

DIETARY INFLAMMATION INDEX AND ASSOCIATION WITH INFLAMMATORY PROFILE IN INDIVIDUALS WITH OBESITY: A CROSS-SECTIONAL STUDY

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ABSTRACT

Dietary patterns are critical in maintaining an inflammatory state, particularly in obesity, which is linked to chronic inflammation and heightened risks of metabolic and cardiovascular conditions. The dietary inflammatory index (DII) was developed to assess diet-related inflammation and its effects on inflammatory markers. This study examined the correlation between DII scores and systemic inflammatory profiles in individuals with obesity. A cross-sectional study included 35 patients from an obesity treatment center. The DII was calculated using 28 dietary components derived from 24-hour recalls. Blood samples were analyzed for biomarkers (IL-4, IL-6, IL-10, IL-17, IFN- γ , TNF- α , and leptin). Statistical analyses included the Mann-Whitney test and Spearman's correlation. The participants had a mean age of 34 years; 71.4% had severe obesity, 25.7% had hypertension, and 8.6% had diabetes. The median DII was +0.18 (range: -2.15 to +2.08). The anti-inflammatory diet group (ANTI-DII) showed lower IL-6 levels compared to the pro-inflammatory group (31.2 pg/ml vs. 36.8 pg/ml; $p=0.034$). Sensitivity analysis excluding diabetics confirmed these findings ($p=0.023$) and revealed differences in IL-17 levels (52.8 pg/ml vs. 52.78 pg/ml; $p=0.049$). Positive correlations were observed between IL-4 and legume intake ($p<0.01$) and IFN- γ and grain intake ($p<0.05$). These results suggest dietary patterns influence inflammatory profiles in obesity, highlighting the role of diet in managing inflammation and related health risks.

Key words: Obesity. Biomarkers. Dietary intake. Inflammation. Interleukin.

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RESUMO

Índice de inflamação dietética e associação com o perfil inflamatório em indivíduos com obesidade: um estudo transversal

Os padrões alimentares são cruciais para a manutenção de um estado inflamatório, particularmente na obesidade, que está associada à inflamação crônica e a riscos aumentados de doenças metabólicas e cardiovasculares. O Índice Inflamatório da Dieta (IID) foi desenvolvido para avaliar a inflamação relacionada à dieta e seus efeitos sobre marcadores inflamatórios. Este estudo examinou a correlação entre os escores do IID e os perfis inflamatórios sistêmicos em indivíduos com obesidade. Um estudo transversal incluiu 35 pacientes de um centro de tratamento da obesidade. O IID foi calculado utilizando 28 componentes dietéticos derivados de recordatórios alimentares de 24 horas. Amostras de sangue foram analisadas para biomarcadores (IL-4, IL-6, IL-10, IL-17, IFN- γ , TNF- α e leptina). As análises estatísticas incluíram o teste de Mann-Whitney e a correlação de Spearman. Os participantes tinham uma idade média de 34 anos; 71,4% apresentavam obesidade grave, 25,7% hipertensão e 8,6% diabetes. A mediana do DII foi de +0,18 (variação: -2,15 a +2,08). O grupo com dieta anti-inflamatória (ANTI-DII) apresentou níveis de IL-6 mais baixos em comparação ao grupo com dieta pró-inflamatória (31,2 pg/ml vs. 36,8 pg/ml; $p=0,034$). A análise de sensibilidade, excluindo os diabéticos, confirmou esses achados ($p=0,023$) e revelou diferenças nos níveis de IL-17 (52,8 pg/ml vs. 52,78 pg/ml; $p=0,049$). Correlações positivas foram observadas entre IL-4 e o consumo de leguminosas ($p<0,01$) e entre IFN- γ e o consumo de grãos ($p<0,05$). Esses resultados sugerem que os padrões alimentares influenciam os perfis inflamatórios na obesidade, destacando o papel da dieta no controle da inflamação e dos riscos à saúde associados.

Palavras-chave: Obesidade. Biomarcadores. Ingestão alimentar. Inflamação. Interleucina.

INTRODUCTION

Obesity is a predominantly chronic disease. More than one-third of the global population is classified as overweight or obese; therefore, it is considered a global epidemic (Chooi, Ding, Magkos, 2019).

It is a primary factor in the development of other chronic non transmittable diseases, such as cardiovascular diseases, diabetes, and cancer (Bray, Kim, Wilding, 2017).

The relationship between obesity and other diseases is related to a low-level chronic inflammatory state (Saltiel, Olefsky, 2017).

Chronic inflammation is directly related to an increase in adipose tissue. Adipose tissue comprises cells capable of secreting proinflammatory molecules such as adipokines. Moreover, infiltrated macrophages in adipose tissue contribute to the maintenance of a proinflammatory environment through the secretion of cytokines and adipokine stimuli.

However, the mechanisms underlying the interactions between these elements are not yet known (Antuna-Puente et al., 2008, Savulescu-Fiedler et al., 2024).

Pro-inflammatory and anti-inflammatory diets play a crucial role in modulating health outcomes, especially concerning chronic inflammation and related diseases. Diets high in processed foods, refined sugars, and unhealthy fats are typically pro-inflammatory, leading to elevated levels of inflammatory markers like C-reactive protein (CRP), as evidenced by recent studies (Mulligan, Lentjes, Welch, 2024; Chen et al., 2024; Norde et al., 2020).

In contrast, anti-inflammatory diets emphasize whole, nutrient-dense foods, primarily of plant origin, such as fruits, vegetables, and whole grains.

These dietary patterns are associated with lower levels of inflammation and improved health outcomes (Chen et al., 2024; Norde et al., 2020).

The impact of diet on inflammatory processes has been increasingly recognized, with diets high in red and processed meats, refined grains, sugary drinks, and ultra-processed foods correlating with pro-inflammatory biomarkers, while healthier eating patterns are linked to anti-inflammatory effects (Mulligan, Lentjes, Welch, 2024; Chen et al., 2024; Norde et al., 2020).

The diet inflammation evaluation stems from the Dietary Inflammatory Index (DII) (Shivappa et al., 2014).

The DII has already been associated with 27 adverse health outcomes (Marx et al., 2021).

In addition, studies have demonstrated that the DII can significantly predict the serum levels of inflammatory biomarkers (Chen et al., 2024; Norde et al., 2020; Shivappa et al., 2014; Marx et al., 2021; Shivappa et al., 2017).

Frequent overeating often involves consuming high-calorie, low-quality food. Both foods and beverages contain elements that can impact inflammatory pathways in peripheral tissues, altering the plasma concentration of inflammatory markers and affecting individuals' health.

A few authors have suggested that individuals with obesity who consume a more proinflammatory diet are more likely to have worse health-related outcomes, a greater probability of developing osteosarcopenia (Park, Na, Sohn, 2018), a worse hepatic function (Cantero et al., 2018), a worse cardiometabolic profile (Camargo-Ramos et al., 2017), a greater waist/hip ratio, and greater waist circumference (Ruiz-Canela et al., 2015), among others.

Given the persistent inflammatory environment observed in obesity and understanding the role of dietary factors, this study aims to investigate the association between dietary inflammatory index (DII) scores and inflammatory biomarkers in individuals with severe obesity, contributing valuable insights into managing obesity-related inflammation.

MATERIALS AND METHODS

Participants and study design

This was a cross-sectional study conducted by the Federal University of Health Sciences of Porto Alegre (UFCSA) and the Center for Obesity Treatment (CTO) of the Irmandade Santa Casa de Misericórdia de Porto Alegre (ISCOMPA).

The sample was selected using a convenience sampling method, patients being invited after verifying the inclusion criteria. Patients from an obesity treatment center were invited during their routine pre-surgical consultations, where patients were informed of the importance of the present study. The data

were collected after the patients self-authorized to participate.

There were patients of both genders, ages between ≥ 18 to ≤ 60 years old, with class II (BMI ≥ 35 kg/m² and >39.9 kg/m²) and III obesity (BMI ≥ 40 kg/m²). The exclusion criteria were chronic inflammatory disease, and pregnancy.

Interviews were conducted between August 2019 and March 2020 and involved in-person sessions. The data questionnaire applied included socio-economic questionnaires, 24-hour dietary recalls, and anthropometric measurements. On the same day, patients were invited to perform blood sample collection at UFCSPA. The research was approved by the ISCOMPA ethics committee through the Brazil platform under protocol number 5.281.452.

The sample size was determined using G*Power software (version 3.1.3, Universität Kiel, Kiel, Germany) to ensure sufficient statistical power for detecting significant associations between the Dietary Inflammatory Index (DII) and inflammatory markers in individuals with obesity. The calculation was based on prior research data (Min et al., 2020), which identified a moderate effect size of 0.577 for similar associations.

For this study, we set the following parameters: Effect size (f): 0.577, indicating a moderate association; Power (1 - β): 0.90 (90%), to reduce the likelihood of Type II errors; Significance level (α): 0.05, to maintain a 5% risk of Type I error; Allocation ratio: 1:1, assuming equal sample sizes in the two comparison groups (anti-inflammatory vs. pro-inflammatory diet). Using these parameters, the minimum required sample size was calculated to be 28 participants to detect statistically significant differences between groups.

To account for potential dropouts or incomplete data, we recruited 35 participants, exceeding the calculated sample size to ensure robust results.

All the patients who agreed to participate in the study signed a term authorizing their participation, which was a volunteer role. The present study is based on item IV of Resolution 196/96, which outlines the ethical guidelines for research involving human participants, ensuring their rights and well-being (Brasil, 1996). All the patients provided written informed consent, and the study was carried out in accordance with the Declaration of Helsinki.

Clinical data and laboratories

The clinical data and laboratory data (fasting glucose, glycosylated hemoglobin, total cholesterol, triglycerides, high-density lipoprotein (HDL)-cholesterol, and low-density lipoprotein (LDL)-cholesterol) were obtained from the patients' medical records in the obesity treatment center. The arterial pressure was collected during the interview conducted by a trained physician, and the patients' history of infection was self-reported.

Anthropometric Data

The individuals' weight and height were collected by a trained professional physician. Weight and height were measured on a calibrated Balmak scale with a maximum capacity of 300 kg, available for the study, with the stadiometer attached. The patient was instructed to take off their shoes and any excess clothing, such as coats or jewelry. The weight is reported in kg, and the height is reported in cm.

Food intake evaluation

The alimentary intake of the patients was evaluated via 24-hour dietary recall. The report was conducted using the US Department of Agriculture Automated Multiple-Pass Method (AMPM) method to avoid memory lapses and to obtain the maximum detail of the food and beverages consumed, therefore limiting the error of the dietary measurements. The AMPM includes a five-step dietary interview, which is as follows: 1: Quick List, to collect the foods consumed on the previous day. 2: Forgotten foods list, to elicit the recall of frequently forgotten items. 3: Time and occasion, to collect information on the time and occasion of each food-related event. 4: Detail and review, to collect a detailed description of each food and review eating occasions. 5: Final probe to provide a final opportunity to recall foods (Moshfegh et al., 2008).

The nutrient calculations (energy, macronutrients, and micronutrients) were performed using a food chemical compositional chart, utilizing the first option as the Brazilian Food Composition Table through the Brazilian Food Composition Table using the Dietbox® software, and the remaining data were checked via the USDA database.

Additionally, the foods were divided into food groups according to the grams consumed

by each individual, namely, legumes and vegetables, fruit, wholegrain cereals, refined grains, legumes, and red meat.

The food groups were defined based on the highest amongst consumed by the patients, with only those with significant consumption were included.

Dietary Inflammatory Index (DII) Calculations

The DII was calculated according to Shivappa et al., (2014) for each individual from the sum of the added scores and every dietary and nutrient-specific component.

The DII is composed of 45 items (Shivappa et al., 2014). In the present study, of the 45 items, only 28 were considered (supplementary table 1). It was not possible to estimate, and therefore, the following components were not considered when calculating the DII: alcohol, b-carotene, caffeine, eugenol, ginger, turmeric, green/black tea, pepper, oregano, rosemary, flavan-3-ol, flavones, flavonoids, flavanones, anthocyanidins, and isoflavones.

Initially, an adjustment regarding the total calories to each item of the DII (stem from linear regression and residue analysis) was performed (Willett and Stampfer, 1986).

Then, each food item considered in the DII was transformed into a z score. For example, for carbohydrates, the amount consumed by the individual subtracted by the average consumption of carbohydrates of all individuals, separated by the standard deviation of consumed carbohydrates by all individuals, was considered. The goal was to minimize the effects of different measurement units (for example, grams and micrograms) in the DII.

With the goal of minimizing the effect of nonsymmetrical variables, the third step was to identify each patient's consumption percentile of each dietary component. To obtain a symmetrical distribution, the values were centered on 0. The fourth step was to utilize the previously determined percentile and multiply it by two and subtract 1. This resulted in a value between -1 (maximum anti-inflammatory activity) and 1 (maximum proinflammatory activity). The fifth step was to utilize the patronize percentile and multiply by the inflammatory coefficient available at Shivappa et al., (2014).

After that, the 28 items were added, reaching the final DII of each individual.

Blood sample collection and cytokine analyses

Venous blood samples were collected without anticoagulants (8 ml) and with EDTA (6 ml). The serum was separated through a 10-minute centrifugation at 2.500 rpm. As soon as it was separated, the samples were aliquoted and frozen for subsequent analysis.

The cytokines were analyzed using an aliquoted serum sample via ELISA utilizing a kit (Mini ELISA Development Kit, 900-M21, PeproTech Inc., USA) according to the manufacturer's instructions.

The residue of the biological samples was discarded as prescribed in Resolução RDC no. 306 of December 7th, 2004, which describes the technical regulation to manage residue in public service by National Health Surveillance Agency (ANVISA).

Statistical analysis

As described below, several statistical steps were conducted to analyze the association between the Dietary Inflammatory Index (DII) and inflammatory markers. All analyses were conducted using SPSS statistical software (version 22.0, IBM Corp., Armonk, NY), and p-values <0.05 were considered significant. The Shapiro-Wilk test was used to verify the normality of the distribution of inflammatory markers (IL-4, IL-6, IL-10, IL-17, IFN- γ , TNF- α , and Leptin) in the groups defined by IBD (anti-inflammatory diet and pro-inflammatory diet).

Most inflammatory markers did not show a normal distribution, indicating the need for non-parametric tests for comparison between groups. The Mann-Whitney test was used to compare inflammatory marker levels between these groups.

This test evaluated the differences in the medians of the inflammatory markers between the ANTI-DII and PRO-DII groups, allowing us to investigate whether there are significant differences in the inflammatory response as a function of diet quality.

Spearman's correlation coefficient was calculated to assess the association between DII and the levels of inflammatory markers.

This coefficient made it possible to measure the strength and direction of the monotonic relationship between IBD (as a continuous variable) and inflammatory markers.

A sensitivity analysis was carried out excluding participants diagnosed with diabetes to assess whether this health condition influenced the results.

This analysis was conducted to check the robustness of the associations observed between IBD and inflammatory markers. Again, Spearman's correlation coefficient was used.

RESULTS

The present study analyzed thirty-five patients. Twenty-three patients (65.7%) were female. Ten patients (28.6%) were classified by BMI as having class II obesity, and twenty-five (71.4% %) were classified as having class III obesity.

Concerning the comorbidities that were presented by the patients, nine (25.7% %) had a previous diagnosis of systemic arterial hypertension. Three patients (8.6%) had already been diagnosed with type II diabetes.

DII, clinical and the anthropometric data

Table 1 presents the clinical and the anthropometric data of the samples. The median DII was +0.18, with a maximum anti-inflammatory value of -2.15 and a maximum proinflammatory value of +2.08. The parameters that contributed the most to the proinflammatory values were saturated fat, total fat, and trans-fat.

The DII-related anti-inflammatory parameters that contributed the most were fibers, magnesium, and vitamin D. According to the data, the samples were divided into two groups: an anti-inflammatory diet group (ANTI-DII) and a proinflammatory diet group (PRO-DII). ANTI-DII consisted of individuals with results ranging from -0.01 to -2.15, while PRO-DII included those with results ranging from +0.01 to +2.08. There was no significant difference between the groups.

Table 1 - Patients characteristics, anthropometric data and comparisons between groups stratified according to DII scores (n=35).

	Total Sample (n=35)	ANTI-DII (n=15)	PRO-DII (n=20)	p
Age (years)	34 (28 – 41)	31 (28 – 37)	35 (27 – 41)	0.559
Weight (kg)	120.0 (106.0 – 136.0)	119.0 (105.0 – 137.0)	120.5 (106.0 – 134.4)	0.934
Height (m)	1.67 (1.61 – 1.75)	1.64 (1.61 – 1.70)	1.68 (1.61 – 1.77)	0.161
BMI (kg/m ²)	42.05 (39.33 – 47.18)	42.32 (40.01 – 53.51)	40.79 (38.61 – 48.46)	0.271
DII	0.18 (-1.00 – 0.88)	-1.05 (-1.38 – -0.50)	0.62 (0.44 – 1.23)	

The data are presented as the median (percentile 25 - 75); DII: Dietary Inflammatory Index; ANTI-DII: Dietary Inflammatory Index with anti-inflammatory values; PRO-DII: Dietary Inflammatory Index with proinflammatory values; BMI: Body Mass Index; Significance Level p<0,05.

ANTI-IID and PRO-IID Groups

Table 2 presents the bioquimic exam results and the arterial pressure of the total sample divided into two groups according to the DII. Comparisons between the ANTI and PRO groups were performed. There was a significant difference only for HBA1C. In the sensitivity analysis, patients with diabetes were excluded. Fasting glicemia remained not associated [PRO-DII: 91.0 (83.0–98.0); ANTI-DII: 86.00 (82.50–92.50) p=0.797], and HBA1C remained not significantly different between the groups [PRO-DII: 5,6 (5.3–5.9); ANTI-DII: 5.4 (5.2–5.8) p=0.157].

Inflammatory Markers and DII

Table 3 presents the inflammatory marker data. After comparing the ANTI and PRO results, there was a greater concentration of interleukin 6 (IL-6) [PRO-DII: 36.86 (35.43-41.14); ANTI-DII: 31.14 (28.2–38.29) p=0.034] in the anti-inflammatory group. Additionally, there was a tendency toward elevated interleukin 4 (IL-4) concentrations [PRO-DII: 23.55 (21.54-24.61); ANTI-DII: 24.52 (22.87 – 29.69) p=0.055] in the anti-inflammatory group.

The analysis without the patients with type 2 diabetes revealed that IL-6 maintained

this association [ANTI-DII: 31,14 (28,29–36,86); PRO-DII: 36.86 (35.43–41.14) p=0.023], that IL-4 diminished this association [ANTI-DII: 24,52 (22.87–29.81); PRO-DII: 24.00 (21.22–24.64) p=0.077], and that of IL-17 became significantly

associated [ANTI-DII: 54.89 (51.67–60.23); PRO-DII: 52.74 (42.03–54.89) p=0.049], with higher concentrations in the anti-inflammatory group.

Table 2 - Bioquimic and arterial pressure data of the general sample and the comparison between both groups in accordance with the DII (n=35).

	Total (n=35)	PRO-DII (n=20)	ANTI-DII (n=15)	p
Fasting Glicemia (ml/dL)	91.50 (83.75 – 104.25)	89.50 (74.75 – 96.0)	85.00 (81.00 – 91.5)	0.199
HBA1C (%)	5.50 (5.30 – 6.05)	5.55 (5.25 – 5.75)	5.40 (5.20 – 5.60)	0.050
Cholesterol (mg/dL)	189.0 (161.0 – 210.0)	154.5 (145.5 – 173.2)	189.0 (157.0 – 208.5)	0.329
Tryglicerides (mg/dL)	134.5 (92.25 – 214.50)	148.5 (75.5 – 241.0)	150.0 (90.0 – 229.0)	0.757
HDL (mg/dL)	48.00 (37.50 – 58.75)	43.0 (33.25 – 51.25)	46.00 (34.50 – 51.0)	0.631
LDL (mg/dL)	106.20 (87.49 – 138.95)	79.50 (74.10 – 89.98)	108.0 (96.20 – 140.7)	0.127
SAP (mmHg)	132.0 (121.7 – 141.2)	132.5 (120.5 – 144.5)	132.5 (122.7 – 141.0)	0.713
DAP (mmHg)	84.00 (80.50 – 93.50)	83.00 (78.50 – 94.20)	86.00 (81.20 – 93.50)	0.752

The data are presented as the average (percentile 25 - 75) HbAC1: glycated haemoglobin; HDL: high-density lipoprotein; LDL: low-density lipoprotein; DM: diabetes mellitus; SAP: systolic arterial pressure; PAD: diastolic arterial pressure; DII: dietary inflammatory index; ANTI-DII: dietary inflammatory index with anti-inflammatory values; PRO-DII: dietary inflammatory index with pro-inflammatory values; significance level p<0,05.

Table 3 - Concentrations of inflammatory markers in the total sample and in the groups divided according to the DII (n=35).

	Total (n=35)	ANTI-DII (N=15)	PRO-DII (N=20)	p
IFN-y (pg/mL)	25.50 (23.52 – 35.28)	25.30 (21.83 – 37.50)	25.75 (24.21 – 34.18)	0.894
IL-4 (pg/mL)	24.05 (22.52 – 26.99)	24.52 (22.87 – 29.69)	23.55 (21.54 – 24.61)	0.055
IL-17 (pg/mL)	52.74 (46.31 – 57.03)	54.89 (50.60 – 59.16)	52.74 (42.56 – 54.89)	0.081
IL-6 (pg/mL)	35.43 (31.14 – 41.14)	31.14 (28.29 – 38.29)	36.86 (35.43 – 41.14)	0.034
IL-10 (pg/mL)	20.00 (17.40 – 29.00)	20.53 (18.00 – 31.13)	18.96 (16.34 – 27.75)	0.264
TNF-a (pg/mL)	42.40 (35.70 – 47.00)	42.40 (34.40 – 45.00)	43.20 (38.00 – 50.05)	0.325
Leptin (pg/mL)	82.25 (66.73 – 105.53)	78.37 (64.79 – 105.53)	84.70 (67.69 – 105.43)	0.677

The data are presented as an average (percentile 25 - 75); DII: Dietary Inflammatory Index; ANTI-DII: Dietary Inflammatory Index with anti-inflammatory values; PRO-DII: Dietary Inflammatory Index with proinflammatory values; IFN-y: Interferon gamma; TNF-a: Alfa Tumoral Necrosis Factor; Significance Index p<0,05.

The components analyzed in the DII calculation that showed significant differences in consumption between groups were fibers [ANTI-DII: 21.97 (19.00–29.90); PRO-DII: 13.28 (8.20–17.70) $p<0.001$], vitamin E [ANTI-DII: 3.01 (2.67–3.71); PRO-DII: 2.08 (1.51–3.11) $p=0.016$], vitamin C [ANTI-DII: 71.77 (34.05–195.77); PRO-DII: 28.79 (4.19–61.25) $p=0.018$] and magnesium [ANTI-DII: 249.31 (213.82–283.94); PRO-DII: 196.04 (132.21–232.09) $p=0.006$]. The medians of food intake and all the parameters and differences between groups are presented in the Supplementary Information.

Food groups and DII

Based on the 24-h dietary recall, the food intake data were classified according to the food group. The comparison between the ANTI and PRO groups revealed a significant difference in legumes intake [ANTI-DII: 80 (0–166); PRO-DII: 0 (0–77) $p=0.019$], with a greater difference in the ANTI-DII group. Red meat intake was significantly greater in the PRO-DII group [PRO-DII: 90 (7.5–180); ANTI-DII: 63 (0–100) $p=0.041$]. These data are presented in Table 4.

Table 4 - The food intake of both groups is categorized in the total sample and in the groups divided according to the DII (n=35)

Food Group	Total (n=35)	ANTI-DII (n=15)	PRO-DII (n=20)	p
Legumes and Vegetables (g)	78.0 (33.3 – 126.0)	124.0 (40.0 – 170)	54.5 (0 – 97.0)	0.061
Fruits (g)	80.0 (0 – 250.0)	150.0 (70.0 – 300.0)	32.5 (0 – 241.6)	0.119
Wholegrain cereals (g)	0 (0 – 50.0)	25.0 (0 – 80.0)	0 (0 – 22.9)	0.127
Refined Grains(g)	221.7 (100.0 – 415.0)	247.5 (85.0 – 415.0)	221.0 (142.0 – 326.0)	0.973
Legumes (g)	46.7 (0 – 140.7)	80.0 (0 – 166.0)	0 (0 – 77.5)	0.019
Red Meat(g)	83.3 (0 – 120.0)	63.0 (0 – 100.0)	90.0 (7.5 – 180.0)	0.041
Chicken or Pork(g)	63.0 (0 – 100.0)	80.0 (30.0 – 120.0)	40.0 (0 – 78.3)	0.138
Milk and dairy products(g)	211.0 (102.0 – 290.0)	231.7 (170.0 – 370.0)	195.0 (90.3 – 250.0)	0.125

The data are presented as an average (percentile 25 - 75); DII: Dietary Inflammatory Index; ANTI-DII: Dietary Inflammatory Index with anti-inflammatory values; PRO-DII: Dietary Inflammatory Index with proinflammatory values; g: grams; significance level $p<0,05$.

Spearman correlations were calculated for the food groups and inflammatory markers. There was a positive correlation between the intake of legumes and the IL-4 level ($r: 0,609$; $p<0.010$). The intake of refined grains had a positive correlation with IFN- γ levels ($r: 0,359$; $p<0.050$), and wholegrain cereal intake had negative correlation with IFN- α levels ($r: -0.360$; $p<0.050$).

DISCUSSION

The present cross-sectional study included 35 patients with class II or III obesity and revealed a positive association between DII proinflammatory scores and higher

concentrations of IL-6. The DII anti-inflammatory results demonstrated a positive tendency to elevate the IL-4 concentration. The analysis that excluded individuals with type II diabetes revealed a significant difference in the level of IL-17: the anti-inflammatory group presented a greater concentration of IL-17.

An increase in the anti-inflammatory DII was associated with an elevated in the intake of fibers, vitamin E, vitamin C, magnesium, and legumes and a decrease in the intake of red meat. Legumes intake had a positive correlation with IL-4 levels, whole-grain cereal intake had an inverted correlation with IFN- γ levels, and refined grain intake had a positive correlation with the same marker.

Other studies have shown that the proinflammatory DII is associated with increased IL-6 levels in healthy individuals (Shivappa et al., 2015; Bodén et al., 2017).

Padin et al., (2019) [15, in a sample with a BMI between 22 kg/m² and 43.8 kg/m², found an association between DII and IL-6 only when the data were stratified via visceral adiposity, as well as between TNF- α and IL-1 β .

The studies that evaluated the DII and IL-6 correlation in overweight and obese populations involved weight-reducing interventions. However, the results are controversial. Mayr et al. (2018), in a study with only women (n=56; BMI: 29.9 kg/m²), observed a change in the DII as an anti-inflammatory profile and presented a positive correlation with lower values of IL-6 at the end of a period of six months. In another study (n=73; BMI: 30.7 kg/m²) before the intervention, a significant association was observed between DII and IL-6 levels, but at the end of 12 months, there was a change to a more anti-inflammatory DII that was no longer associated with lower levels of inflammation after weight loss (Duggan et al., 2021).

Only one study has investigated the correlation between IL-4 and the DII. However, the research included pregnant women who presented a different inflammatory profile (McCullough et al., 2017). IL-4 is an anti-inflammatory cytokine, and the present study showed a tendency toward higher levels of this cytokine in the anti-inflammatory diet group.

In homeostasis, this cytokine decreases resistance to insulin; inhibits the synthesis of proinflammatory cytokines, such as IL-1, IL-6, IL-8 and TNF- α ; and promotes the recruitment of M1 macrophages (Bhattacharjee et al., 2013), which increases the potential for modulation through diet.

A significant difference was found in the IL-17 levels of the DII groups: higher levels were found in the anti-inflammatory group in the analysis that excluded diabetic patients. IL-17 is a proinflammatory cytokine that is elevated in individuals with obesity. Currently, there is an association between IL-17 levels and the individual's satiety, with an inverse concentration to spontaneous intake, which makes the cytokine considered an appetite regulatory adipocyte (Nogueira et al., 2020), which can be a justification for its higher concentration in the proinflammatory diet profile group. However, the present study has limited

value gaps concerning the DII and the sample size.

Additionally, the underlying mechanism has still not been elucidated in individuals with obesity, limiting the findings of this study.

The intake of an anti-inflammatory diet is associated with a greater volume of fibers, vitamin E, vitamin C, magnesium, and legumes flavors as well as a lower intake volume of red meat. This association is consistent with the findings of other studies (Norde et al., 2020; Shivappa et al., 2015; Oliveira et al., 2020), which support that a healthy food ingestion profile is associated with lower DIIs.

Therefore, per the data found in the literature, it can be inferred that a healthy nutritional profile, independent of the tool/questionnaire used to access the information, can modulate inflammatory processes related to chronic diseases (Guillermo et al., 2019; Corley et al., 2019; Ruiz-Canela et al., 2015).

In individuals with obesity, an occidental alimentary profile (higher red meat ingestion and deserts) is related to a higher BMI and a greater fat mass.

However, it is not associated with increased inflammatory marker expression. A healthy alimentary profile characterized by lower ingestion of saturated fat and trans-fatty acids has been proven to be associated with lower proinflammatory marker concentrations (Saghafi-Asl et al., 2021).

The present study revealed a positive correlation between the intake of legumes (black beans and lentils) and the IL-4 concentration. This food group is characterized by high nutritional quality, high concentrations of vitamins and minerals, a healthy alimentary profile, and better lipidic profiles.

Additionally, glicemic profiles are associated with a reduction in cardiovascular disease risk factors (Mudryj, Yu, Aukema, 2014).

Few studies have evaluated the effects of ingestion of legumes water on systemic inflammation. In a study performed with Iranian women, ingestion was inversely associated with the serum concentrations of PCR, TNF α and IL-6 (Esmailzadeh and Azadbakht, 2011).

The effect of a rich diet of 250 g of legumes/day water was analyzed, and the intervention significantly decreased the serum levels of PCR and TNF- α in elderly men (Hartman et al., 2010).

In the present study, a correlation between IFN- γ and cereal intake was found. It was positively correlated with refined grains such as white rice, pasta, wheat bread, and cornmeal and negatively correlated with whole grains such as whole-grain rice, whole-meal bread, and whole-meal pasta.

This association has not been studied in depth. The refined grains are a part of the occidental dietary profile, and the whole grains are a part of the anti-inflammatory diet. Both diets have been linked to alterations in inflammatory markers, such as IL-6 and leptin (Norde et al, 2020).

IFN- γ is a stimulant factor in inflammation, especially through macrophage activation. Moreover, it is capable of inhibiting an increase in anti-inflammatory cytokines, maintaining a prominent inflammatory profile (Chawla, Nguyen, Goh, 2011).

Considering that macrophages contribute to the maintenance of the inflammatory framework in adipose tissue, there is a relationship between whole grains and reduced cytokine levels, which is an indicator of the importance of a quality diet in the modulation of the inflammatory framework present in this disease.

The DII utilized 28 alimentary parameters. Some parameters are not easily acquired in an interview or are a populational habit, limiting the calculation. The use of 28 parameters may explain why the associations found were reduced.

One option for evaluating the effects of alimentation on a health-disease process and creating alimentary indices is to look at the literature.

Through other studies, it is possible to identify nutrients and/or food that can be associated with an interesting outcome (for example, inflammatory markers) and define an algorithm to characterize individuals. This is the case for DII, which uses 25 macro- and micronutrients and only nine alimentary components. There is a limitation in the index that makes it unattainable to directly interpret: we do not intake nutrients but rather food (Hoffmann, 2004).

Moreover, the level of effectiveness of each component is based on the frequency of citations of that component in pro- and anti-inflammatory research, not on the amount of effect they showed on previous studies.

Additionally, 24-h dietary recall was utilized as an approximation of the average food

intake. This method, as well as other methods used to evaluate food intake, has limitations.

For example, it does not consider the possible variability of a daily diet and can underestimate food intake. To determine the DII, a total caloric adjustment was performed, which reduced the underestimation of individuals with obesity.

This study was conducted with patients in an obesity treatment facility. They were in the interview stage of possible surgical intervention, and it is believed that they knew the dietary value of weight loss, which could have influenced their 24-h dietary recall, modifying to a healthier version with smaller food portions.

Other limitations need to be considered. For example, not taking into account physical activity could have influenced cytokine levels.

Additionally, the small number of patients in the study did not allow for a large statistical inference. It is not possible to determine whether the cross-sectional nature of the study influenced the observed components.

The present study revealed an association between DII and IL-6 and IL-17 concentrations. When the diet was separated into food groups, the legumes diet was positively correlated with the IL-4 concentration, and the cereal diet was positively correlated with the IFN- γ concentration.

These results suggest that the best-quality dietary intake can be associated with a better inflammatory profile in class II and III individuals. This association can be evaluated through an inflammatory diet index. However, more research on this subject is needed.

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