	187			
Breakfast-EDII[2]						
Mean (95%CIs)[3]	−1.42 (−2.96–0.04)	0.08 (−1.44–1.62)	1.93 (1.06–4.17)			
Crud	Ref.	0.52 (−0.60–1.66)	**2.45 (0.72–4.19)**	0.19	0.96	1.73 (1.36–2.04)
Model1	Ref.	0.68 (−0.45–1.83)	**2.50 (0.75–4.21)**	0.20	0.97	1.70 (1.36–2.05)
Model2	Ref.	0.67 (−0.46–1.81)	**2.41 (0.66–4.15)**	0.20	0.99	1.63 (1.28–1.98)
Lunch-EDII[2]						
Mean (95%CIs)[3]	−1.72 (−3.7–0.29)	−1.09 (−2.75–0.57)	1.76 (−0.23–3.12)			
Crud	Ref.	**2.09 (1.20–2.92)**	**4.78 (3.73–5.54)**	0.24	0.90	2.31 (1.76–2.49)
Model1	Ref.	**2.22 (1.31–3.12)**	**4.78 (3.87–5.68)**	0.24	0.91	2.17 (1.81–2.53)
Model2	Ref.	**2.24 (1.34–3.15)**	**4.86 (3.94–5.79)**	0.25	0.91	2.22 (1.84–2.59)
Afternoon snack-EDII[2]						
Mean (95%CIs)[3]	−2.36 (−2.90 - −1.73)	2.02 (0.96–4.08)	4.21 (3.11–7.31)			
Crud	Ref.	**4.96 (3.86–6.07)**	**8.03 (6.91–9.14)**	0.35	0.94	3.80 (3.37–4.23)
Model1	Ref.	**8.01 (6.88–9.12)**	**4.81 (3.69–5.92)**	0.36	0.96	3.77 (3.34–4.20)
Model2	Ref.	**7.91 (6.76–9.06)**	**4.78 (3.66–5.89)**	0.36	0.97	3.76 (3.32–4.26)
Dinner-EDII[2]						
Mean (95%CIs)[3]	−1.54 (−3.35–0.25)	0.90 (−0.94–2.74)	2.89 (1.01–4.77)			
Crud	Ref.	**2.51 (1.53–3.50)**	**4.50 (3.50–5.49)**	0.28	0.86	2.35 (1.96–2.74)
Model1	Ref.	**2.42 (1.43–3.41)**	**4.41 (3.41–5.40)**	0.28	0.87	2.34 (1.95–2.72)
Model2	Ref.	**2.43 (1.43–3.42)**	**4.43 (4.40–5.45)**	0.29	0.89	2.37 (1.98–2.77)

Model 1: Adjusted for age, energy intake, physical activity, marital status, socio-economic status (SES), smoking status, education level, menopausal status, chronotype, sleep duration, number of EOs, and supplement intake

Model 2: Further adjusted for body mass index (BMI)

Abbreviations: *T* Tertile, *CI*, Confidence interval, *EDII* Energy-adjusted dietary inflammatory index, *BMI* Body mass index, *EOs* Eating occasions

[1] Obtained from linear regression

[2] beta (β) of meal-specific of EDII are reported as the dependent variable

[3] Mean are adjusted for covariates included in Model 2

Table 5 The relationship between each 10-point of EDII and meal-specific EDII, *n* = 562

Meal-specific EDII	10-point of Habitual-EDII			
	Beta [1]	CIs	R2	P_{FDR} [2]
Breakfast-EDII				
Crud	1.69	1.37–2.006	0.16	**< 0.001**
Adjusted[3]	1.76	1.36–2.09	0.19	**< 0.001**
Lunch-EDII				
Crud	2.20	1.87–2.53	0.23	**< 0.001**
Adjusted[3]	2.13	1.77–2.50	0.24	**< 0.001**
Afternoon snack-EDII				
Crud	3.60	3.24–4.05	0.33	**< 0.001**
Adjusted[3]	3.77	3.34–4.20	0.35	**< 0.001**
Dinner-EDII				
Crud	2.45	2.09–2.81	0.26	**< 0.001**
Adjusted[3]	2.37	1.97–2.76	0.29	**< 0.001**

Abbreviations: *CI* Confidence interval, *FDR* False discovery rate, *EDII* Energy-adjusted dietary inflammatory index, *BMI* Body mass index, *EOs* Eating occasions

[1]obtained[1]from linear regression

[2]Significant p-values are presented in bold

[3]Adjusting for age, socioeconomic status, physical activity, education level, marital status, menopausal status, smoking status, chronotype, sleep duration, number of EOs, BMI, and total energy intake

0.01]. Habitual-EDII were linked to an increased risk of CMI in T3 vs. T1 [1.95 (1.23–3.04), pFDR = 0.008], and API in T3 vs. T1 [2.53 (1.57–4.08), pFDR = 0.006]. Every 10-point increase of Habitual-EDII was related to an increased BMI [1.42 (1.17–1.72)], VAI [1.28 (1.06–1.54)], CMI [1.27 (1.06–1.54)], and API [1.40 (1.15–1.71)] in the adjusted model, supporting a positive association between pro-inflammatory diets and poorer metabolic health.

Afternoon snack-EDII was significantly associated with an elevated BMI [1.93 (1.21–3.11), pFDR = 0.02] and each 10-point increase in Afternoon snack-EDII was associated with a greater BMI [1.54 (1.12–2.11)] in adjusted models. Furthermore, Dinner-EDII was associated with greater VAI in T3 vs. T1 [1.85 (1.17–2.92), pFDR = 0.01] and CMI, T3 vs. T1 [1.77 (1.12–2.80), pFDR = 0.03]. Whilst every 10-point increase in Dinner-EDII was associated to elevated VAI [1.94 (1.31–2.87)] and CMI [1.81 (1.22–2.68)] in the adjusted model (Table 7).

Table 6 Association of energy adjusted habitual dietary inflammatory index (EDII) and cardiometabolic health in Iranian women

Variables	*Energy adjusted habitual dietary inflammatory index (Habitual EDII)*					
	T1 ≥ −2.42	**−2.42 < T2 < 6.06 OR (95%CI)**	***P*-$_{FDR}$[1]**	**T3 ≥ 6.06 OR (95%CI)**	***P*-$_{FDR}$[1]**	**Every 10 point of habitual EDII OR (95%CI)**
Subject	187	188		187		
BMI						
(BMI < 25 Ref.)						
Crud	Ref.	1.21 (0.85–1.91)	0.33	**2.27 (1.50–3.51)**	**0.006**	**1.36 (1.15–1.65)**
Adjusted[3]	Ref.	**1.16 (1.02–1.43)**	0.04	**2.38 (1.51–3.81)**	**0.006**	**1.42 (1.17–1.72)**
VAI						
(VAI < 4.28 Ref.)						
Crud	Ref.	1.29 (0.83–1.99)	0.32	**1.81 (1.16–2.83)**	**0.01**	**1.24 (1.03–1.49)**
Adjusted[3]	Ref.	1.33 (0.85–2.09)	0.37	**1.92 (1.21–3.03)**	**0.01**	**1.28 (1.06–1.54)**
CMI						
(CMI < 0.55)						
Crud	Ref.	1.32 (0.87–2.09)	0.33	**1.94 (1.25–3.04)**	**0.006**	**1.45 (1.06–1.53)**
Adjusted[3]	Ref.	1.36 (0.87–2.12)	0.31	**1.95 (1.23–3.04)**	**0.008**	**1.27 (1.06 1.54)**
API						
(API < 0.11)						
Crud	Ref.	1.13 (0.70–1.83)	0.77	**2.45 (1.54–3.91)**	**0.006**	**1.38 (1.14–1.68)**
Adjusted[3]	Ref.	1.14 (0.70–1.88)	0.65	**2.53 (1.57–4.08)**	**0.006**	**1.40 (1.15–1.71)**

Abbreviations: *CI* Confidence interval, *EDII* Energy-adjusted dietary inflammatory index, *FDR* False discovery rate, *BMI* Body mass index, *EOs* Eating occasions, *VAI* Visceral adiposity index, *CMI* Cardiometabolic index, *API* Atherogenic index of plasma

[1] Obtained from Generalized linear regression (GLR). OR (95% CI) is reported

[2] Adjusted for age, energy intake, physical activity, marital status, socio-economic status, education, smoking status, chronotype, sleep duration, number of EOs, supplement intake and body mass index (BMI) exception for when outcome was BMI

[3] Significant p-$_{FDR}$ are presented in bold

Sensitivity analysing

Interaction by age (< 40 vs. ≥40 years) was examined for all associations. Accordingly, no significant interaction was detected (Table S3, Table S3 presents the interaction by age in the association between EDII and cardiometabolic health).

Discussion

In this study, we found that greater overall dietary inflammatory potential, as indicated by higher Habitual-E-DII scores, was significantly associated with greater obesity (BMI) and atherogenic indices (CMI and API). Among individual meals, afternoon snacks and dinner contributed most strongly to the Habitual-E-DII, followed by lunch and breakfast. In meal-specific analyses, a higher inflammatory potential of afternoon snacks was associated with increased obesity risk, while dinner E-DII was linked to elevated VAI and CMI. These findings suggest that consuming pro-inflammatory foods overall, especially later in the day, may disproportionately contribute to increased adiposity and adverse cardiometabolic risk profiles.

Regarding dietary patterns, our data align with previous research in Iran [5, 7, 31] and internationally [6, 39]. Indeed, we showed that pro-inflammatory diets are typically characterized by higher intake of refined grains, sugars, red and processed meats, and lower consumption of fiber-rich foods such as vegetables, legumes, and healthy oils. Similarly, Meneguelli et al., in a cross-sectional study of Brazilian adults and the elderly participating in a cardiovascular health program, examined the association between DII scores, cardiometabolic risk factors, and dietary patterns. The authors indicated that a pro-inflammatory diet was associated with higher obesity risk and poorer overall dietary quality [39]. In our analysis, individuals in the highest tertile of day- or meal-specific E-DII consumed less fiber and MUFA, and more saturated fats, reflecting a suboptimal nutrient profile. Notably, those with the highest dinner E-DII scores also had significantly higher energy intake at dinner, suggesting a possible link between pro-inflammatory evening eating patterns and energy overconsumption. Additionally, we found that participants in the highest E-DII tertile in the afternoon and at dinner had significantly higher contributions to Habitual-EDII. Previously, dinner has been reported to elicit the greatest contribution to habitual dietary patterns in both German [26]and Iranian [21] adults, followed by lunch. However, in Japanese adults, breakfast showed the highest contribution, followed by lunch and dinner [40]. These differences in meal contributions may be explained by cultural and behavioral variations in dietary habits, such as meal timing, composition, and portion sizes. For example, the traditional Japanese diet emphasizes nutrient-dense breakfasts,

Table 7 Association of meal-specific energy adjusted dietary inflammatory index (EDII) and cardiometabolic health in Iranian women, *n* = 562

	Breakfast-EDII						***Lunch-EDII***					
	T1 ≥ - 1.58	***-1.58 < T2 < 2.03OR (95%CI)***	***P_{-FDR}[1]***	***T3 ≥ 2.03 OR (95%CI)***	***P_{-FDR}[1]***	***Every 10 point of Breakfast-EDII***	***T1 ≥ - 1.67***	***-1.66 < T2 < 2.13 OR (95%CI)***	***P_{-FDR}[1]***	***T1 ≥ 2.13 OR (95%CI)***	***P_{-FDR}[1]***	***Every 10 point of Lunch-EDII***
Subjects	*187*	*188*		*187*			*187*	*188*		*187*		
BMI												
(BMI<25 Ref.)												
Crud	Ref.	0.92 (0.59 – 1.42)	0.72	1.72 (1.10 – 2.72)	0.11	1.51 (0.97 – 2.35)	Ref.	1.37 (0.88 – 2.13)	065	1.31 (0.85 – 2.03)	0.66	1.36 (0.91 – 2.02)
Adjusted3	Ref.	0.95 (0.60 – 1.49)	0.83	1.79 (1.12 – 2.86)	0.11	1.49 (0.94 – 2.36)	Ref.	1.33 (0.84 – 2.09)	0.61	1.28 (0.82 – 2.01)	0.67	1.31 (0.87 – 1.97)
VAI												
(VAI < 4.28 Ref.)												
Crud	Ref.	1.18 (0.76 – 1.82)	0.65	1.16 (0.74 – 1.80)	0.75	1.05 (0.68 – 1.61)	Ref.	1.20 (0.77 – 1.86)	0.60	0.96 (0.62 – 1.49)	0.88	1.04 (0.68 – 1.50)
Adjusted3	Ref.	1.29 (0.82 – 2.02)	0.46	1.11 (0.78 – 1.76)	0.68	0.97 (0.62 – 1.51)	Ref.	1.13 (0.72 – 1.77)	0.68	0.92 (0.59 – 1.43)	0.72	0.96 (0.64 – 1.43)
CMI												
(CMI < 0.55)												
Crud	Ref.	1.01 (0.65 – 1.57)	0.94	1.12 (0.72 – 1.74)	0.67	0.99 (0.64 – 1.53)	Ref.	1.04 (0.67 – 1.61)	0.83	1.25 (0.81 – 1.94)	0.61	1.09 (0.73 – 1.61)
Adjusted3	Ref.	1.10 (0.70 – 1.74)	0.65	1.07 (0.67 – 1.69)	0.79	0.91 (0.58 – 1.42)	Ref.	1.17 (0.74 – 1.83)	0.69	0.99 (0.63 – 1.55)	0.98	1.04 (0.69 – 1.56)
API												
(API <0.11)												
Crud	Ref.	1.31 (0.82 – 2.07)	0.45	1.38 (0.86 – 2.19)	0.28	1.23 (0.78 – 1.92)	Ref.	1.14 (0.72 – 1.79)	0.67	1.16 (0.73 – 1.82)	0.62	1.23 (0.82 – 1.97)
Adjusted3	Ref.	1.33 (0.82 – 2.14)	0.40	1.25 (0.77 – 2.03)	0.45	1.09 (0.68 – 1.74)	Ref.	1.09 (0.68 – 1.75)	0.70	1.10 (0.68 – 1.75)	0.69	1.17 (0.67 – 1.78)
	Afternoon snack-EDII						**Dinner-EDII**					
	T1 ≥ - 1.20	**-1.20 < T2 < 3.44 OR (95%CI)**	**P_{-FDR}[1]**	**T1 ≥ 3.44 OR (95%CI)**	**P_{-FDR}[1]**	**Ever y10 point of Afternoon snack -EDII**	**T1 ≥ - 1.50**	**-1.50 < T2 < 2.30 OR (95%CI)**	**P_{-FDR}[1]**	**T1 ≥ 2.30 OR (95%CI)**	**P_{-FDR}[1]**	**Every 10 point of Dinner-EDII**
Subjects	187	188		187			187	188		187		
BMI												
(BMI<25 Ref.)												
Crud	Ref.	1.27 (0.82 – 1.96)	0.38	1.79 (1.14 – 2.81)	**0.04**	**1.43 (1.06 – 1.93)**	Ref.	1.19 (0.77 – 1.87)	0.32	1.45 (0.93 – 2.28)	0.10	1.22 (0.84 – 1.77)
Adjusted3	Ref.	1.34 (0.84 – 2.14)	0.31	1.93 (1.21 – 3.11)	**0.02**	**1.54 (1.12 – 2.11)**	Ref.	1.18 (0.76 – 1.86)	0.34	1.57 (0.99 – 2.49)	0.11	1.34 (0.91 – 1.97)
VAI												

Table 7 (continued)

	Afternoon snack-EDII						Dinner-EDII					
	T1 ≥ - 1.20	**-1.20 < T2 < 3.44 OR (95%CI)**	**P_{-FDR}[1]**	**T1 ≥ 3.44 OR (95%CI)**	**P_{-FDR}[1]**	**Ever y10 point of Afternoon snack -EDII**	**T1 ≥ - 1.50**	**-1.50 < T2 < 2.30 OR (95%CI)**	**P_{-FDR}[1]**	**T1 ≥ 2.30 OR (95%CI)**	**P_{-FDR}[1]**	**Every 10 point of Dinner-EDII**
(VAI < 4.28 Ref.)												
Crud	Ref.	1.02 (0.66 – 1.58)	0.62	1.24 (0.80 – 1.94)	0.32	1.13 (0.84 – 1.54)	Ref.	1.47 (0.95 – 2.26)	0.21	1.84 (1.18 – 2.86)	**0.01**	**1.89 (1.29 – 2.77)**
Adjusted3	Ref.	1.09 (0.69 – 1.72)	0.61	1.27 (0.80 – 2.02)	0.30	1.15 (0.85 – 1.57)	Ref.	1.45 (0.93 – 2.25)	0.22	1.85 (1.17 – 2.92)	**0.01**	**1.94 (1.31 – 2.87)**
CMI												
(CMI < 0.55)												
Crud	Ref.	1.15 (0.74 – 1.78)	0.42	1.33 (0.85 – 2.76)	0.30	1.23 (0.91 – 1.65)	Ref.	1.45 (0.94 – 2.24)	0.23	1.81 (1.16 – 2.81)	**0.03**	**1.81 (1.24 – 2.66)**
Adjusted3	Ref.	1.18 (0.74 – 1.87)	0.48	1.31 (0.82 – 2.08)	0.36	1.20 (0.87 – 1.63)	Ref.	1.38 (0.88 – 2.17)	0.24	1.77 (1.12 – 2.80)	**0.03**	**1.81 (1.22 – 2.68)**
API												
(API <0.11)												
Crud	Ref.	1.11 (0.72 – 1.85)	0.34	1.59 (1.004 – 2.54)	0.10	**1.44 (1.05 – 1.99)**	Ref.	1.16 (0.73 – 1.84)	0.53	1.58 (1.003 – 2.51)	0.09	**1.51 (1.03 – 2.23)**
Adjusted3	Ref.	1.21 (0.73 – 1.98)	0.45	1.58 (0.97 – 2.56)	0.11	**1.44 (1.03 – 2.01)**	Ref.	1.08 (0.67 – 1.74)	0.53	1.53 (0.95 – 1.74)	0.10	**1.49 (1.004 – 2.24)**

Abbreviations: *CI* Confidence interval, *EDII* Energy-adjusted dietary inflammatory index, *FDR* False discovery rate, *BMI* Body mass index, *EOs* Eating occasions, *VAI* Visceral adiposity index, *CMI* Cardiometabolic index, *API* Atherogenic index of plasma

[1] Obtained from Generalized linear regression (GLR). OR (95% CI) is reported

[2] Adjusted for age, energy intake, physical activity, marital status, socio-economic status, education, smoking status, chronotype, sleep duration, number of EOs, supplement intake and body mass index (BMI) exception for when outcome was BMI

[3] Significant p_{FDR} are presented in bold

while Western and Middle Eastern diets tend to concentrate energy and pro-inflammatory components later in the day.

Consistent with the results of the present study regarding the associations between Habitual-EDII, CMI, and AIP, some previous studies have also reported significant associations between higher DII scores and increased cardiometabolic risk. However, the specific composite biomarkers utilised in the present study, CMI and AIP, were not assessed in earlier studies. Higher DII scores have been consistently associated with increased cardiometabolic risk [41]. For instance, Mazidi et al. demonstrated that higher energy-adjusted DII scores were significantly related to greater odds of metabolic syndrome (MetS), obesity, and elevated lipid profile in a large U.S. population. Supporting these results, a study conducted among 90 overweight, sedentary adults in Bogotá, Colombia, showed that a lower DII score was associated with better cardiometabolic health, including higher HDL-C levels, enhanced endothelial function, lower triglycerides, and reduced arterial stiffness [42]. Moreover, a systematic review and dose–response meta-analysis of observational studies confirmed that a higher DII score, indicating a more pro-inflammatory diet, was significantly associated with increased risk of cardiometabolic diseases (CMDs), CMD-related mortality, MetS, and hypertension [43]. Notably, individuals in the highest DII category had a 29% greater risk of CMD-related mortality compared to those in the lowest category [43]. While most studies support these associations, some discrepancies exist [5, 8]. For instance, a previous cross-sectional study of 404 Iranian adults did not find a significant association between DII and MetS; however, the authors did report that higher DII scores were significantly associated with lower levels of HDL-C [5]. In contrast, Haji-Hosseini-Gazestani et al. found no significant association between DII and BMI among Iranian women [44], while Shahinfar et al. (2021) reported an inverse association in unadjusted analyses that lost significance after adjustment [45]. Collectively, these findings underscore the detrimental effects of pro-inflammatory diets and highlight the potential protective role of anti-inflammatory dietary patterns in cardiometabolic health.

Despite the novelty of our findings linking afternoon and dinner-specific EDII scores to obesity and cardiometabolic markers, only a limited number of studies have explored the association between meal-specific dietary inflammation and health outcomes. One Iranian study reported no significant association between Food groups-DII scores and waist-related indices after adjustment [8]. In contrast, analyses of U.S. NHANES data showed that pro-inflammatory diets and breakfast skipping were positively associated with obesity, with dietary inflammation partially mediating this relationship, especially in people with diabetes [46]. Evidence also supports the assertion that irregular meal timing and higher evening energy intake lead to an increased cardiometabolic burden [47, 48]. Pot et al. found that inconsistent energy intake, particularly during breakfast and between meals, was associated with a higher risk of metabolic syndrome [47]. Furthermore, consumption of a large portion of daily calories in the evening has been associated with an increased risk of obesity, MetS, and non-alcoholic fatty liver disease [49].

Mechanistically, diets high in vegetables, legumes, seeds, and healthy oils have been associated with lower inflammation and improved metabolic health due to their abundant content of fiber, flavonoids, and unsaturated fats, which foster a favorable gut microbiota, regulate appetite hormones and suppress pro-inflammatory pathways [50–52]. In contrast, red and processed meats, which are rich in saturated fats and pro-oxidants, are known to increase systemic inflammation and metabolic dysregulation [53, 54]. Timing may further modulate these effects; indeed, evening consumption has been associated with obesity [55] and metabolic dysfunction, partly due to lower nocturnal energy expenditure and impaired glucose tolerance [56, 57]. Moreover, obesity, particularly visceral adiposity, is closely related to insulin resistance, disrupted glucose homeostasis [58], and greater inflammatory responses, likely mediated by circadian misalignment and disrupted expression of core clock proteins like PER2 [59, 60]. Thus, late eating may disrupt PER2, increasing obesity risk and metabolic disturbances [61]. Such misalignment may amplify the metabolic consequences of poor dietary quality in the evening.

Overall, our findings underscore the combined influence of pro-inflammatory dietary patterns and meal timing in shaping cardiometabolic risk. Although the results provide important insight into meal-specific dietary inflammation, their generalisability is limited by the fact that the study was conducted among adult women from a specific urban region in southern Tehran. Longitudinal and interventional studies are warranted to confirm these associations and to further elucidate the physiological mechanisms linking dietary inflammation, chrono-nutrition, and metabolic health.

Strength and limitations

This study has several limitations that warrant consideration. First, its cross-sectional design precludes causal inferences being drawn; although reverse causality cannot be entirely ruled out, it is more plausible that pro-inflammatory dietary patterns contribute to adverse cardiometabolic outcomes rather than the reverse. Second, dietary intake was assessed using 24-hour dietary recalls, a short-term method subject to self-reporting bias and measurement error. However, compared to food

frequency questionnaires, 24-hour recalls offer more precise information on food types, timing and quantities [62] and are well suited for meal-specific analyses [63]. Finally, although the results provide valuable insight into meal-specific dietary inflammation, their generalisability is limited by the fact that the study sample consisted solely of adult women from a specific urban area in southern Tehran, which may not reflect dietary patterns or metabolic characteristics in other populations. Despite these limitations, this study has notable strengths. Indeed, to our knowledge, it is the first to evaluate both daily and meal-specific E-DII in relation to cardiometabolic indices among Iranian women. Furthermore, the analyses accounted for chronotype and diurnal preference, which are important confounding factors often overlooked in similar research. Finally, we utilised a robust DAG-based approach to model selection, thereby reducing the likelihood of erroneous retention of variables.

Conclusion

In summary, this study demonstrates that higher dietary inflammatory potential, both at the daily level and in afternoon snacks and dinner, is significantly associated with increased obesity and adverse cardiometabolic indices among Iranian women. These findings highlight the importance of not only the quality of dietary intake, but also the timing of pro-inflammatory food consumption in association with metabolic health. The present results support the growing evidence that dietary inflammation and circadian misalignment may interact to exacerbate cardiometabolic risk. Promoting anti-inflammatory, plant-based dietary patterns and mindful timing of eating may offer a practical strategy to reduce inflammation and improve cardiometabolic outcomes. Further research is warranted to clarify causal pathways and the role of meal timing in metabolic risk reduction.

Abbreviations

ANOVA	Analysis of variance
ANCOVA	Analysis of covariance
API	Atherogenic index of plasma
BMI	Body mass index
BMR	Basal metabolic rate
CIM	Cardiometabolic index
DAG	Directed acyclic graph
DII	Dietary inflammatory index
EI	Energy intake
E-DII	Energy-adjusted
EOs	Eating occasions
FDR	False discovery rate
HDL	High-density lipoprotein cholesterol
IPAQ	International physical activity questionnaire, GLM, generalized linear regression
LDL	Low-density lipoprotein cholesterol
MEQ	Morningness-eveningness questionnaire
MET/min	Metabolic equivalent of task per minute
MUFA	Monounsaturated fatty acids
PCA	Principal component analysis
PUFA	Polyunsaturated fatty acids
SES	Socioeconomic status
SFA	Saturated fatty acid
TEI	Total energy intake
TG	Triglyceride
TC	Total cholesterol
VAI	Visceral adiposity index
WC	Waist circumference
WHO	World health organization
WHR	Waist circumference to hip circumference ratio
WHtR	Waist-to-height ratio

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12937-026-01280-3.

Supplementary Material 1

Supplementary Material 2

Supplementary Material 3

Supplementary Material 4

Acknowledgements

We sincerely thank all the participants for their time, effort, and invaluable contribution to this study. We also would like to express our gratitude to Islamic Azad University, Tehran Medical Sciences—Pharmacy and Pharmaceutical Sciences Faculty, for their generous funding and support, which made this research possible.

Authors' contributions

A.L. and M.M. jointly conceived and designed the research. Both authors contributed to data acquisition, analysis, and interpretation. A.L. drafted the manuscript, while M.M., E.D., M.C.M., C.C., H.Z., and S.V critically reviewed and revised the content. M.M. and A.L assumed equal responsibility for the integrity and accuracy of the work. Authors have read and approved the final version of the manuscript.

Funding

This study was funded by the Islamic Azad University of Medical Sciences, Tehran (Grant Number: 39052).

Data availability

The data are available from the corresponding author upon reasonable request.

Declarations

Ethics approval and consent to participate

This study was approved by the Research Ethics Committee of Islamic Azad University, Tehran Medical Sciences—Pharmacy and Pharmaceutical Sciences Faculty (IR.IAU.REC.1403.487). Also, written informed consent was obtained from all participants.

Consent for publication

All authors have reviewed and approved the final version of this manuscript and consent to its publication.

Competing interests

The authors declare no competing interests.

Author details

[1]Department of Community Nutrition, School of Nutritional Sciences and Dietetics, Tehran University of Medical Sciences (TUMS), Tehran, Iran
[2]Endocrinology and Metabolism Research Center, Endocrinology and Metabolism Clinical Sciences Institute, Tehran University of Medical Sciences, Tehran, Iran
[3]Non-Communicable Diseases Research Center, Alborz University of Medical Sciences, Karaj, Iran
[4]Department of Laboratory Medicine, University Clinical Hospital of Santiago de Compostela, Santiago de Compostela, Spain

[5]Research Methodology Group, Health Research Institute of Santiago de Compostela (IDIS), Santiago de Compostela, Spain
[6]Faculty of Health, Education, and Life Sciences, City South Campus, Birmingham City University, Birmingham, United Kingdom
[7]Department of Clinical Nutrition and Dietetics, Faculty of Nutrition Sciences and Food Technology, National Nutrition and Food Technology Research Institute, Shahid Beheshti University of Medical Sciences, Tehran, Iran
[8]Hematopoietic Stem Cell Research Center, Shahid Beheshti University of Medical Sciences, Tehran, Iran
[9]EPIUnit ITR, Instituto de Saúde Pública da Universidade do Porto, Universidade do Porto, Rua das Taipas, nº 135, Porto 4050-600, Portugal
[10]Department of Food Science and Technology, TeMS.C, Islamic Azad University, Tehran, Iran
[11]Nutrition and Food Sciences Research Center, TeMS.C, Islamic Azad University, Tehran, Iran

Received: 3 August 2025 / Accepted: 2 January 2026
Published online: 30 January 2026

References

1. Health effects of dietary risks. In 195 countries, 1990–2017: a systematic analysis for the global burden of disease study 2017. Lancet. 2019;393:1958–72. https://doi.org/10.1016/s0140-6736(19)30041-8.
2. Neale EP, Batterham MJ, Tapsell LC. Consumption of a healthy dietary pattern results in significant reductions in C-reactive protein levels in adults: a meta-analysis. Nutr Res. 2016;36:391–401. https://doi.org/10.1016/j.nutres.2016.02.009.
3. Theoharides TC, et al. Mast cells and inflammation. Biochim Biophys Acta. 2012;1822:21–33. https://doi.org/10.1016/j.bbadis.2010.12.014.
4. Berg AH, Scherer PE. Adipose tissue, inflammation, and cardiovascular disease. Circ Res. 2005;96:939–49. https://doi.org/10.1161/01.Res.0000163635.62927.34.
5. Ghorabi S, et al. Association between dietary inflammatory index and components of metabolic syndrome. J Cardiovasc Thorac Res. 2020;12:27–34. https://doi.org/10.34172/jcvtr.2020.05.
6. Shivappa N, et al. Associations between dietary inflammatory index and inflammatory markers in the Asklepios study. Br J Nutr. 2015;113:665–71. https://doi.org/10.1017/s000711451400395x.
7. Darbandi M, et al. Anti-inflammatory diet consumption reduced fatty liver indices. Sci Rep. 2021;11:22601. https://doi.org/10.1038/s41598-021-98685-3.
8. Mirrafiei A, et al. The association of meal-specific food-based dietary inflammatory index with cardiovascular risk factors and inflammation in a sample of Iranian adults. BMC Endocr Disord. 2023;23:10. https://doi.org/10.1186/s12902-023-01265-x.
9. Lesani A, et al. Meal-specific dietary patterns and biomarkers of insulin resistance in a sample of Iranian adults: a cross-sectional study. Sci Rep. 2023;13:7423. https://doi.org/10.1038/s41598-023-34235-3.
10. Lesani A, Karimi M, Akbarzade Z, Djafarian K, Shab-Bidar S. The mediating role of obesity in the associations of meal-specific dietary patterns and chrono-nutrition components with cardiometabolic risk factors: structural equation modeling. Nutr Metab (Lond). 2024;21:93. https://doi.org/10.1186/s12986-024-00868-y.
11. Perrar I, et al. Circadian eating patterns track from infancy to pre- and primary school-age, but are not prospectively associated with body composition in childhood - results of the DONALD cohort study. Eur J Nutr. 2025;64:165. https://doi.org/10.1007/s00394-025-03673-2.
12. Teixeira GP, et al. Role of chronotype in dietary intake, meal timing, and obesity: a systematic review. Nutr Rev. 2022. https://doi.org/10.1093/nutrit/nuac044.
13. Adan A, et al. Circadian typology: a comprehensive review. Chronobiol Int. 2012;29:1153–75. https://doi.org/10.3109/07420528.2012.719971.
14. Rešetar J, et al. Eveningness in energy intake among adolescents with implication on anthropometric indicators of nutritional status: the CRO-PALS longitudinal study. Nutrients. 2020. https://doi.org/10.3390/nu12061710.
15. Simmons R, et al. The metabolic syndrome: useful concept or clinical tool? Report of a WHO expert consultation. Diabetologia. 2010;53:600–5.
16. Ashwell M, Gibson S. A proposal for a primary screening tool: 'keep your waist circumference to less than half your height.' BMC Med. 2014;12:207. https://doi.org/10.1186/s12916-014-0207-1.
17. Committee IR. Guidelines for data processing and analysis of the International Physical Activity Questionnaire (IPAQ)-short and long forms. 2005.
18. Ainsworth BE et al. 2011 Compendium of Physical Activities: a second update of codes and MET values. Med Sci Sports Exerc. 2011;43:1575–1581. https://doi.org/10.1249/mss.0b013e31821ece12.
19. Horne JA, Ostberg O. A self-assessment questionnaire to determine morningness-eveningness in human circadian rhythms. Int J Chronobiol. 1976;4:97–110.
20. Rahafar A, sadeghi jujilee M, Sadeghpour A, Mirzaei S. Surveying psychometric features of Persian version of Morning-Eventide questionnaire. Clin Psychol. 2013;11:109–22.
21. Lesani A, Djafarian K, Akbarzade Z, Janbozorgi N, Shab-Bidar S. Meal-specific dietary patterns and their contribution to habitual dietary patterns in the Iranian population. Br J Nutr. 2022. https://doi.org/10.1017/s0007114521005067.
22. Gibney M. Periodicity of eating and human health: present perspective and future directions. Br J Nutr. 1997;77:S3–5.
23. Ghaffarpour M, Houshiar-Rad A, Kianfar H. The manual for household measures, cooking yields factors and edible portion of foods. Tehran: Nashre Olume Keshavarzy. 1999;7:213.
24. Aghayan M, et al. Secular trend in dietary patterns of Iranian adults from 2006 to 2017: Tehran lipid and glucose study. Nutr J. 2020;19:110. https://doi.org/10.1186/s12937-020-00624-x.
25. Manoogian ENC, Chaix A, Panda S. When to eat: the importance of eating patterns in health and disease. J Biol Rhythms. 2019;34:579–81. https://doi.org/10.1177/0748730419892105.
26. Schwedhelm C, Iqbal K, Knüppel S, Schwingshackl L, Boeing H. Contribution to the understanding of how principal component analysis-derived dietary patterns emerge from habitual data on food consumption. Am J Clin Nutr. 2018;107:227–35. https://doi.org/10.1093/ajcn/nqx027.
27. Akbarzade Z, et al. The association between lunch composition and obesity in Iranian adults. Br J Nutr. 2022;127:1517–27. https://doi.org/10.1017/S0007114521002543.
28. Shivappa N, Steck SE, Hurley TG, Hussey JR, Hébert JR. Designing and developing a literature-derived, population-based dietary inflammatory index. Public Health Nutr. 2014;17:1689–96. https://doi.org/10.1017/s1368980013002115.
29. Azizi F, Esmaillzadeh A, Mirmiran P. Correlates of under- and over-reporting of energy intake in tehranians: body mass index and lifestyle-related factors. Asia Pac J Clin Nutr. 2005;14:54–9.
30. Salari-Moghaddam A, Keshteli AH, Afshar H, Esmaillzadeh A, Adibi P. Association between dietary inflammatory index and psychological profile in adults. Clin Nutr. 2019;38:2360–8. https://doi.org/10.1016/j.clnu.2018.10.015.
31. Saber N, et al. Empirical dietary inflammatory index and lifestyle inflammation score relationship with obesity: a population-based cross-sectional study. Food Sci Nutr. 2023;11:7341–51. https://doi.org/10.1002/fsn3.3660.
32. Willett WC, Howe GR, Kushi LH. Adjustment for total energy intake in epidemiologic studies. Am J Clin Nutr. 1997;65:1220S-1228S. https://doi.org/10.1093/ajcn/65.4.1220S.
33. Baveicy K, et al. Predicting metabolic syndrome by visceral adiposity index, body roundness index and a body shape index in adults: a cross-sectional study from the Iranian RaNCD cohort data. Diabetes Metab Syndr Obes. 2020;13:879–87. https://doi.org/10.2147/dmso.S238153.
34. Liu X, et al. Cardiometabolic index: a new tool for screening the metabolically obese normal weight phenotype. J Endocrinol Invest. 2021;44:1253–61. https://doi.org/10.1007/s40618-020-01417-z.
35. Wakabayashi I, Daimon T. The "cardiometabolic index" as a new marker determined by adiposity and blood lipids for discrimination of diabetes mellitus. Clin Chim Acta. 2015;438:274–8. https://doi.org/10.1016/j.cca.2014.08.042.
36. Schipf S, Knüppel S, Hardt J, Stang A. [Directed acyclic graphs (DAGs) - the application of causal diagrams in epidemiology]. Gesundheitswesen. 2011;73:888–92. https://doi.org/10.1055/s-0031-1291192.
37. Zeraattalab-Motlagh S, Lesani A, Majdi M, Shab-Bidar S. Association of chronotype with eating habits and anthropometric measures in a sample of Iranian adults. Br J Nutr. 2022. https://doi.org/10.1017/S0007114522001842.
38. Benjamini Y, Hochberg Y. Controlling the false discovery rate: a practical and powerful approach to multiple testing. J Roy Stat Soc: Ser B (Methodol). 2018;57:289–300. https://doi.org/10.1111/j.2517-6161.1995.tb02031.x.
39. Meneguelli TS, et al. Dietary inflammatory index is associated with excessive body weight and dietary patterns in subjects with cardiometabolic risk. J Food Nutr Res. 2019;7:491–9.

40. Murakami K, Livingstone MBE, Sasaki S. Meal-specific dietary patterns and their contribution to overall dietary patterns in the Japanese context: findings from the 2012 National Health and Nutrition Survey, Japan. Nutrition. 2019;59:108–15. https://doi.org/10.1016/j.nut.2018.07.110.
41. Mazidi M, et al. Dietary inflammatory index and cardiometabolic risk in US adults. Atherosclerosis. 2018;276:23–7. https://doi.org/10.1016/j.atherosclerosis.2018.02.020.
42. Camargo-Ramos CM, Correa-Bautista JE, Correa-Rodríguez M, Ramírez-Vélez R. Dietary inflammatory index and cardiometabolic risk parameters in overweight and sedentary subjects. Int J Environ Res Public Health. 2017;14:1104.
43. Aslani Z, et al. Association of dietary inflammatory potential with cardiometabolic risk factors and diseases: a systematic review and dose-response meta-analysis of observational studies. Diabetol Metab Syndr. 2020;12:86. https://doi.org/10.1186/s13098-020-00592-6.
44. Haji-Hosseini-Gazestani N, Keshavarz SA, Hosseini-Esfahani F, Ataie-Jafari A. The association of dietary inflammatory index and obesity phenotypes in women. Food Health. 2020;4(3):19.
45. Shahinfar H, et al. The association between dietary inflammatory index, muscle strength, muscle endurance, and body composition in Iranian adults. Eat Weight Disord. 2022;27:463–72. https://doi.org/10.1007/s40519-020-01096-y.
46. Sun M, et al. The mediating role of dietary inflammatory index in the association between eating breakfast and obesity: a cross-sectional study. Nutrients. 2022. https://doi.org/10.3390/nu14204378.
47. Pot GK, Hardy R, Stephen AM. Irregular consumption of energy intake in meals is associated with a higher cardiometabolic risk in adults of a British birth cohort. Int J Obes (Lond). 2014;38:1518–24. https://doi.org/10.1038/ijo.2014.51.
48. Bo S, et al. Consuming more of daily caloric intake at dinner predisposes to obesity. A 6-year population-based prospective cohort study. PLoS One. 2014;9:e108467. https://doi.org/10.1371/journal.pone.0108467.
49. Hermenegildo-López Y. A higher intake of energy at dinner is associated with incident metabolic syndrome: a prospective cohort study in older adults. Nutrients. 2021. https://doi.org/10.3390/nu13093035.
50. Koh A, De Vadder F, Kovatcheva-Datchary P, Bäckhed F. From dietary fiber to host physiology: short-chain fatty acids as key bacterial metabolites. Cell. 2016;165:1332–45. https://doi.org/10.1016/j.cell.2016.05.041.
51. Saraf-Bank S, Esmaillzadeh A, Faghihimani E, Azadbakht L. Effect of non-soy legume consumption on inflammation and serum adiponectin levels among first-degree relatives of patients with diabetes: a randomized, crossover study. Nutrition. 2015;31:459–65. https://doi.org/10.1016/j.nut.2014.09.015.
52. Barkhidarian B, Soveid N, Samadi M, Lesani A, Aghakhani A, Yekaninejad M, Saedisomeolia A, Karbasian M, Siadat S, Mirzaei K. Plant-based dietary indices association with appetite, appetite regulating peptides and gut microbiota in healthy women: a cross-sectional study. Eur J Nutr. 2025;64:166. https://doi.org/10.1007/s00394-025-03671-4.
53. Zheng JS, Hu XJ, Zhao YM, Yang J, Li D. Intake of fish and marine n-3 polyunsaturated fatty acids and risk of breast cancer: meta-analysis of data from 21 independent prospective cohort studies. BMJ. 2013;346:f3706. https://doi.org/10.1136/bmj.f3706.
54. Micha R, Wallace SK, Mozaffarian D. Red and processed meat consumption and risk of incident coronary heart disease, stroke, and diabetes mellitus: a systematic review and meta-analysis. Circulation. 2010;121:2271–83. https://doi.org/10.1161/circulationaha.109.924977.
55. Wang JB, et al. Timing of energy intake during the day is associated with the risk of obesity in adults. J Hum Nutr Diet. 2014;27(Suppl 2):255–62. https://doi.org/10.1111/jhn.12141.
56. Bo S, et al. Is the timing of caloric intake associated with variation in diet-induced thermogenesis and in the metabolic pattern? A randomized cross-over study. Int J Obes (Lond). 2015;39:1689–95. https://doi.org/10.1038/ijo.2015.138.
57. Sato M, et al. Acute effect of late evening meal on diurnal variation of blood glucose and energy metabolism. Obes Res Clin Pract. 2011;5:e169–266. https://doi.org/10.1016/j.orcp.2011.02.001.
58. Kohlgruber A, Lynch L. Adipose tissue inflammation in the pathogenesis of type 2 diabetes. Curr Diab Rep. 2015;15:92. https://doi.org/10.1007/s11892-015-0670-x.
59. Johnston JD, Ordovás JM, Scheer FA, Turek FW. Circadian rhythms, metabolism, and chrononutrition in rodents and humans. Adv Nutr. 2016;7:399–406. https://doi.org/10.3945/an.115.010777.
60. Foster RG. Sleep, circadian rhythms and health. Interface Focus. 2020;10:20190098. https://doi.org/10.1098/rsfs.2019.0098.
61. Zhao E, Tait C, Minacapelli CD, Catalano C, Rustgi VK. Circadian rhythms, the gut microbiome, and metabolic disorders. Gastro Hep Adv. 2022;1:93–105. https://doi.org/10.1016/j.gastha.2021.10.008.
62. Tucker KL. Assessment of usual dietary intake in population studies of gene-diet interaction. Nutr Metab Cardiovasc Dis. 2007;17:74–81. https://doi.org/10.1016/j.numecd.2006.07.010.
63. Livingstone MB, Black AE. Markers of the validity of reported energy intake. J Nutr. 2003;133(Suppl 3):895s–920s. https://doi.org/10.1093/jn/133.3.895S.

Publisher's note