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The dietary inflammatory index and insulin resistance or metabolic syndrome in young adults

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ABSTRACT

Objective: The aim of this study was to assess the association between the inflammatory potential of diet, as measured by the dietary inflammatory index (DII) and insulin resistance (IR) or metabolic syndrome (MetS).

Methods: A cross-sectional study (nested within a cohort) was conducted on 2017 adults 23 to 25 y of age in Ribeirão Preto, Brazil. Food consumption was assessed using a validated food frequency questionnaire. DII scores were calculated from 35 available food parameters. IR was determined from the classification of homeostatic model assessment (HOMA) values (≥ 2.7 uU mL⁻¹). MetS was diagnosed based on the Joint Interim Statement (JIS) criterion. The association of DII score with IR or MetS was determined by Poisson regression analysis. The variables included in the multivariable model were selected from directed acyclic graphs.

Results: The diet of the young adults studied showed a high inflammatory potential, with a mean DII score of +1.10 (range: -4.69 to +5.28). The prevalence of MetS was 12.2% and IR 12.3%; both were higher in men than in women. The correlation between DII and HOMA-IR values was -0.038 ($P=0.09$). The DII was not associated with IR or MetS in either sex.

Conclusion: Although the association between DII and the outcomes was not detected in this sample, the study demonstrated that the diets of these young adult Brazilians had a high inflammatory potential when compared with other studies. Future studies, preferably using longitudinal designs, are recommended.

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Introduction

The worldwide increase in the prevalence of non-communicable diseases and disorders (NCDD) has motivated studies searching for the causes of these diseases to identify preventive measures.

Insulin resistance (IR) is a metabolic disorder characterized by reduced sensitivity to the action of insulin on tissues [1]. Several studies have indicated a role of chronic inflammation in the pathogenesis of IR [2–4]. In turn, IR is considered to be an important causative factor of metabolic syndrome (MetS), acting as a link to all other metabolic abnormalities that characterize this condition [2,5].

MetS consists of a set of changes including disorders of glucose metabolism, abdominal obesity, dyslipidemia, and increased blood pressure [6]. Chronic inflammation also interferes with the genesis of MetS, acting via IR and abdominal obesity [7–9]. Thus, a chronic inflammatory state may be a common cause of the development of IR and MetS.

Several studies have suggested a role of diet in the regulation of chronic inflammation [5,10–14]. The release of anti-inflammatory cytokines may be stimulated according to diet composition, contributing to the prevention of NCDD [5,10–14]. The dietary inflammatory index (DII) has been created to characterize the inflammatory potential of the diet [15]. The DII is a score obtained from individual food intake based on the pro- or anti-inflammatory properties of foods, nutrients, or food constituents with evidence showing an effect on inflammatory markers [16].

Some studies have explored the association between DII and MetS; however, the results are still inconclusive [17–21]. One cohort study has demonstrated the existence of an association between DII and the development of MetS [20]. However, three cross-sectional studies [17,18,22] and two other cohort studies [19,21] have not detected this association. IR is considered a main precursor of MetS; however, the association between DII and IR has received little attention and the relationship is still unclear. One study with a cross-sectional design [22] and another cohort study [19], using the current DII, were conducted and did not detect any association. On the other hand, van Woudenberg et al. [23], using an adapted DII in a cross-sectional study, demonstrated an association between DII and IR. All of these studies investigated these outcomes in older adults, including the elderly. However, it is known that deterioration of the quality of the diet of the population, as well as the onset of NCDD, are occurring increasingly earlier [24,25].

Thus, we hypothesized that a proinflammatory diet may promote an inflammatory state, which could lead to the occurrence of cardiovascular risk factors such as IR and MetS even in younger individuals in Brazil. There are few studies about this association in middle-income countries such as Brazil and the nutrition transition is known to occur in different ways across high-, middle-, and low-income countries [26]. It is well known that dietary factors can interact with aspects of socioeconomic status [27] and Brazil is known to have concentrations of food insecurity and poor dietary quality with a transition toward processed foods that may exacerbate problems with IR and MetS [28–30]. Thus, the objective of the present study was to assess the inflammatory potential of the diet and the association between DII and IR or MetS in a population of young Brazilian adults.

Materials and methods

Study population

A cross-sectional study was conducted on 2017 young adults 23 to 25 y of age who are part of the fourth follow-up period of the Epidemiologic-Social Study of Perinatal Health in Ribeirão Preto, São Paulo, Brazil cohort study, carried out from 2002 to 2004.

The city of Ribeirão Preto, located 320 km from the capital of the state of São Paulo, in the southeast region of Brazil (the richest in the country), had 527,733 inhabitants in 2003. It is one of the most developed cities in the country with a monthly per capita income of R\$1070.28 (US\$339.09) and a human development index of 0.733 in 2000 [31,32].

A total of 9067 newborns delivered at all the hospitals in Ribeirão Preto between June 1978 and May 1979 were evaluated in the first part of the study, representing 98% of all births. Children whose families did not reside in the municipality (2094 babies) and twins (146 babies) were excluded, resulting in 6827 newborns [33].

Of these, 343 died before completing 20 y of age. Thus, 6484 individuals were potentially eligible in the fourth phase of the study. The search for these individuals was based on the records of the Unified Health System and private health plans

and on contacts obtained during the second and third phases of the study, permitting us to locate 5665 adults. For data collection, the city of Ribeirão Preto was divided into four regions according to family head income. Next, it was decided to interview one-third of the potentially eligible individuals, corresponding to 2161 participants. Thus, one of each three individuals belonging to the cohort in each region was invited to participate in the follow-up. In instances when an individual refused to participate in the study or was not located, the next participant on the list was contacted. There were 209 refusals, 34 deaths after 20 y of age, 31 individuals who were not interviewed because they were in jail, and 431 who did not attend the interview. Thus, 2063 adults were evaluated [33].

The sample size was calculated considering the prevalence of the studied outcomes of 13%, accuracy of 1% and the design effect of 2. The sample size required for a 99% confidence level was 987 individuals.

Two participants were excluded from the study because they did not complete the food frequency questionnaire (FFQ). An additional 44 participants were excluded because their energy intake was outside the ± 3 standard deviation (SD) limit. Thus, the final study sample consisted of 2017 adults ages 23 to 25 y. More details about the study are available elsewhere [33].

This study was approved by the ethics committee of the Faculdade de Medicina de Ribeirão Preto and performed in accordance with the ethical standards. All participants gave their informed consent before their inclusion in the study.

Food intake and DII calculation

The DII was used to assess the inflammatory potential of the diet [16]. The DII is a score that ranges from low values (minimum –8) that represent an anti-inflammatory diet to high values (maximum +8) that indicate a proinflammatory diet. This index was created based on an extensive literature review to assess the effect of food parameters (foods/nutrients) on the inflammatory markers interleukin (IL)-1 β , IL-4, IL-6, IL-10, tumor necrosis factor (TNF)- α , and C-reactive protein. The score of the inflammatory effect of each of 45 food parameters identified in the search was calculated on the basis of this review [16].

To avoid obtaining arbitrary values owing to the use of crude food intake quantities with different measuring units, a regionally representative database was created with the food intake representative of the populations of 11 countries (United States, Bahrain, Australia, Denmark, India, Japan, Zealand, Taiwan, South Korea, Mexico, United Kingdom). The food intake data of the present study were standardized on the basis of the mean and SD of each food parameter of the DII originating from this database. The standardized values of each food parameter were then converted to centered percentiles and multiplied by their respective inflammatory effect scores calculated from the literature review regarding the DII, resulting in the final DII for each food parameter. Finally, the total DII was calculated from the sum of the final DII value for each food parameter. More details about the method for the calculation of the DII can be obtained from Shivappa et al. [16].

Food intake of the study sample was assessed using a 75-item FFQ validated for the Japanese-Brazilian community of São Paulo, with food of Japanese origin being excluded. The reproducibility of the FFQ and the relative validity were evaluated by repeated administration at 1-mo intervals and by comparison with dietary intake obtained from four 3-d weighed dietary records, respectively [34]. Participants were asked to refer to their habitual dietary intake during the 12 mo preceding data collection [35].

The food intake data were evaluated based on tables of national food composition such as the Survey of Family Budgets and the Brazilian Table of Food Composition (TACO). To assess some DII parameters that were not available in Brazilian tables, such as caffeine and alcohol, we used the North American food composition table (U.S. Department of Agriculture). In view of unavailable quantitation of flavonoids in the above tables, we consulted the study by Santos [36].

In this study, 35 food parameters were used to calculate the DII: folic acid; monounsaturated fatty acids (MUFAs); polyunsaturated fatty acids (PUFAs); alcohol; caffeine; carbohydrates; black tea; cholesterol; energy; iron; fibers; flavonol; flavones; isoflavones; flavonones; flavon-3-ol; anthocyanidins; saturated fat; total fat; trans fat; magnesium; niacin; ω -3; ω -6; proteins; riboflavin; selenium; thiamine; and vitamins A, B₆, B₁₂, C, D, E, and zinc. The food parameters oregano, rosemary, onion, garlic, ginger, saffron, curcumin, pepper, β -carotene, and eugenol were not included because they were not part of the FFQ used in the study, in the food composition tables, or both.

The DII was categorized into tertiles. Negative DII values indicated that the diet had a low inflammatory potential and positive values indicated a high inflammatory potential [16].

Metabolic syndrome and insulin resistance

A blood sample was collected from each participant aseptically by a doctor or nursing technician after a fast of ≥ 12 h. Fasting glycemia was determined by the GOD/PAP human diagnostic colorimetric enzymatic method (Chronolab AG, Zug, Switzerland). Fasting insulin was measured by radioimmunoassay (insulin kit, DPC, Los Angeles, CA, USA). Total cholesterol (TC), high-density lipoprotein

cholesterol (HDL-C), and triacylglycerols (TGs) were determined by an enzymatic colorimetric method using the Dade Behring XPand apparatus (Dade Behring, Liederbach, Germany) and reagents of Dade Behring Dimension clinical chemistry [33]. More details about the techniques used to obtain these measurements have been described by Barbieri et al. [33].

The diagnosis of MetS was established according to the Joint Interim Statement (JIS) criterion based on the presence of at least three of the following criteria:

- fasting glucose ≥ 100 mg/dL;
- blood pressure ≥ 130 mm Hg or ≥ 85 mm Hg;
- HDL-C < 40 mg/dL for men and < 50 mg/dL for women;
- TGs ≥ 150 mg/dL;
- waist circumference ≥ 90 cm for men and ≥ 80 cm for women [37].

IR was determined by HOMA-IR using the formula:

$$\text{HOMA-IR} = \text{insulin } (\mu\text{U/mL}) \times (\text{glycemia mg/dL} \div 18) \div 22.5.$$

An IR diagnosis was considered when HOMA-IR was > 2.7 [38].

Covariables

The level of physical activity over the previous week was determined using the International Physical Activity Questionnaire (IPAQ) [39]. The IPAQ was validated for use in Brazilians adults by means of a movement sensor Computer Science & Applications and the reproducibility was determined 7 d later. The individuals were classified as sedentary, sufficiently active, or active [40].

Daily total energy value and alcohol consumption were calculated using the information from the FFQ and the amount of kcal/d was calculated. Alcohol intake was expressed in g/d. Family income, schooling, smoking status, and sex were obtained using semistructured questionnaires.

The directed acyclic graph (DAG) was used to select the confounding factors that would be included in the adjusted analysis. The DAG is a causal diagram that illustrates theoretical models by graphically representing relationships between variables and by applying heuristic rules. This method was used because it permits selection of the variables that need to be adjusted in the analysis to minimize the magnitude of confounding bias; it also helps to avoid overadjustment of the variables [41,42].

Figure 1 illustrates the DAG representing the DII as the exposure variable and MetS and IR as outcomes. The factors causing MetS and IR were considered to be the same, so that it is possible to assume a single DAG for both outcomes [6]. IR is considered to be an event that precedes the development of MetS [6,9], so that it would act as a mediator in the relationship between DII and MetS, requiring no adjustment in the analysis. Again, for the study of the association between DII and IR, it is not necessary to adjust for MetS because MetS is a consequence of IR. In the present study, obesity was measured by means of the body mass index and was

not included in the adjustment because it is not correct to adjust for a variable that mediates the outcomes under study.

Based on the proposed DAG, the minimum set of variables needed to be adjusted for the control of confounding included age (y), family income in multiples of the monthly minimum wage (tertiles), and schooling (0–4, 5–8, 9–11, and ≥ 12 y).

Statistical analysis

The data were analyzed statistically using Stata software, version 14 (Stata, College Station, TX, USA). The distribution of the variables was described using boxplot graphs, a histogram, and by examining kurtosis and asymmetry values. According to their distribution, the variables were presented as means or medians followed by SD and range.

Because of the occurrence of follow-up losses inverse probability of selection weighting was performed. The purpose of this procedure is to reestablish the initial composition of the sample at the baseline. The groups that had lower follow-up rates were given higher weights, whereas groups with higher follow-up rates were given lower weights.

Socioeconomic, lifestyle, and metabolic variables were compared within tertiles of the DII using the χ^2 test. Analysis of variance was used to compare the means and the nonparametric Kruskal–Wallis test was used to compare the medians. To evaluate the variables distribution, we analyzed boxplots and histograms, values of skewness and kurtosis, and applied the Shapiro–Wilk test.

Nutrients, HOMA-IR, and family income that did not show normal distribution were transformed into logarithms or square-root values. After this, nutrients were adjusted for energy by the residual method [43]. After analysis, the variables were converted to their original units of measurement for the purpose of presenting the results.

The correlation between DII and HOMA-IR values was determined using the Pearson correlation test and the association of DII with IR or MetS was determined using Poisson regression with robust variance. In the multivariate analysis, the variables pointed out by DAG were included for confounding control and selection bias. The analysis of the association between DII and isolated components of MetS (blood pressure, waist circumference, TGs, and HDL) also was performed. The level of significance was set at 0.05 in all analyses.

This study was approved by the Research Ethical Committee and all participants signed a written consent form and received a copy for their records.

Results

The mean age of the participants in the present study was 23.9 y and 53.1% were women. Their DII ranged from -4.69 to 5.28 , with a mean of $+1.10$. The mean values were $+0.74$ (± 1.72) for men and $+1.42$ (± 1.83) for women. The overall prevalence of IR was 12.3%

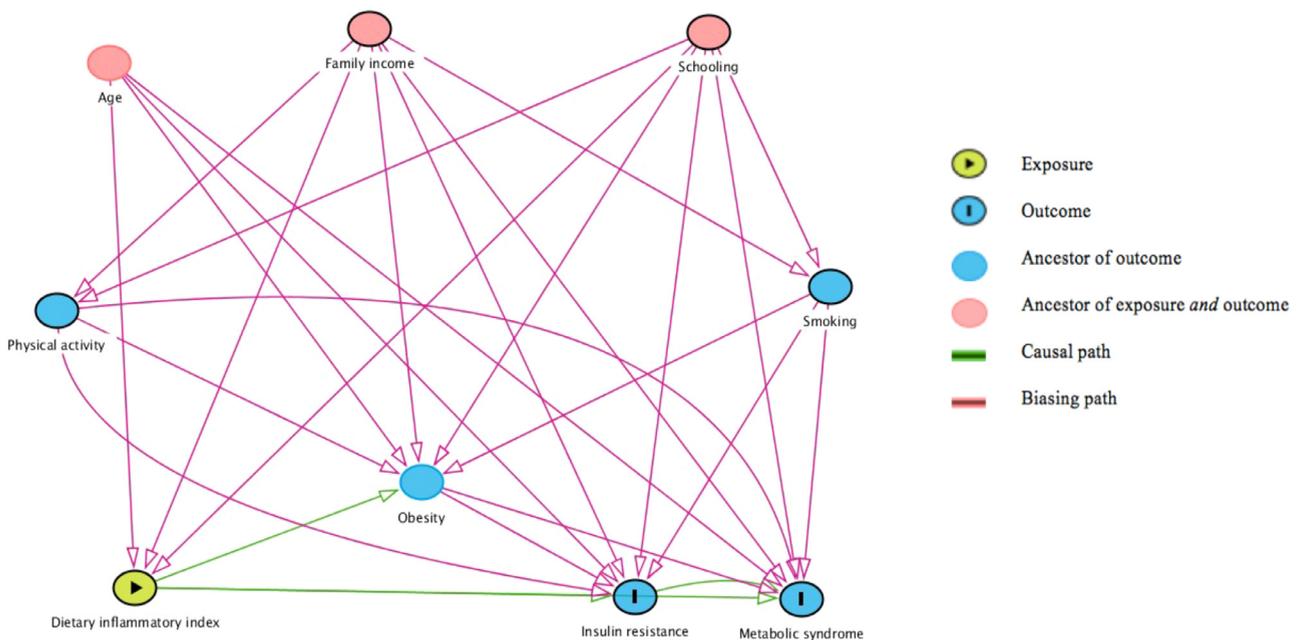


Fig. 1. Directed acyclic graph illustrating the relationship between dietary inflammatory index and insulin resistance or metabolic syndrome.

(14.1% for men and 10.7% for women). The prevalence of MetS was 12.2% (18.9% for men and 6.3% for women).

Table 1 presents the socioeconomic, lifestyle, and metabolic characteristics by DII tertile according to sex. Among men, there was a higher proportion of active individuals ($P < 0.001$) in the lowest DII tertile (anti-inflammatory) and individuals with lower levels of education ($P = 0.012$) in the second tertile. Among women, the prevalence of sedentary lifestyle ($P < 0.001$) was higher among those who belonged to the higher DII tertile (proinflammatory).

The mean daily intake of the food parameters used in calculating the DII is presented in **Table 2**. Among men, higher consumption of the food parameters was observed in those in the lowest (most anti-inflammatory) DII tertile, except for total ($P < 0.001$), saturated ($P < 0.001$), trans ($P < 0.001$), and monounsaturated fats ($P < 0.001$). Similarly, among women, most of the dietary parameters were most consumed by those belonging to the lowest DII tertile, except total ($P < 0.001$), saturated ($P < 0.001$), trans ($P = 0.025$), and monounsaturated fats ($P < 0.001$), which were most consumed in the higher DII (proinflammatory) tertile.

The correlation between DII and HOMA-IR values was $r = -0.005$ ($P = 0.87$) for men and $r = -0.040$ ($P = 0.19$) for woman. Adjusted analysis for confounding variables did not reveal an

association of the increase in DII with IR among men (prevalence ratio [PR], 0.98; 95% confidence interval [CI], = 0.89–1.08; $P = 0.76$) or women (PR, 0.96; 95% CI, 0.87–1.07; $P = 0.48$). Similarly, the increase in DII was not associated with MetS among men (PR, 0.98; 95% CI, 0.91–1.07; $P = 0.67$) or women (PR, 1.05; 95% CI, 0.91–1.20; $P = 0.53$; **Table 3**). The DII and the isolated components of MetS also were not associated (data not shown).

Discussion

No association was detected in the present study between DII scores and IR or MetS among young adults. However, comparing with other studies that investigated the relation between DII and metabolic outcomes [17,19,20,22,44], the mean DII score was high in this sample.

Only one cohort study detected an association between DII and MetS among French individuals, who were 48.9 y of age, on average, and who were followed up over a mean period of 12.4 y [20]. All other studies [17,18,22], with a cross-sectional design and conducted on significantly older populations than the present one did not detect an association. Similarly, Alkerwi et al. [22], in a study of Luxembourg adults, did not detect a significant association

Table 1
Socioeconomic, metabolic and lifestyle characteristics according to the tertiles of the dietary inflammatory index for each sex, Ribeirão Preto, 2002 to 2004

	Tertiles of DII									
	Men					Women				
	Total	T1	T2	T3	P-value	Total	T1	T2	T3	P-value**
DII* (score mean)	957	-0.96	1.11	2.94	<0.01	1060	-0.93	1.19	3.17	<0.01
SD		0.93	0.49	0.71			0.95	0.51	0.81	
BMI* (kg/m ² mean)	954	24.89	24.88	25.22	0.62	1056	23.88	23.50	23.65	0.67
SD		4.39	4.31	4.45			4.97	5.30	4.91	
Family income* (mean)	798	4.45	4.85	5.06	0.18	836	4.38	4.41	5.25	0.06
SD		5.20	5.39	4.65			5.44	5.30	5.91	
Schooling (y) (%)					0.01					0.68
≥12	312	35.2	32.2	32.6		385	24.3	31.0	44.7	
9–11	490	42.6	34.4	23.1		520	28.2	33.9	37.9	
5–8	130	42.6	39.1	18.4		125	29.3	31.7	39.1	
0–4	25	28.0	58.1	13.8		30	23.4	36.7	39.9	
Physical activity (%)					<0.01					<0.01
Active	400	43.1	37.7	19.3		221	34.7	31.3	34.1	
Sufficiently active	156	43.3	35.5	21.2		232	34.1	31.8	34.0	
Sedentary	400	34.9	33.0	32.1		602	21.3	33.9	44.8	
Smoking (%)					0.37					0.42
No	767	40.5	34.2	25.3		914	26.2	32.6	41.2	
Yes	190	37	39.9	23.2		146	30.8	33.7	35.5	
HDL-C (%)					0.27					0.98
Normal	584	37.8	36	26.2		596	27.1	32.4	40.5	
Low	373	43	34.4	22.6		464	26.7	33.1	40.2	
Triacylglycerols (%)					0.07					0.08
Normal	797	38	36.7	25.3		939	26.5	32.2	41.3	
High	150	48.4	28.3	23.3		103	31.6	38.8	29.6	
Glycemia (%)					0.66					0.38
Normal	905	40.1	35.3	24.6		1022	26.7	32.9	40.4	
High	48	33.7	36.8	29.5		22	39.8	23	37.2	
Blood pressure (%)					0.71					0.86
Normal	562	39.4	36.5	24.1		990	26.8	32.6	40.6	
High	394	40.6	33.8	25.6		68	27.8	35.1	37.1	
Waist circumference (%)					0.50					0.58
Normal	607	41	35.4	23.6		760	26.2	33.6	40.2	
High	350	37.7	35.3	27.0		300	28.8	30.5	40.7	
Insulin resistance (%)					0.23					0.59
No	808	39.2	36.7	24.1		924	26.7	32.6	40.7	
Yes	134	43.1	28.7	28.2		110	30.5	33.6	35.9	
Metabolic syndrome (%)					0.75					0.58
No	766	39.2	36.0	24.8		969	26.9	32.5	40.6	
Yes	180	41.8	33.0	25.2		67	24.5	38.8	36.7	

DII, dietary inflammatory index; BMI, body mass index; HDL-C, high density lipoprotein cholesterol.

*Difference between tertiles.

**Significance for values $P < 0.05$.

Table 2
Distribution of food parameters into DII tertiles according to sex, Ribeirão Preto, 2002 to 2004.

Nutrients	Tertiles of DII									
	Men					Women				
	Mean					Mean				
	T1	T2	T3	P-value	T1	T2	T3	P-value		
Energy (kcal/kg/d)	38.71	30.87	24.19	<0.01	42.19	34.80	26.81	<0.01		
Carbohydrates (g)	287.60	283.60	276.98	0.01	296.70	290.51	284.90	<0.01		
Proteins (g)	90.90	88.98	85.20	<0.01	88.32	85.66	83.10	<0.01		
Total fat (g)	65.32	67.95	72.57	<0.01	62.43	66.36	69.99	<0.01		
Saturated fat (g)	23.73	25.36	27.89	<0.01	22.62	24.69	26.46	<0.01		
Trans fat (g)	0.79	0.83	0.85	<0.01	0.83	0.85	0.86	0.03		
Cholesterol (mg)	258.00	258.63	243.84	0.14	246.89	258.28	245.84	0.27		
MUFA (g)	22.42	23.74	25.72	<0.01	21.44	23.06	24.60	<0.01		
PUFA (g)	12.34	12.21	11.54	<0.01	11.93	11.81	11.34	0.02		
ω-3 (g)	1.56	1.55	1.52	0.358	1.50	1.49	1.45	0.12		
ω-6 (g)	10.32	10.32	9.77	0.01	10.04	10.05	9.72	0.17		
Fibers (g)	24.58	20.88	17.00	<0.01	26.94	22.08	17.90	<0.01		
Thiamine (mg)	1.51	1.40	1.36	<0.01	1.57	1.47	1.38	<0.01		
Riboflavin (mg)	1.71	1.58	1.54	<0.01	1.74	1.62	1.56	<0.01		
Niacin (mg)	16.83	16.57	15.78	0.01	16.95	16.36	15.13	<0.01		
Pyridoxine (mg)	1.55	1.33	1.19	<0.01	1.61	1.37	1.25	<0.01		
Folic acid (μg)	423.93	373.35	343.31	<0.01	434.94	377.15	342.19	<0.01		
Vitamin B ₁₂ (μg)	3.24	3.23	3.19	0.86	3.24	3.15	3.10	0.41		
Vitamin A (μg)	417.36	350.23	305.45	<0.01	482.36	412.79	357.51	<0.01		
Vitamin C (mg)	162.93	98.16	72.33	<0.01	199.90	130.70	93.63	<0.01		
Vitamin D (μg)	2.82	2.66	2.49	0.03	2.97	2.66	2.57	0.01		
Vitamin E (mg)	5.55	4.75	4.03	<0.01	6.03	5.05	4.27	<0.01		
Iron (mg)	12.20	12.23	11.19	<0.01	12.06	11.71	10.66	<0.01		
Zinc (mg)	11.36	11.40	10.96	0.01	11.26	11.08	10.70	0.01		
Selenium (μg)	106.05	100.65	92.04	<0.01	97.68	96.82	87.03	<0.01		
Magnesium (mg)	275.60	251.67	213.18	<0.01	287.51	251.90	210.95	<0.01		
Flavonoids (mg)	41.28	39.36	29.59	<0.01	40.15	32.57	25.22	<0.01		
Flavones (mg)	0.97	0.62	0.49	<0.01	1.21	0.83	0.55	<0.01		
Flavonones (mg)	23.57	13.19	8.84	<0.01	26.61	17.34	13.27	<0.01		
Flavanols (mg)	16.75	8.99	6.51	<0.01	16.86	14.80	10.78	<0.01		
Anthocyanidins (mg)	32.97	19.96	14.73	<0.01	39.87	30.31	18.06	<0.01		
Caffeine (g)	52.48	48.84	46.05	0.25	44.68	47.62	42.07	0.36		
Alcohol (g)	9.40	7.57	7.14	0.01	5.81	4.81	4.04	0.03		
Black tea (g)	107.06	55.97	39.29	0.01	90.05	77.87	56.39	0.09		

DII, dietary inflammatory index; MUFA, monounsaturated fatty acid; PUFA, polyunsaturated fatty acid.

between DII and IR. Van Woudenberg et al. [23], using an adapted DII, detected the presence of an association between DII and IR in sample of adult Dutch individuals, including elderly. However, we would like to point out that the adaptations made in the DII used by Van Woudenberg et al. [23], such as the exclusion of some foods that were present in the original DII and the adjustment of nutrients according to energy before calculating the final DII, impair a comparison with the present study.

The prevalence of MetS in the present study was lower (12.2%) than in other studies that evaluated the association of this outcome with the DII. Alkerwi et al. [22], studying a sample of adults and the elderly with a mean age of 44 y, found a prevalence of 26.2%. In Poland, Sokol et al. [17] found a prevalence of 30% of MetS among adults aged 45 to 64 y. It should be considered that these studies included populations older than those in the present study, which helps explain the higher prevalence.

Table 3
Crude and adjusted analysis of the association between insulin resistance or metabolic syndrome with the dietary inflammatory index, Ribeirão Preto, 2002 to 2004

	Crude PR	Men		Crude PR	Women	
		CI 95%	P-value		CI 95%	P-value
Insulin resistance DII (unit)	0.97	0.88–1.07	0.55	0.95	0.86–1.05	0.29
Insulin resistance DII (unit)	Adjusted PR ^a	CI 95%	P-value	Adjusted ^a PR	CI 95%	P-value
	0.98	0.89–1.08	0.76	0.96	0.87–1.07	0.48
Metabolic syndrome DII (unit)	Crude PR	CI 95%	P-value	Crude PR	CI 95%	P-value
	0.98	0.91–1.07	0.68	1.04	0.91–1.20	0.57
Metabolic syndrome DII (unit)	Adjusted PR ^a	CI 95%	P-value	Adjusted ^a PR	CI 95%	P-value
	0.98	0.91–1.07	0.67	1.05	0.91–1.20	0.53

CI, confidence interval; DII, dietary inflammatory index; PR, prevalence ratio

^aAdjusted by age, family income in multiples of the month minimum wage and schooling.

The no association of DII with MetS and IR in the present study may be related to the fact that risk factors for chronic diseases act over a long period of time until they culminate in the development of the disease [45,46]. Therefore, it is possible that the effect of a proinflammatory diet in promoting these outcomes has not yet had time to manifest itself.

The occurrence of these diseases in the sample studied may be associated with several other mechanisms, in addition to diet, that influence the occurrence of inflammation and may also cause these outcomes [47,48].

Originally, the DII was validated in a sample of US individuals with a mean age of 49 y in SEASONS (Seasonal Variation of Blood Cholesterol Study) [15]. Subsequently, it was validated in three additional populations in the United States: In the Women's Health Initiative [49], in a population of African-Americans in South Carolina [18] and in NHANES (National Health and Nutrition Examination Study) [50]. It also was subjected to construct validation in three studies conducted among adults in Europe (i.e., in Belgium [51] and France [44]) and among children in Greece, Germany, Belgium, France, Austria, and Spain [44,52]. When testing the validity of this index in a population of adults in Luxembourg, Alkerwi et al. [22] did not detect an association between CRP levels and DII scores, which made it the only study to date in which a statistically significant association was not observed. Without access to data on inflammatory markers for validation in the present study we could not verify its validity in this population.

DII scores in the current sample ranged from -4.69 to $+5.28$, with a mean of $+1.07$, indicating the proinflammatory characteristic of the diet. This result indicates that the diet is harmful to health, with the predominance of food parameters that promote inflammation. In the original validation study [15], DII scores ranged from -5.3 to $+4.3$, with a mean of $+0.84$, thus revealing a lower inflammatory potential than that observed in the present sample. Among all the studies that evaluated MetS and IR as outcomes, the present one showed the most proinflammatory mean value. It has been demonstrated that the food intake of younger people, characterized by high consumption of saturated fat, sodium, and sugar and low consumption of whole foods, fruits, and vegetables, is less healthy than that of older people [53–56]. This may help explain the more proinflammatory diet observed in the present sample.

Regarding the high mean DII of the sample, it should be pointed out that the 10 parameters not estimated were of an anti-inflammatory nature. Despite this, many of the non-estimated parameters are foods used as seasonings, consumed in lesser amounts in the diet, such as turmeric, saffron, and rosemary. The difficulty in quantifying these parameters is common in studies that use the FFQ as the assessment method because spices are not normally listed in the questionnaire [15].

The present study had some limitations. First, its cross-sectional design did not allow us to establish a temporal relationship between the independent variables and the outcomes analyzed. Indeed, reverse causation may obscure a real association. The effect of diet on the occurrence of chronic outcomes is more likely to be evidenced in studies with longitudinal design. Second, the DII consisted of 45 food parameters, but only 35 were used in the present study because some parameters were not included in the FFQ or were not available in the food composition tables. However, studies that did not assess parameters similar to those missing in the present study have suggested that the reduction in the number of parameters does not compromise the discriminatory potential of the DII [15,49]. Nevertheless, the FFQ is commonly used to assess the association between dietary intake and NCDD because it evaluates the habitual food intake of the sample [57].

Despite its weaknesses, the present study had strengths that require mention. These included its large sample size and the population-based representation. The use of the DAG for the construction of the theoretical model and the use of the backdoor criterion to select a minimal set of adjustments for confounding factors prevented unnecessary adjustment in multivariable analysis. Further, to our knowledge this was the first study to assess the association between the DII and IR or MetS in young adults of a middle-income country, like Brazil. Although it did not demonstrate such an association, it revealed that the average diet of the young adults studied has a high inflammatory potential when compared with other studies. Future studies, preferably using longitudinal designs, are recommended to further test the hypothesis that diet-associated inflammation is associated with IR or MetS.

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Conflict of interest

Declarations of interest: none.

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