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Association between the inflammatory potential of the diet and incidence of metabolic syndrome in middle-aged adults

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SUMMARY

Background & aims: Chronic low-grade inflammation is a central pathogenic mechanism underlying metabolic disorders, including obesity, type 2 diabetes, and cardiovascular disease. Metabolic syndrome (MetS) is closely associated with systematic inflammation, and dietary patterns are a major modifiable determinant of inflammatory status. However, prospective evidence on the association between dietary inflammatory potential and MetS incidence remains limited, particularly in Korean populations.

Methods: We analyzed 4890 adults (mean age: 50.4 years, 50.2% women) without MetS at baseline from the Ansan–Ansung cohort of the Korean Genome and Epidemiology Study. Dietary inflammatory potential was assessed using the newly developed Comprehensive Dietary Inflammation Index (CDII), derived from baseline food frequency questionnaire data. Incident MetS was defined using the National Cholesterol Education Program Adult Treatment Panel III criteria, and incident cases were defined as the first occurrence of MetS during the follow-up period from baseline through the 9th follow-up visit. Participants were categorized into CDII quartiles. Multivariable Cox proportional hazards models estimated hazard ratios (HRs) and 95% confidence intervals (CIs), adjusting for age, sex, residential area, total energy intake, drinking, smoking, physical activity, education, household income, and occupation. Additionally, associations between energy-adjusted residuals of individual food groups and MetS risk were examined to identify dietary components most strongly related to MetS.

Results: The median CDII score was −10.7 (interquartile range: −46.9 to 42.5). The mean age of the participants was 50.4 years, and the mean body mass index (BMI) was 23.7 kg/m^2. Over 18 years of follow-up, 2301 participants (47%) developed MetS. Higher CDII scores were associated with increased MetS risk (quartile 4 vs. quartile 1: HR = 1.14, 95% CI: 1.04–1.25; P for trend = 0.006). Among individual CDII components, higher sugar-sweetened beverage (SSB) intake was associated with increased MetS incidence (HR = 1.0002, 95% CI: 1.0000–1.0003), whereas higher low-fat dairy intake was associated with decreased MetS incidence (HR = 0.999, 95% CI: 0.999–1.000).

Conclusions: Higher dietary inflammatory potential, as measured by the CDII, was prospectively associated with an increased MetS risk in Korean adults. Although lower SSBs and higher low-fat dairy consumption were individually associated with a lower MetS risk, the overall dietary inflammatory profile appeared more relevant to metabolic health than single food groups.

1. Introduction

Metabolic syndrome (MetS) is defined by the presence of at least three metabolic abnormalities: abdominal obesity, hyperglycemia, hypertriglyceridemia, low high-density lipoprotein (HDL) cholesterol, and elevated blood pressure [1]. MetS substantially increases the risk of cardiovascular disease (CVD) and

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type 2 diabetes (T2D) [2,3]. Its global prevalence continues to increase, due to urbanization, sedentary lifestyles, and dietary changes, highlighting the urgency of addressing MetS [4]. In South Korea, the age-adjusted MetS prevalence increased from 27.1% in 2001 to 33.2% in 2020, with a markedly sharp increase of 19.0% points between 2010 and 2020 compared with only 3.0% points in the previous decade [5]. These trends emphasize the importance of MetS prevention, especially in the Korean population.

MetS is a multifactorial condition resulting from the integration of genetic susceptibility, lifestyle factors, adiposity, and metabolic disturbances, rather than a single determinant [6–9]. Accumulating evidence identifies chronic low-grade inflammation as a central pathogenic mechanism in MetS, contributing to insulin resistance, visceral obesity, and dyslipidemia [10,11]. Consistently, elevated inflammatory biomarkers, including tumor necrosis factor-alpha (TNF-α), C-reactive protein (CRP), and interleukin-6 (IL-6), have been associated with the MetS presence [12,13].

Several dietary factors have been identified as important contributors to inflammation. Higher intakes of antioxidant vitamins and minerals, ω-3 polyunsaturated fatty acids (PUFAs), monounsaturated fatty acids (MUFAs), flavonoids, and polyphenols are associated with lower serum levels of inflammatory markers [14,15]. In contrast, higher consumption of saturated fatty acids (SFAs), trans fatty acids (TFAs), fructose, cholesterol, and a high ω-6/ω-3 PUFA ratio are associated with increased inflammation levels [14,15]. Furthermore, dietary patterns rich in fruits, vegetables, whole grains, and unsaturated fats exhibit anti-inflammatory properties, whereas Western-type diets high in red and processed meats, refined grains, and sugar-sweetened foods are consistently associated with chronic low-grade inflammation [16,17].

Recent nutritional epidemiology has increasingly focused on dietary pattern analysis rather than individual foods or nutrients [18]. In this context, several dietary inflammatory indexes have been developed to quantify the inflammatory potential of diets, the most widely used the Dietary Inflammatory Index (DII) and the Empirical Dietary Inflammatory Index (EDII) [19]. The DII is based on 45 dietary components, largely nutrients, whereas the EDII includes 18 food components representing pro- and anti-inflammatory foods [19,20]. However, both indexes have limitations. The nutrient-based DII may not fully account for potential interactions among bioactive components within whole foods, and both the DII and EDII rely on a limited number of inflammatory biomarkers to represent complex inflammation processes [20,21]. To address these limitations, the Comprehensive Dietary Inflammation Index (CDII) was recently developed to better reflect the overall inflammatory profile of the diet [22]. Compared with the DII and EDII, the CDII has several advantages. It is based on 8 food groups rather than numerous individual nutrients, facilitating interpretation and application. Because dietary assessment tools and nutrient databases vary across studies, the nutrient-based DII may introduce between-study heterogeneity [23], whereas the food group–based CDII could be easily applied across populations using FFQs, even when biomarker or detailed nutrient data are unavailable. In addition, the CDII was derived using a larger number of inflammatory biomarkers (n = 13) than the DII or EDII (six for DII and three for EDII) [22]. Given these strengths, the CDII might be useful in Korean cohort studies, where dietary intake is commonly assessed using FFQs but inflammatory biomarkers are not available.

Associations between the DII and multiple health outcomes, including MetS, obesity, T2D, and CVD, have been reported in several countries [24–26]. However, most studies have focused on Western populations, and evidence from Asian populations, particularly Koreans, remains limited [27,28]. In addition, because the CDII is a recently developed index, research on its relevance to chronic disease is scarce. Only one study has examined its association with chronic kidney disease [22], and no studies have investigated its relationship with other chronic diseases, particularly MetS. Therefore, the present study aimed to investigate the prospective association between the CDII and MetS incidence in middle-aged and older Korean adults using a large community-based cohort.

2. Methods

2.1. Study population and data source

This study used prospective data from the Ansan–Ansung cohort of the Korean Genome and Epidemiology Study (KoGES), a large prospective cohort established by the Korea Centers for Disease Control and Prevention to investigate risk factors for chronic diseases in Koreans. The Ansan–Ansung cohort has been conducted since 2001 with biennial in-person follow-up surveys. At baseline, participants were 40–69 years old and resided in either the urban area of Ansan or the rural area of Ansung. For the present analysis, we used data from baseline through the 9th follow-up surveys conducted in 2019–2020 (follow-up rate: 58.4%), the most recent follow-up data available. At each follow-up, participants provided biospecimens, completed questionnaires on lifestyle and medical history, and underwent physical examinations including anthropometric and blood pressure measurements. The study protocol was reviewed and approved by the Institutional Review Board of Ewha Womans University, Korea (institutional review board number ewha-202603-0023-01).

Of the 10,030 baseline participants, we excluded individuals with missing energy intake data (n = 326) or implausible energy intake (<500 kcal/day or >5,000 kcal/day; n = 87), consistent with criteria commonly applied in KoGES-based studies (Fig. 1) [29,30]. Then, we excluded those with a history of hypertension, diabetes, dyslipidemia, and cancer (n = 2214) to minimize reverse causation and confounding from pre-existing chronic diseases, including disease-related dietary modifications and medical treatment [31–34]. In addition, we excluded participants with missing baseline information on disease-defining variables or covariates (n = 1121) and those with MetS at baseline (n = 1392). After these exclusions, 4890 participants were included in the main analysis of dietary inflammatory potential and incident MetS. For subanalyses of individual MetS components (hyperglycemia, hypertriglyceridemia, low HDL cholesterol, elevated blood pressure, and abdominal obesity), separate analytic samples were constructed. Participants with missing data for a given component or with prevalent abnormality at baseline were excluded from the corresponding analysis. As a result, sample sizes varied across MetS component-specific analyses.

2.2. Dietary inflammatory potential

Dietary inflammatory potential was assessed using the CDII [22]. Details of its development have been reported previously [22]. CDII scores were calculated from responses to a 103-item semi-quantitative food frequency questionnaire (FFQ) at baseline. Participants reported consumption frequency (from never or seldom to ≥ three times/day), and portion sizes (small, medium, or large) using visual representations (glasses, plates, bowls) to improve estimation. The FFQ has been validated against 12-day dietary records and shown to have acceptable reproducibility [35]. FFQ items were classified into CDII food groups, and daily intake of each food group was estimated by summing relevant items (Supplementary Table 1). Intakes were energy-adjusted

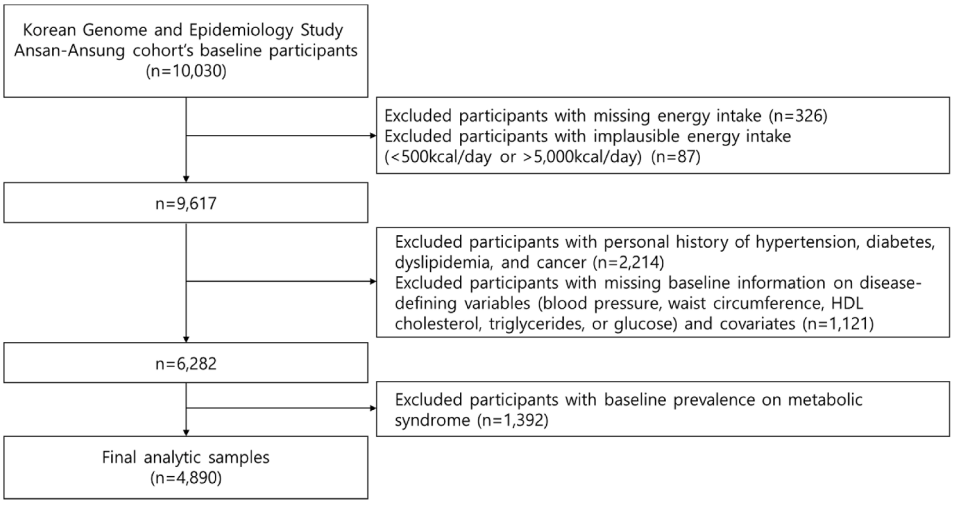

Fig. 1. Flow chart of study participants selection. HDL, high-density lipoprotein.

using the residual method, and multipled by published factor loadings from the original CDII study [36]. The overall CDII score was calculated by summing weighted food group contributions, with higher scores indicating a more pro-inflammatory dietary profile and lower scores indicating a lower inflammatory potential.

2.3. Metabolic syndrome ascertainment

MetS was defined according to the National Cholesterol Education Program Adult Treatment Panel III criteria as the presence of at least three of the following components: hyperglycemia (fasting glucose ≥100 mg/dL or current diabetes medication use), hypertriglyceridemia (triglycerides ≥150 mg/dL or current dyslipidemia medication use), low HDL cholesterol (<40 mg/dL in men or <50 mg/dL in women), elevated blood pressure (systolic ≥130 mmHg, diastolic ≥85 mmHg, or current hypertension medication use), and abdominal obesity (waist circumference ≥90 cm in men or ≥85 cm in women) [37]. Waist circumference cut-offs were based on the Korean-specific criteria for abdominal obesity [38]. In survival analyses, the survival time to event was calculated from baseline to the first occurrence of MetS or its individual components. Participants who remained free of MetS or its components throughout the study period were censored at their last follow-up visit, and those lost to follow-up were censored at the date of their last completed assessment.

2.4. Covariates

Sociodemographic and lifestyle characteristics were assessed at baseline using a self-administered questionnaire. Covariates included sex, total energy intake (kcal/day), drinking status (non-drinker, former drinker, current drinker), smoking level (pack-years), education level (≤high school or ≥college), household income level (<200, 200–400, or ≥400 ten-thousand KRW per month), and occupation (white-collar, blue-collar [agriculture and factory production], and others [housewives and unspecified occupations]). Physical activity was quantified as weekly metabolic equivalent of task (MET) scores by multiplying reported activity duration by intensity-specific MET values and summing across activity levels. Information on the intensity and duration of daily physical activity was obtained using a structured questionnaire, which assessed sedentary (1.5 METs), light (3 METs), moderate (5 METs), and vigorous (7 METs) activities [39–41]. Total physical activity was expressed as MET-minutes per week and categorized into quartiles.

Age and residential area were included as stratification variables in Cox proportional hazards models. Age was categorized into 40–49, 50–59, and ≥60 years, and residential area was classified as rural (Ansung) or urban (Ansan).

2.5. Statistical analysis

Participants were categorized into quartiles of CDII. Baseline characteristics overall and by quartiles were presented as means±standard deviations (SDs) for continuous variables and frequencies (percentages) for categorical variables. Trends across quartiles were assessed using generalized linear regression for continuous variables, the Cochran–Mantel–Haenszel linear-by-linear association test for ordinal variables (drinking status, household income level, and occupation), and the Cochran–Armitage trend test for binary variables (sex, residential area, and education level).

Multivariable Cox proportional hazards models estimated hazard ratios (HRs) and 95% confidence intervals (CIs) for the association between CDII and incident MetS and its individual components. The proportional hazards assumption was evaluated using Schoenfeld residuals and was violated for age and residential area; therefore, both were included as stratification variables. Three models were adjusted for potential confounders: Model 1 adjusted for sex and total energy intake, with age and residential area included as stratification variables; Model 2 additionally adjusted for drinking status, smoking level, and physical activity level; Model 3 further adjusted for education level, household income level, and occupation. Quartile analyses used raw CDII values. Linear trends across CDII quartiles were tested by assigning the median value of each quartile of the raw CDII and modeling it as a continuous variable. Incidence density was expressed as incident cases per 1000 person-years. Associations were also examined per 1-SD increase in standardized CDII. The dose–response relationship was evaluated using restricted cubic splines with four knots (5th, 35th, 65th, and 95th percentiles) with the 10th percentile of CDII as reference, and adjusted for covariates in Model 3.

To examine independent dietary component effects, residuals of individual CDII food groups were included simultaneously in

Table 1
Baseline characteristics of study participants overall and by CDII quartiles.

	All (n = 4890)	Quartile 1 (n = 1222)	Quartile 2 (n = 1223)	Quartile 3 (n = 1223)	Quartile 4 (n = 1222)	P for trend
CDII	−10.7 (−46.9, 42.5)[a]	−84.4 (−121.9, −63.78)	−28.2 (−35.8, −20.7)	15.9 (3.1, 28.5)	97.8 (72.8, 138.7)	<0.0001
Sex						<0.0001
Men	2437 (49.8)	431 (35.3)	527 (43.1)	674 (55.1)	805 (65.9)	
Women	2453 (50.2)	791 (64.7)	696 (56.9)	549 (44.9)	417 (34.1)	
Age groups						<0.0001
40–49 years	2795 (57.2)	659 (53.9)	602 (49.2)	731 (59.8)	803 (65.7)	
50–59 years	1139 (23.3)	306 (25.1)	296 (24.2)	277 (22.6)	260 (21.3)	
≥60 years	956 (19.5)	257 (21.0)	325 (26.6)	215 (17.6)	159 (13.0)	
Residential area						<0.0001
Rural, Ansung	2099 (42.9)	567 (46.4)	633 (51.8)	423 (34.6)	476 (39.0)	
Urban, Ansan	2791 (57.1)	655 (53.6)	590 (48.2)	800 (65.4)	746 (61.1)	
Drinking status						<0.0001
Non-drinker	2110 (43.2)	655 (53.6)	595 (48.7)	430 (35.2)	430 (35.2)	
Former drinker	271 (5.5)	60 (4.9)	51 (4.2)	71 (5.8)	89 (7.3)	
Current drinker	2509 (51.3)	507 (41.5)	577 (47.2)	722 (59.0)	703 (57.5)	
Smoking level (pack-years)	9.4 ± 15.4	5.8 ± 12.2	6.7 ± 13.3	10.0 ± 15.3	15.0 ± 18.4	<0.0001
Physical activity level (MET score)	166.9 ± 102.1	169.4 ± 102.6	177.0 ± 104.3	162.9 ± 101.1	158.5 ± 99.7	<0.0001
Education level						<0.0001
≤High school	4172 (85.3)	1052 (86.1)	1084 (88.6)	1035 (84.6)	1001 (81.9)	
≥College	718 (14.7)	170 (13.9)	139 (11.4)	188 (15.4)	221 (18.1)	
Household income level (ten-thousand, KRW)						<0.0001
<200	2914 (59.6)	754 (61.7)	816 (66.7)	684 (55.9)	660 (54.0)	
200-400	1569 (32.1)	356 (29.1)	327 (26.7)	436 (35.7)	450 (36.8)	
≥400	407 (8.3)	112 (9.2)	80 (6.5)	103 (8.4)	112 (9.2)	
Occupation						<0.0001
White-collar	1304 (26.7)	250 (20.5)	222 (18.1)	363 (29.7)	469 (38.4)	
Blue-collar	1521 (31.1)	379 (31.0)	457 (37.4)	337 (27.6)	348 (28.5)	
Others	2065 (42.2)	593 (48.5)	544 (44.5)	523 (42.7)	405 (33.1)	
BMI (kg/m^2)	23.7 ± 2.8	23.6 ± 2.8	23.3 ± 2.8	23.8 ± 2.7	23.9 ± 2.8	<0.0001
WC (cm)	79.4 ± 7.7	78.8 ± 7.8	78.9 ± 7.7	79.5 ± 7.4	80.4 ± 7.6	<0.0001
SBP (mmHg)	114.7 ± 14.9	114.7 ± 14.5	115.9 ± 16.2	114.7 ± 14.5	113.7 ± 14.3	0.005
DBP (mmHg)	76.6 ± 10.1	76.4 ± 10.2	77.0 ± 10.6	76.5 ± 9.5	76.6 ± 10.0	0.436
Fasting blood glucose (mg/dL)	83.1 ± 12.1	82.4 ± 10.5	83.0 ± 12.0	83.7 ± 14.0	83.4 ± 11.7	0.062
TG (mg/dL)	134.2 ± 80.2	129.2 ± 77.7	131.8 ± 80.3	136.2 ± 79.8	139.5 ± 82.6	0.008
HDL (mg/dL)	46.8 ± 10.1	47.6 ± 10.5	47.2 ± 10.0	47.0 ± 10.1	45.4 ± 9.5	<0.0001
Total energy intake (kcal/day)	2004.9 ± 610.0	2067.8 ± 678.2	1865.5 ± 582.5	2004.7 ± 580.4	2081.9 ± 569.2	<0.0001
Energy-adjusted dietary intake						
Processed meat (g/day)	1.2 ± 3.6	0.9 ± 3.2	1.1 ± 3.3	1.4 ± 3.6	1.3 ± 4.1	0.003
Eggs (g/day)	12.7 ± 14.8	12.9 ± 14.9	11.9 ± 14.1	13.5 ± 15.2	12.4 ± 14.8	0.052
Organ meats (g/day)	0.9 ± 3.1	0.7 ± 2.9	0.9 ± 2.7	1.0 ± 2.5	1.3 ± 4.1	<0.0001
Sugar sweetened beverages (g/day)	250.1 ± 264.8	55.9 ± 93.7	104.7 ± 103.1	239.0 ± 110.0	600.9 ± 258.9	<0.0001
Dark yellow vegetables (g/day)	54.0 ± 72.5	89.4 ± 117.2	49.9 ± 43.8	39.4 ± 39.8	37.1 ± 45.1	<0.0001
Coffee (g/day)	20.8 ± 105.4	81.7 ± 198.2	1.4 ± 13.1	0.2 ± 4.8	−0.2 ± 1.4	<0.0001
Low fat dairy (g/day)	103.9 ± 124.3	193.1 ± 163.7	88.1 ± 94.1	75.2 ± 90.5	59.1 ± 83.2	<0.0001
Sweets and desserts (g/day)	4.3 ± 10.6	4.1 ± 10.7	4.6 ± 11.6	4.2 ± 8.5	4.4 ± 11.5	0.689
Carbohydrate (g/day)	359.1 ± 35.9	358.4 ± 39.0	364.4 ± 33.9	358.1 ± 34.1	355.5 ± 35.8	<0.0001
Protein (g/day)	65.6 ± 11.5	67.9 ± 12.3	64.8 ± 10.6	65.3 ± 10.8	64.3 ± 11.9	<0.0001
Fat (g/day)	31.5 ± 12.4	31.7 ± 13.3	29.2 ± 11.9	31.7 ± 11.9	33.6 ± 12.2	<0.0001
Fiber (g/day)	29.5 ± 7.3	31.6 ± 8.3	30.3 ± 7.1	28.7 ± 6.5	27.5 ± 6.6	<0.0001
Sodium (mg/day)	2458.7 ± 1004.0	2512.1 ± 1033.8	2400.6 ± 1021.4	2449.2 ± 1005.5	2472.9 ± 951.2	0.0481

[a] Values are presented as mean ± standard deviation for continuous variables and n (%) for categorical variables, except for the CDII score, which is presented as median (Q1, Q3). The p-values for trend across quartiles of CDII scores were calculated using generalized linear regression for continuous variables, the Cochran–Mantel–Haenszel linear-by-linear association test for ordinal variables (drinking status and household income level), and the Cochran–Armitage trend test for binary variables (sex, residential area, education level, and occupation). Quartiles of CDII were based on the raw CDII score. CDII, Comprehensive Dietary Inflammatory Index; MET, metabolic equivalent of task; BMI, body mass index; WC, waist circumference; SBP, systolic blood pressure; DBP, diastolic blood pressure; TG, triglycerides.

a multivariable Cox model with mutual adjustment. Multicollinearity was assessed using variance inflation factors (VIFs) and tolerance statistics. As all VIF values were <2.0 (maximum VIF = 1.04) and all tolerance values were >0.2 (minimum tolerance = 0.96), multicollinearity was not considered problematic. Additional models including each food group residual separately were conducted and reported in the supplementary table.

All statistical analyses were performed using SAS software (version 9.4; SAS Institute Inc., Cary, NC, USA) and the restricted cubic spline analysis was performed using R software (version 4.4.3; R Foundation for Statistical Computing, Vienna, Austria).

3. Results

3.1. Characteristics of the study subjects

The median CDII score was −10.7 (interquartile range: −46.9 to 42.5; Table 1). The mean age of the participants was 50.4 years, and the mean body mass index (BMI) was 23.7 kg/m^2. Across increasing CDII quartiles, indicating a more pro-inflammatory dietary pattern, participants were more likely to be men, younger, urban residents, current drinkers, and white-collar workers, and to have a higher smoking level, lower physical activity, higher education, and higher household income (all p for trend <0.05). In

Table 2
Associations between quartiles of the CDII and risk of metabolic syndrome.

Models	Quartile 1	Quartile 2	Quartile 3	Quartile 4	P for trend	Per SD higher
Metabolic syndrome (n = 4890)						
No. of events	580	554	581	586		
Incidence density	43.22	41.50	43.37	45.05		
Model 1	Ref.	0.94 (0.84–1.06)[a]	1.08 (0.96–1.22)	1.13 (1.01–1.28)[b]	0.008	1.06 (1.01–1.10)[b]
Model 2	Ref.	0.95 (0.86–1.04)	1.09 (0.99–1.20)	1.14 (1.04–1.25)[b]	0.006	1.06 (1.02–1.10)[b]
Model 3	Ref.	0.95 (0.86–1.04)	1.09 (0.99–1.20)	1.14 (1.04–1.25)[b]	0.006	1.06 (1.02–1.10)[b]
Abdominal obesity (n = 4702)						
No. of events	500	449	470	489		
Incidence density	37.63	33.68	34.54	37.62		
Model 1	Ref.	0.89 (0.78–1.01)	1.03 (0.91–1.17)	1.16 (1.02–1.31)[b]	0.006	1.06 (1.01–1.11)[b]
Model 2	Ref.	0.88 (0.77–1.02)	1.04 (0.91–1.18)	1.19 (1.04–1.37)[b]	0.003	1.07 (1.02–1.12)[b]
Model 3	Ref.	0.89 (0.78–1.02)	1.04 (0.91–1.19)	1.20 (1.04–1.37)[b]	0.003	1.07 (1.02–1.12)[b]
Elevated blood pressure (n = 4157)						
No. of events	597	562	573	567		
Incidence density	55.10	51.72	52.78	54.05		
Model 1	Ref.	0.90 (0.80–1.01)	1.01 (0.89–1.13)	1.01 (0.90–1.14)	0.508	1.03 (0.99–1.08)
Model 2	Ref.	0.98 (0.89–1.08)	1.01 (0.91–1.11)	1.01 (0.92–1.11)	0.803	1.00 (0.97–1.05)
Model 3	Ref.	0.98 (0.89–1.08)	1.01 (0.91–1.11)	1.01 (0.92–1.11)	0.800	1.00 (0.97–1.05)
Hyperglycemia (n = 5806)						
No. of events	608	618	626	610		
Incidence density	37.30	38.62	39.40	39.42		
Model 1	Ref.	0.99 (0.88–1.11)	1.01 (0.90–1.13)	0.94 (0.84–1.05)	0.308	0.99 (0.95–1.03)
Model 2	Ref.	0.97 (0.88–1.06)	0.97 (0.88–1.06)	0.90 (0.82–0.99)[b]	0.069	0.98 (0.94–1.02)
Model 3	Ref.	0.96 (0.88–1.06)	0.97 (0.85–1.06)	0.90 (0.82–0.99)[b]	0.074	0.98 (0.94–1.02)
Hypertriglyceridemia (n = 3913)						
No. of events	510	525	552	546		
Incidence density	49.20	51.49	54.87	56.88		
Model 1	Ref.	1.02 (0.90–1.16)	1.11 (0.99–1.26)	1.19 (1.05–1.35)[b]	0.002	1.06 (1.01–1.11)[b]
Model 2	Ref.	1.04 (0.94–1.15)	1.12 (1.02–1.24)[b]	1.16 (1.06–1.29)[b]	0.007	1.05 (1.01–1.10)[b]
Model 3	Ref.	1.04 (0.94–1.15)	1.12 (1.02–1.23)[b]	1.16 (1.05–1.28)[b]	0.008	1.05 (1.01–1.10)[b]
Low HDL cholesterol (n = 2997)						
No. of events	421	429	391	410		
Incidence density	61.04	64.73	54.60	58.17		
Model 1	Ref.	1.06 (0.92–1.21)	0.98 (0.85–1.13)	1.11 (0.97–1.28)	0.208	1.00 (0.95–1.05)
Model 2	Ref.	1.10 (0.98–1.23)	0.97 (0.86–1.083)	1.09 (0.98–1.22)	0.397	0.99 (0.94–1.04)
Model 3	Ref.	1.10 (0.98–1.22)	0.97 (0.86–1.08)	1.10 (0.97–1.22)	0.412	0.99 (0.94–1.04)

[a] Values are presented as HRs (95% CIs).
[b] $P < 0.05$. Incidence density is expressed as the number of incident cases per 1000 person-years. Hazard ratios across quartiles of CDII were estimated using the raw CDII. P for trend was calculated by modeling the median value of each quartile of raw CDII as a continuous variable in the Cox proportional hazards models, and hazard ratios per 1-SD increase were estimated using standardized CDII. Model 1 was adjusted for sex and total energy intake, and stratified by age group (40–49, 50–59, and ≥60 years) and residential area; Model 2 was adjusted for covariates in model 1 and drinking status, smoking level, and physical activity level; Model 3 adjusted for covariates in model 2 and education level, household income level, and occupation. The analytic sample size varied across metabolic syndrome component analyses because participants with missing data or prevalent abnormality of the corresponding component at baseline were excluded. CDII, comprehensive dietary inflammatory index; HR, hazard ratio; CI, confidence interval; SD, standard deviation; Q, quartile.

addition, higher CDII quartiles were associated with higher BMI and waist circumference, lower systolic blood pressure (SBP), higher triglyceride (TG) levels, and lower HDL cholesterol levels (all p for trend <0.05). Dietary patterns differed substantially across quartiles. Individuals with higher CDII quartiles consumed more processed meat, organ meats, and sugar-sweetened beverages (SSBs), and fewer dark yellow vegetables, coffee, and low-fat dairy products (all p for trend <0.05). In addition, higher CDII quartiles were associated with lower carbohydrate, protein, and fiber intake, but higher fat intake (all p for trend <0.001). Sodium intake was highest in the lowest CDII quartile but showed an overall increasing trend across higher quartiles (p for trend = 0.048).

3.2. Relationship between CDII and MetS

During 18 years of follow-up (mean: 10.87 years), 2301 participants (47%) developed MetS. In multivariable models, individuals in the highest CDII quartile had a 14% higher risk of MetS compared with those in the lowest quartile (HR = 1.14, 95% CI: 1.04–1.25; p for trend = 0.006; Table 2). Each 1-SD increase in CDII was associated with a 6% higher MetS risk (HR = 1.06, 95% CI: 1.02–1.10). These associations were consistent across all adjustment models. For individual MetS components, higher CDII was associated with increased risk of abdominal obesity (quartile 4 vs. quartile 1: HR = 1.20, 95% CI: 1.04–1.37; p for trend = 0.003) and hypertriglyceridemia (quartile 4 vs. quartile 1: HR = 1.16, 95% CI: 1.05–1.28; p for trend = 0.008). Each 1-SD increase in CDII was also significantly associated with abdominal obesity (HR = 1.07, 95% CI: 1.02–1.12) and hypertriglyceridemia (HR = 1.05, 95% CI: 1.01–1.10). However, a significant inverse association was observed for hyperglycemia when comparing the highest and lowest quartiles (HR = 0.90, 95% CI: 0.82–0.99), but this relationship was not significant in the trend or continuous analyses (p for trend = 0.074; HR = 0.98, 95% CI: 0.94–1.02). Restricted cubic spline analysis indicated a nonlinear dose–response association between CDII and MetS incidence (Fig. 2). The HR was approximately 1.0 at lower CDII values and increased gradually when standardized CDII was above the mean (0.00).

3.3. Exploring the relationship between individual CDII components and MetS

In the mutually adjusted model including all energy-adjusted food groups, higher intake of SSBs was associated with increased MetS risk (HR = 1.0002, 95% CI: 1.0000–1.0003; Table 3), whereas

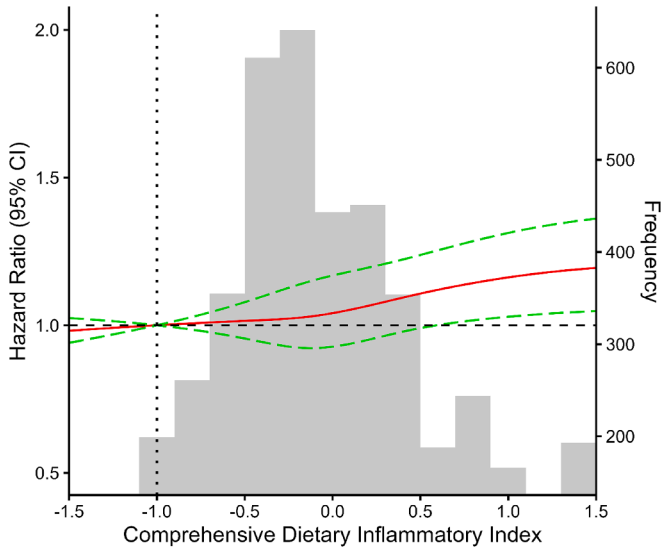

Fig. 2. Associations between CDII and risk of metabolic syndrome. The histogram represents the frequency distribution of standardized CDII scores. The red solid line indicates the adjusted HR for metabolic syndrome incidence, modeled using restricted cubic splines with four knots at the 5th, 35th, 65th, and 95th percentiles. The reference value was set at the 10th percentile of the CDII (z-score: −1.00), shown as the vertical dotted line. Green dashed lines represent 95% CIs. The model was adjusted for age, total energy intake, drinking status, smoking level, physical activity level, education level, household income level, and occupation, and stratified by age group (40–49, 50–59, and ≥60 years) and residential area. CDII, Comprehensive Dietary Inflammatory Index; HR, hazard ratio; CI, confidence interval.

higher intake of low-fat dairy was associated with reduced MetS risk (HR = 0.999, 95% CI: 0.999–1.000). Among the individual components of MetS, higher SSBs intake was positively associated with abdominal obesity and elevated blood pressure (abdominal obesity: HR = 1.0002, 95% CI: 1.0001–1.0004; elevated blood pressure: HR = 1.0002, 95% CI: 1.0000–1.0003). In contrast, higher low-fat dairy intake was inversely associated with hypertriglyceridemia (HR = 1.000, 95% CI: 0.999–1.000). Hyperglycemia was associated with higher intakes of organ meats (HR = 1.016, 95% CI: 1.005–1.027) and coffee (HR = 1.000, 95% CI: 1.000–1.001), as well as lower intake of sweets and desserts (HR = 0.995, 95% CI: 0.991–1.000). Low HDL cholesterol was inversely associated with eggs intake (HR = 0.997, 95% CI: 0.993–1.000). In additional models without mutual adjustment, similar results were observed (Supplementary Table 2). Higher SSBs intake was associated with incident MetS and abdominal obesity, while lower low-fat dairy intake was associated with incident MetS and hypertriglyceridemia. Hyperglycemia was also associated with higher intakes of organ meats and coffee, and lower intakes of sweets and desserts.

4. Discussion

This study examined the association between the dietary inflammatory potential, asmeasured by the CDII, and MetS risk in a recent community-based prospective cohort. Higher CDII scores, indicating a more pro-inflammatory diet, were associated with increased MetS risk, especially abdominal obesity and hypertriglyceridemia. Among the individual dietary components of the CDII, SSBs and low-fat dairy intake showed a significant association with MetS; however, their modest effects suggest that the overall dietary inflammatory profile is more relevant to metabolic health than any single food component.

Dietary inflammatory indexes such as the DII have been widely investigated in relation to MetS. However, the reported associations have been inconsistent across populations. Several cohort studies and meta-analyses reported that higher DII is associated with increased MetS risk and adverse metabolic components, including abdominal obesity, elevated blood pressure, hyperglycemia, and hypertriglyceridemia [42–45], whereas other studies have observed null or limited association [46,47]. These discrepancies may reflect differences in population characteristics, such as race/ethnicity, sex, and age, dietary assessment methods, study designs, and sample sizes [43,45–50]. Additionally, the DII is a largely nutrient-based index, which may limit its ability to capture interactions among whole foods [20]. More recently, the Dietary Inflammation Score (DIS), EDII, and CDII have been developed to better reflect the inflammatory potential of the overall diet and improve applicability in FFQ-based studies, and have been associated with metabolic outcomes [19,51–53]. However, because these indexes were originally developed using U.S. cohorts, their direct application to Korean dietary data may introduce measurement challenges related to differences in dietary patterns and food culture. In particular, several components of DIS and EDII cannot be directly mapped to the KoGES FFQ, limiting the feasibility of deriving comparable scores in the present dataset. The DIS includes 19 components, including high-fat dairy, low-fat dairy, and dietary supplements, and the EDII includes 18 food groups such as high-energy beverages and low-energy beverages, many of which are not available in the KoGES FFQ items. In contrast, all CDII components align with the available FFQ data, enabling consistent score calculation within this cohort and reducing misclassification. Together, these findings suggest that dietary inflammatory potential may influence metabolic risk, although its overall relationship with MetS remains incompletely understood [45]. In this context, the present study supports the CDII as a useful and complementary tool for assessing the diet-related inflammation in relation to MetS risk in the Korean population.

In Korea, only a few studies have investigated the relationship between dietary inflammatory indexes and MetS. One study developed an inflammatory index based on 10 food groups and reported that higher inflammatory potential was associated with an increased MetS prevalence [50,54]. However, its association was evaluated using cross-sectional data from the Korea National Health and Nutrition Examination Survey (KNHANES), where dietary intake was assessed by a single 24-h recall. Another KNHANES-based study also reported a positive association between the DII and MetS prevalence, but its cross-sectional design limited causal inference and the DII calculation was restricted to 23 of the original 45 components due to data constraints [48,54]. Despite these limitations, both studies consistently suggested a significant association between dietary inflammatory potential and MetS. The present study extends this evidence by using the CDII derived from FFQ data that better reflect long-term dietary intake and by applying prospective cohort data, providing stronger temporal evidence for the association between dietary inflammation and MetS.

Several CDII components were associated with MetS and its individual abnormalities, consistent with prior evidence. Higher SSBs intake is known to promote weight gain and metabolic dysfunction through excess caloric intake and adverse glycemic responses, including increased triglycerides, hepatic insulin resistance, and steatosis [55–57]. A meta-analysis confirmed that higher SSB consumption is positively associated with MetS risk [58]. In contrast, low-fat dairy intake has been associated with enhanced immune and inflammatory profiles, including IL-2, TNF-α, and IFN-γ, and lower MetS risk [29,59,60]. Consumption of organ meats has also been positively associated with MetS [61,62].

Table 3
Associations between standardized residuals of food group and risk of metabolic syndrome and its components, adjusted for residuals of all other food groups.

	Model 1	Model 2	Model 3
Metabolic syndrome (n = 4890)			
Processed meats	0.995 (0.983–1.007)	0.992 (0.981–1.005)	0.992 (0.980–1.004)
Eggs	0.998 (0.996–1.001)	0.998 (0.995–1.001)	0.998 (0.995–1.001)
Organ meats	0.999 (0.985–1.012)	1.001 (0.987–1.016)	1.001 (0.987–1.016)
Sugar-sweetened beverages	1.0002 (1.0000–1.0003)[a]	1.0002 (1.0000–1.0003)[a]	1.0002 (1.0000–1.0003)[a]
Dark yellow vegetables[b]	1.000 (0.999–1.000)	1.000 (0.999–1.000)	1.000 (0.999–1.000)
Coffee[b]	1.000 (1.000–1.001)	1.000 (1.000–1.001)	1.000 (1.000–1.001)
Low-fat dairy[b]	0.999 (0.999–1.000)[a]	0.999 (0.999–1.000)[a]	0.999 (0.999–1.000)[a]
Sweets and desserts[b]	0.998 (0.993–1.002)	0.997 (0.992–1.001)	0.997 (0.992–1.001)
Abdominal obesity (n = 4702)			
Processed meats	1.000 (0.987–1.012)	0.999 (0.986–1.011)	0.998 (0.986–1.011)
Eggs	0.998 (0.995–1.001)	0.998 (0.995–1.001)	0.998 (0.995–1.001)
Organ meats	1.005 (0.993–1.018)	1.009 (0.996–1.023)	1.009 (0.996–1.022)
Sugar-sweetened beverages	1.0002 (1.0000–1.0004)[a]	1.0002 (1.0001–1.0004)[a]	1.0002 (1.0001–1.0004)[a]
Dark yellow vegetables[b]	1.000 (0.999–1.000)	1.000 (0.999–1.001)	1.000 (0.999–1.001)
Coffee[b]	1.000 (1.000–1.001)	1.000 (1.000–1.001)	1.000 (1.000–1.001)
Low-fat dairy[b]	1.000 (0.999–1.000)	1.000 (0.999–1.000)	1.000 (0.999–1.000)
Sweets and desserts[b]	0.998 (0.993–1.003)	0.999 (0.994–1.003)	0.999 (0.994–1.003)
Elevated blood pressure (n = 4157)			
Processed meats	0.996 (0.984–1.008)	0.999 (0.987–1.011)	1.000 (0.988–1.012)
Eggs	0.998 (0.995–1.001)	0.998 (0.995–1.001)	0.998 (0.995–1.001)
Organ meats	1.011 (0.999–1.023)	1.008 (0.995–1.020)	1.007 (0.995–1.020)
Sugar-sweetened beverages	1.000 (1.000–1.000)	1.0001 (1.0000–1.0003)[a]	1.0002 (1.0000–1.0003)[a]
Dark yellow vegetables[b]	1.000 (0.999–1.000)	1.000 (0.999–1.000)	1.000 (0.999–1.000)
Coffee[b]	1.000 (1.000–1.001)	1.000 (1.000–1.001)	1.000 (1.000–1.001)
Low-fat dairy[b]	1.000 (1.000–1.000)	1.000 (0.999–1.000)	1.000 (0.999–1.000)
Sweets and desserts[b]	1.000 (0.995–1.004)	1.000 (0.995–1.004)	1.000 (0.995–1.004)
Hyperglycemia (n = 5806)			
Processed meats	0.997 (0.986–1.010)	0.997 (0.985–1.009)	0.997 (0.985–1.009)
Eggs	0.999 (0.996–1.002)	0.999 (0.996–1.001)	0.999 (0.996–1.001)
Organ meats	1.012 (1.002–1.023)[a]	1.016 (1.005–1.027)[a]	1.016 (1.005–1.027)[a]
Sugar-sweetened beverages	1.000 (1.000–1.000)	1.000 (1.000–1.000)	1.000 (1.000–1.000)
Dark yellow vegetables[b]	1.000 (0.999–1.000)	1.000 (0.999–1.000)	1.000 (0.999–1.000)
Coffee[b]	1.000 (1.000–1.001)	1.000 (1.000–1.001)[a]	1.000 (1.000–1.001)[a]
Low-fat dairy[b]	1.000 (0.999–1.000)	1.000 (0.999–1.000)	1.000 (0.999–1.000)
Sweets and desserts[b]	0.994 (0.989–0.999)[a]	0.995 (0.991–1.000)[a]	0.995 (0.991–1.000)[a]
Hypertriglyceridemia (n = 3913)			
Processed meats	0.994 (0.982–1.007)	0.991 (0.979–1.004)	0.991 (0.979–1.004)
Eggs	0.998 (0.995–1.001)	0.998 (0.995–1.001)	0.998 (0.995–1.001)
Organ meats	1.006 (0.992–1.021)	1.007 (0.993–1.022)	1.008 (0.994–1.023)
Sugar-sweetened beverages	1.0002 (1.0000–1.0003)[a]	1.000 (1.000–1.000)	1.000 (1.000–1.000)
Dark yellow vegetables[b]	1.000 (0.999–1.001)	1.000 (0.999–1.001)	1.000 (0.999–1.001)
Coffee[b]	1.000 (0.999–1.000)	1.000 (0.999–1.000)	1.000 (0.999–1.000)
Low-fat dairy[b]	1.000 (0.999–1.000)[a]	1.000 (0.999–1.000)[a]	1.000 (0.999–1.000)[a]
Sweets and desserts[b]	0.997 (0.993–1.002)	0.997 (0.993–1.002)	0.997 (0.993–1.002)
Low HDL cholesterol (n = 2997)			
Processed meats	1.004 (0.990–1.019)	1.006 (0.992–1.020)	1.006 (0.993–1.020)
Eggs	0.997 (0.994–1.000)	0.996 (0.993–1.000)[a]	0.997 (0.993–1.000)[a]
Organ meats	1.004 (0.989–1.020)	1.009 (0.993–1.025)	1.009 (0.993–1.025)
Sugar-sweetened beverages	1.000 (1.000–1.000)	1.000 (1.000–1.000)	1.000 (1.000–1.000)
Dark yellow vegetables[b]	1.000 (1.000–1.001)	1.000 (1.000–1.001)	1.000 (1.000–1.001)
Coffee[b]	1.000 (1.000–1.001)	1.000 (1.000–1.001)	1.000 (1.000–1.001)
Low-fat dairy[b]	1.000 (0.999–1.000)	1.000 (0.999–1.000)	1.000 (0.999–1.000)
Sweets and desserts[b]	1.003 (0.998–1.007)	1.003 (0.998–1.007)	1.003 (0.998–1.007)

Values are presented as HRs (95% CIs).
[a] $P < 0.05$.
[b] In the CDII, these items are assigned negative scores, indicating that their association is expected to be in the opposite direction compared to the other food groups. Model 1 was adjusted for sex and total energy intake, and stratified by age group (40–49, 50–59, and ≥60 years) and residential area; Model 2 was adjusted for covariates in model 1 and drinking status, smoking level, and physical activity level; Model 3 adjusted for covariates in model 2 and education level, household income level, and occupation. CDII, comprehensive dietary inflammatory index; HR, hazard ratio; CI, confidence interval.

Evidence regarding coffee consumption and metabolic outcomes is inconsistent, with one study reporting favorable metabolic and inflammatory profiles, including lower CRP, IL-6, sTNF-R2, C-peptide, IGFBP-3, total estradiol, and leptin, whereas other studies reported reduced insulin sensitivity with coffee intake [63,64]. In addition, the inverse association observed between sweets and desserts and hyperglycemia should be interpreted cautiously, as this food group includes cocoa- and chocolate-based products, which contain polyphenols that have been associated with reduced risk of T2D and MetS [65–67], reflecting residual confounding and FFQ classification limitations. Importantly, effect sizes for individual components were modest, with hazard ratios close to 1.00, suggesting that overall dietary inflammatory patterns are more informative than single food groups. These findings support the use of diet quality indexes such as the CDII for evaluating the cumulative dietary effects on metabolic health.

This study has several strengths. First, to our knowledge, this study provides prospective evidence on the association between an inflammation-based dietary index and incident MetS in the Korean population, a context that has been relatively

underexplored. In addition, this study evaluated both the overall dietary inflammatory score and individual dietary components, providing a more detailed relationship. Nevertheless, several limitations should be acknowledged. First, the observational design limits causal inference, and randomized trials are needed to test the effect of anti-inflammatory compared with pro-inflammatory diets on cardiometabolic outcomes. Second, dietary intake was assessed using a single baseline FFQ, which may not fully capture changes in diet over time. Third, participants lost to follow-up had higher baseline CDII than those retained (Supplementary Table 3), which may have attenuated the observed associations. Finally, residual confounding from unmeasured variables cannot be fully excluded despite extensive adjustment for sociodemographic and behaviors factors. Despite these limitations, this study provides valuable evidence supporting that dietary inflammatory potential is associated with the development of MetS. Future studies should incorporate repeated dietary assessments and compare CDII with other dietary inflammatory indexes using standardized metrics such as C-statistics or net reclassification improvement. In addition, studies with improved follow-up retention and more comprehensive measurement of potential confounders may help reduce bias and residual confounding. Such efforts will be important to clarify the role of dietary inflammatory indexes in MetS prevention.

5. Conclusions

This prospective study demonstrated that higher dietary inflammatory potential, measured by the CDII, was associated with an increased risk of MetS, particularly abdominal obesity and hypertriglyceridemia, in Korean adults. Although individual CDII components such as SSBs and low-fat dairy showed statistically significant associations, their modest effect sizes suggest that the overall dietary inflammatory profile is more relevant to metabolic health than any single dietary component. These findings support dietary strategies that emphasize improving overall dietary quality and reducing pro-inflammatory dietary patterns for MetS prevention and management.

Author contribution

Conceptualization, H.K. and D.S.; Data curation, S.H.K., H.K., and D.S.; Formal analysis, S.H.K., H.K., and D.S.; Funding acquisition, D.S.; Methodology, S.H.K., H.K., and D.S.; Project administration, D.S.; Resources, D.S.; Supervision, D.S.; Visualization, S.H.K., H.K., and D.S.; Writing - original draft, S.H.K.; Writing - review & editing, H.K., D.S., and C.M.R. All authors read and approved of the final manuscript. All authors critically reviewed and revised the manuscript for important intellectual content, approved the final version for publication, and agree to be accountable for all aspects of the work, ensuring appropriate investigation and resolution of any questions regarding accuracy or integrity. No one meeting the authorship criteria has been excluded from the author list.

Data availability

The data utilized in this study originated from the Ansan-Ansung cohort within the KoGES, administered by the Korea Disease Control and Prevention Agency (KDCA). For ethical and legal reasons related to participant confidentiality, the KDCA restricts public access to these data. Interested researchers can apply for access to the de-identified KoGES data by submitting a formal request accompanied by institutional ethical approval to the KDCA.

Institutional Review Board statement

The study protocol was reviewed and approved by the Institutional Review Board of Ewha Womans University, Korea (institutional review board number ewha-202603-0023-01). Written informed consent was obtained from all participants. This study was conducted using bioresources from the National Biobank of Korea and Korea Disease Control and Prevention Agency, Republic of Korea (NBK-2024-019).

Declaration of Generative AI and AI-assisted technologies in the writing process

In developing this manuscript, the authors utilized ChatGPT (GPT-5, OpenAI, 2025) for grammatical review and enhancement of language expression. Following the use of this tool, the authors thoroughly examined and modified the content where necessary and assume complete responsibility for the published work.

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Conflict of interest

The authors declare no conflicts of interest.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.clnu.2026.106697.

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