

ASSOCIATION BETWEEN THE DIETARY INFLAMMATORY INDEX AND PLASMA CYTOKINES IN RECREATIONAL ROAD RUNNERS AND SEDENTARY INDIVIDUALS

ASSOCIAÇÃO ENTRE O ÍNDICE INFLAMATÓRIO DA DIETA E CITOCINAS PLASMÁTICAS EM CORREDORES DE RUA RECREACIONAIS E INDIVÍDUOS SEDENTÁRIOS

ASOCIACIÓN ENTRE EL ÍNDICE INFLAMATORIO DE LA DIETA Y LAS CONCENTRACIONES PLASMÁTICAS DE CITOCINAS EN CORREDORES RECREACIONALES DE CALLE E INDIVIDUOS SEDENTARIOS



<https://doi.org/10.56238/sevened2026.020-015>

Priscila da Trindade Flores¹, Joane Severo Ribeiro², Bruna Moraes Isidoro³,
Alessandra Peres⁴

ABSTRACT

Objective: To evaluate the association between the Dietary Inflammatory Index (DII) and plasma cytokine concentrations in recreational road runners and sedentary individuals.

Methods: A cross-sectional study was conducted with adult recreational runners and sedentary individuals. DII, body composition and training variables, and plasma concentrations of inflammatory cytokines were assessed.

Results: Among runners, DII showed a positive association with IL-6 and IL-1 β , even after adjustment for age, sex, and body fat percentage. In the adjusted models, DII remained associated with IL-6 ($\beta = 28.4\%$; 95%CI: 8.5–51.2; $p = 0.006$) and IL-1 β ($\beta = 25.6\%$; 95%CI: 3.1–47.0; $p = 0.021$). Among sedentary individuals, the correlations observed between DII and IL-10, IL-1ra, and TNF- α did not remain significant after adjustment. Runners showed higher concentrations of IL-6 and IL-1 β , whereas sedentary individuals showed higher body fat percentage and a positive association between DII and adiposity.

Conclusion: The inflammatory potential of the diet was associated with the plasma cytokine profile in recreational runners, but not in sedentary individuals after adjustment. These

¹ Doctoral student in Biosciences. Universidade Federal de Ciências da Saúde de Porto Alegre. E-mail: priscila.flores@ufcspa.edu.br Orcid: <https://orcid.org/0000-0001-9659-7996> Lattes: <http://lattes.cnpq.br/609101952440429>

² Dr. in Rehabilitation Sciences. Universidade Federal de Jataí. E-mail: joaneribeiro@gmail.com Orcid: <https://orcid.org/0000-0001-6096-5427> Lattes: <http://lattes.cnpq.br/3855610685623208>

³ Dr. in Biosciences. Universidade Federal de Ciências da Saúde de Porto Alegre. E-mail: isidorombruna@gmail.com Orcid: <https://orcid.org/0000-0002-0188-7163> Lattes: <http://lattes.cnpq.br/6303502638711243>

⁴ Dr. in Genetics and Molecular Biology. Universidade Federal de Ciências da Saúde de Porto Alegre. E-mail: peres@ufcspa.edu.br Orcid: <https://orcid.org/0000-0002-3587-2832> Lattes: <http://lattes.cnpq.br/0885635447030520>

findings suggest distinct inflammatory profiles between groups and indicate that diet quality may be related to the basal inflammatory profile in physically active individuals.

Keywords: Diet. Cytokines. Inflammation. Running. Body Composition.

RESUMO

Objetivo: Avaliar a associação entre o Índice Inflamatório da Dieta (IID) e as concentrações plasmáticas de citocinas em corredores recreacionais de rua e indivíduos sedentários.

Métodos: Estudo transversal conduzido com adultos corredores recreacionais e sedentários. Foram avaliados o IID, variáveis de composição corporal e treinamento e concentrações plasmáticas de citocinas inflamatórias.

Resultados: Entre os corredores, o IID apresentou associação positiva com IL-6 e IL-1 β , mesmo após ajuste por idade, sexo e percentual de gordura corporal. Nos modelos ajustados, o IID manteve associação com IL-6 ($\beta = 28,4\%$; IC95%: 8,5–51,2; $p = 0,006$) e IL-1 β ($\beta = 25,6\%$; IC95%: 3,1–47,0; $p = 0,021$). Entre os sedentários, as correlações observadas entre IID e IL-10, IL-1ra e TNF- α não permaneceram significativas após os ajustes. Os corredores apresentaram maiores concentrações de IL-6 e IL-1 β , enquanto os sedentários apresentaram maior percentual de gordura corporal e associação positiva entre IID e adiposidade.

Conclusão: O potencial inflamatório da dieta associou-se ao perfil de citocinas plasmáticas em corredores recreacionais, mas não em sedentários após os ajustes. Os achados sugerem perfis inflamatórios distintos entre os grupos e indicam que a qualidade da dieta pode estar relacionada ao perfil inflamatório basal em indivíduos fisicamente ativos.

Palavras-chave: Dieta. Citocinas. Inflamação. Corrida. Composição Corporal.

RESUMEN

Objetivo: Evaluar la asociación entre el Índice Inflamatorio de la Dieta (IID) y las concentraciones plasmáticas de citocinas en corredores recreacionales de calle e individuos sedentarios.

Métodos: Se realizó un estudio transversal con adultos corredores recreacionales y sedentarios. Se evaluaron el IID, variables de composición corporal y entrenamiento, y las concentraciones plasmáticas de citocinas inflamatorias.

Resultados: Entre los corredores, el IID mostró asociación positiva con IL-6 e IL-1 β , incluso después del ajuste por edad, sexo y porcentaje de grasa corporal. En los modelos ajustados, el IID mantuvo asociación con IL-6 ($\beta = 28,4\%$; IC95%: 8,5–51,2; $p = 0,006$) e IL-1 β ($\beta = 25,6\%$; IC95%: 3,1–47,0; $p = 0,021$). Entre los sedentarios, las correlaciones observadas entre el IID e IL-10, IL-1ra y TNF- α no permanecieron significativas después de los ajustes. Los corredores presentaron mayores concentraciones de IL-6 e IL-1 β , mientras que los sedentarios mostraron mayor porcentaje de grasa corporal y asociación positiva entre el IID y la adiposidad.

Conclusión: El potencial inflamatorio de la dieta se asoció con el perfil plasmático de citocinas en corredores recreacionales, pero no en individuos sedentarios tras los ajustes. Los hallazgos sugieren perfiles inflamatorios distintos entre los grupos e indican que la calidad de la dieta puede estar relacionada con el perfil inflamatorio basal en individuos físicamente activos.



Palabras clave: Dieta. Citocinas. Inflamación. Carrera. Composición Corporal.

1 INTRODUCTION

Regular physical exercise is widely recognized for its beneficial effects on metabolism and the immune system. In addition to contributing to body weight control and improved insulin sensitivity, physical exercise plays a role in modulating systemic inflammation, reducing the risk of chronic diseases associated with persistent inflammatory states. Physically active individuals tend to present lower concentrations of pro-inflammatory cytokines and greater release of mediators with regulatory functions, which favors immunometabolic balance (Bernhart et al., 2022; Chamberlin et al., 2022; Gleeson, 2013).

In individuals who engage in regular physical exercise, acute inflammatory peaks become progressively attenuated, while physiological adaptations emerge that promote a more regulated immune environment (Chamberlin et al., 2022). The body adjusts inflammatory responses according to the type, load, and intensity of training, reducing basal inflammation and improving communication between the immune and metabolic systems. In this context, diet also influences these responses, since physically active individuals with a balanced diet tend to show lower inflammatory variability and more efficient recovery after physical exertion (Almeida-Neto et al., 2025; Zhou et al., 2025).

Considering the role of diet in the regulation of inflammation, Shivappa et al. (2014) developed the Dietary Inflammatory Index (DII) as a tool to quantitatively assess the inflammatory potential of dietary patterns. Diets with pro-inflammatory characteristics present higher scores, whereas anti-inflammatory dietary patterns exhibit negative scores. Studies have demonstrated a direct association between higher DII scores and increased concentrations of inflammatory cytokines, such as IL-6, IL-1 β , and TNF- α (Hass et al., 2022; Millar et al., 2022; Godala et al., 2025), reinforcing the usefulness of the DII in identifying the impact of diet quality on the modulation of the inflammatory response.

Another relevant factor in inflammatory mechanisms is body composition. Adipose tissue is recognized as an endocrine organ responsible for the synthesis of regulatory hormones and the production of pro-inflammatory cytokines, such as IL-6 and TNF- α . In contrast, muscle mass plays essential roles in metabolic regulation and in the control of inflammatory processes (Doustmohammadian et al., 2024; Zhang et al., 2023). Recent evidence indicates that a higher body fat percentage is associated with higher DII scores and greater concentrations of pro-inflammatory cytokines, whereas individuals with a higher percentage of lean mass present lower DII scores and a more balanced immune profile (Dave et al., 2025; Li et al., 2025). In runners, the interaction between body composition, diet, and training load plays a relevant role in the modulation of inflammation and physical

performance, reinforcing the importance of analyzing these factors in an integrated manner (Shi et al., 2023).

Although the independent impact of physical exercise and inflammatory diet on inflammatory markers has been well described, studies evaluating the direct association between the Dietary Inflammatory Index and plasma cytokines in recreational road runners, a group characterized by substantial heterogeneity in training volume and dietary patterns, remain scarce. Therefore, the present study aimed to evaluate the association between the DII and plasma cytokine concentrations in recreational road runners and sedentary individuals.

2 METHODS

This was an analytical cross-sectional study conducted between September 2022 and March 2024 in the city of Porto Alegre, Rio Grande do Sul, Brazil. The study was conducted in accordance with the principles of the Declaration of Helsinki and Resolution No. 466/12 of the Brazilian National Health Council, and was approved by the Research Ethics Committee involving Human Beings of the Federal University of Health Sciences of Porto Alegre (CAAE: 58330022.6.0000.5345). All volunteers were recruited through social media and sports advisory services, were informed about the objectives and procedures of the study, and signed an informed consent, prior to enrollment.

Participants

The sample consisted of recreational road runners and sedentary individuals aged 18 to 50 years. The runner group included participants who had been participating in road running for at least six months, with a minimum frequency of 150 minutes of physical activity per week, and who performed long-distance running sessions of at least 5 km per session. The sedentary group consisted of individuals who had not engaged in regular physical activity for at least six months and were classified as inactive or insufficiently active according to the short version of the International Physical Activity Questionnaire (IPAQ) (Matsudo et al., 2001).

The exclusion criteria were: (i) body mass index (BMI) ≥ 30 kg/m²; (ii) smoking; (iii) pregnancy; (iv) history of surgery within the six months prior to data collection; (v) presence of acute inflammatory disease, recent infection, or gingival/periodontal disease; (vi) use of anti-inflammatory drugs or corticosteroids; and (vii) occurrence of musculoskeletal injury during the week prior to data collection.

Sociodemographic and anthropometric data collection

Sociodemographic information and training-related parameters were collected using

an online questionnaire. Anthropometric assessment was performed in person by a single trained evaluator, using a digital scale with stadiometer (capacity of 150 kg, precision of 100 g) to measure body weight and height. For body composition assessment, seven skinfolds were measured (chest, midaxillary, triceps, subscapular, suprailiac, abdominal, and medial thigh), all on the right side of the body, using a Cescorf® scientific skinfold caliper (sensitivity of 0.1 mm, reading range of 88 mm), following the recommendations of the International Society for the Advancement of Kinanthropometry (ISAK). Each skinfold was measured in triplicate, and the mean value was used. Body density was estimated using the equations of Jackson and Pollock (1978) for men and Jackson, Pollock, and Ward (1980) for women. Body fat percentage was obtained using Siri's equation (1961), $\%BF = (495/D) - 450$, from which lean mass (kg) and fat mass (kg) values were derived.

Dietary intake assessment and DII calculation

Habitual dietary intake was assessed using a semi-quantitative Food Frequency Questionnaire (FFQ), validated for the Brazilian population and based on the reduced version of the Brazilian Longitudinal Study of Adult Health (ELSA-Brasil) (Molina et al., 2013). The instrument evaluated the frequency of consumption of 76 food items over the previous 12 months, distributed into seven food groups (bread, cereals, and tubers; fruits; vegetables, legumes, and pulses; meat, eggs, milk, and dairy products; pasta and mixed dishes; sweets; and beverages). Responses were recorded in nine frequency categories, ranging from “never or almost never” to “more than three times a day.” Nutrient content was calculated based on the Brazilian Food Composition Table (TBCA, 2023), and household measures were converted into grams (Fisberg; Villar, 2002).

The DII was calculated according to the method proposed by Shivappa et al. (2014), considering 25 of the 45 food parameters available in the database. The evaluated components included vitamins A, C, D, E, B6, and B12, folic acid, β -carotene, calcium, iron, magnesium, zinc, selenium, niacin, riboflavin, thiamine, protein, carbohydrates, total fat, saturated fat, monounsaturated fat, polyunsaturated fat, trans fat, cholesterol, and fiber.

Dietary intake values were standardized as z-scores, multiplied by the respective inflammatory effect scores, and summed to generate the total DII score. Positive values indicate a diet with pro-inflammatory characteristics, whereas negative values reflect a diet with anti-inflammatory characteristics.

Plasma collection and cytokine determination by ELISA

A total of 20 mL of venous blood was collected from the cubital vein after 24–48 hours without vigorous exercise and without alcohol intake. No dietary restrictions were imposed before sample collection. Samples were placed in EDTA tubes and centrifuged at 3000 rpm

for 10 minutes to obtain plasma, which was subsequently stored at -80°C until analysis.

Plasma concentrations of IL-6, IL-10, IL-1 β , IL-1ra, IFN- γ , and TNF- α were determined by enzyme-linked immunosorbent assay (ELISA), according to the manufacturer's instructions (Peprotech®, USA). Standard curves were prepared using eight calibration points, and readings were performed on an automated optical reader SpectraMax® ($\lambda = 540$ nm), using SoftMax Pro® software for curve fitting and concentration calculations.

The intra-assay coefficient of variation was $< 7.5\%$ and the inter-assay coefficient of variation was $< 10\%$. The detection limits were: IL-6 (24–1500 pg/mL), IL-10 (23–3000 pg/mL), IL-1 β (8–1000 pg/mL), IL-1ra (23–1500 pg/mL), IFN- γ (8–3000 pg/mL), and TNF- α (8–2000 pg/mL).

Sample size calculation

Sample size calculation was performed using GPower software version 3.1.3 (Franz Faul, Universität Kiel, Germany). The sample size was estimated considering the difference in plasma interleukin-6 (IL-6) levels between physically active and sedentary individuals, a marker widely used in studies on low-grade inflammation related to physical exercise and diet (Haß U, et al., 2022). Based on evidence from the literature indicating moderate to large differences in baseline IL-6 levels between groups with different levels of physical activity, a significance level of 5% and statistical power of 80% were adopted. A minimum of 25 participants per group was estimated. Considering possible sample losses, the final planned sample size was approximately 30 participants in each group.

Statistical analysis

The normality of continuous variables was assessed using the Shapiro–Wilk test. Variables with normal distribution were expressed as mean \pm standard deviation (SD) and compared between groups using Student's t-test. For variables with non-normal distribution, the Mann–Whitney U test was applied.

Correlations between the DII score and plasma cytokines (IL-6, IL-10, IL-1 β , IL-1ra, IFN- γ , and TNF- α) were evaluated using Spearman's correlation coefficient, with analyses stratified by group (runners and sedentary individuals).

The association between DII and training volume was analyzed after stratifying weekly mileage and weekly training time into tertiles, representing three levels of training load: low (≤ 33.3 rd percentile), moderate (33.3rd–66.6th percentile), and high (> 66.6 th percentile). Comparisons between tertiles were performed using the nonparametric Kruskal–Wallis analysis of variance.

Multiple linear regression models were applied to investigate the independent effect of DII on plasma cytokines, using natural logarithmic transformation of the dependent

variables [$\ln(\text{cytokine})$]. The models were adjusted for age, sex, and body fat percentage, and were conducted separately for each group (runners and sedentary individuals). The results were expressed as β coefficient (percentage change per +1 point in DII), 95% confidence interval (95% CI), p-value, and adjusted R^2 . The statistical significance level adopted was $p < 0.05$. All statistical analyses were performed using SPSS software, version 25.

3 RESULTS

The sample consisted of 80 participants, including 55 recreational runners and 25 sedentary individuals. As shown in Table 1, runners were significantly older on average (35.96 ± 7.51 years) compared with sedentary individuals (26.12 ± 5.06 years; $p < 0.001$). Regarding body composition, runners had a lower body fat percentage ($p < 0.001$) and higher lean mass ($p = 0.027$) than sedentary individuals. In addition, the DII score was significantly lower among runners (-0.04 ± 1.09) than among sedentary individuals (0.84 ± 1.39 ; $p = 0.011$), indicating a more anti-inflammatory dietary pattern in the physically active group. Regarding cytokines, Table 1 shows that runners had higher plasma concentrations of IL-6 (12.21 ± 5.91 pg/mL vs. 6.97 ± 0.93 pg/mL; $p < 0.001$) and IL-1 β (17.88 ± 11.38 pg/mL vs. 8.42 ± 5.53 pg/mL; $p < 0.001$). Sedentary individuals, in contrast, had higher levels of IL-10 (19.87 ± 1.73 pg/mL vs. 12.34 ± 5.24 pg/mL; $p < 0.001$) and IL-1ra (112.70 ± 60.72 pg/mL vs. 37.17 ± 15.69 pg/mL; $p < 0.001$).

The correlations between DII and plasma cytokines, presented in Table 2, showed distinct patterns between the groups. Among runners, DII was positively correlated with IL-6 ($r = 0.44$; $p = 0.009$) and IL-1 β ($r = 0.35$; $p = 0.028$). Among sedentary individuals, positive correlations were observed between DII and IL-10 ($r = 0.39$; $p = 0.044$), IL-1ra ($r = 0.42$; $p = 0.031$), and TNF- α ($r = 0.41$; $p = 0.036$).

As shown in Table 3, DII was positively correlated with body fat percentage in the sedentary group ($r = 0.472$; $p = 0.017$). In contrast, no significant associations were observed between DII and any body composition variable in the runner group.

For the analysis of the association between DII and training volume, weekly mileage (km/week) and weekly training time were stratified into tertiles and are presented in Table 4. Mean weekly mileage was 41.8 ± 23.2 km, while total weekly training time was 346.1 ± 197.2 minutes. An inverse trend was observed between weekly mileage and DII ($p = 0.061$), and a significant difference was found according to training time per session ($p = 0.042$).

All individual DII components were correlated with plasma cytokines. Table 5 presents those that showed statistically significant correlations. Among runners, a negative correlation

was observed between omega-3 intake and IL-6 ($r = -0.45$; $p = 0.018$), and between fiber intake and IL-1 β ($r = -0.38$; $p = 0.041$). In addition, saturated fat intake showed a negative correlation with IL-10 ($r = -0.43$; $p = 0.027$). Among sedentary individuals, trans fat intake showed a positive correlation with IL-6 ($r = 0.46$; $p = 0.022$), while polyunsaturated fat intake was positively correlated with IL-10 ($r = 0.44$; $p = 0.029$).

The multiple linear regression models (Figure 1) indicated that, among runners, DII remained positively and independently associated with IL-6 levels ($\beta = 28.4\%$; 95% CI: 8.5 to 51.2; $p = 0.006$) and IL-1 β levels ($\beta = 25.6\%$; 95% CI: 3.1 to 47.0; $p = 0.021$), even after adjustment for age, sex, and body fat percentage. Among sedentary individuals, no cytokine showed a significant association with DII.

Table 1

Participant characteristics regarding body composition, total DII score, and plasma cytokines in road runners and sedentary individuals

Variable	Runners (mean \pm SD) n=55	Sedentary individuals (mean \pm SD) n=25	p-value
Age (years)	35,96 \pm 7,51	26,12 \pm 5,06	< 0,001
BMI (kg/m ²)	23,71 \pm 2,91	24,45 \pm 4,65	0,467
Body Fat (%)	15,96 \pm 7,12	26,97 \pm 8,80	< 0,001
Lean mass (kg)	58,53 \pm 11,89	45,61 \pm 7,08	0,027
Fat mass (kg)	10,88 \pm 4,92	17 \pm 9,15	< 0,001
DII (total score)	-0,04 \pm 1,09	0,84 \pm 1,39	0,011
IL-6 (pg/mL)	12,21 \pm 5,91	6,97 \pm 0,93	< 0,001
IL1-ra (pg/mL)	37,17 \pm 15,69	112,70 \pm 60,72	< 0,001
IL-10 (pg/mL)	12,34 \pm 5,24	19,87 \pm 7,13	< 0,001
IL-1 β (pg/mL)	17,88 \pm 11,38	8,42 \pm 5,53	< 0,001
IFN- γ (pg/mL)	15,67 \pm 7,49	18,15 \pm 7,42	0,188
TNF- α (pg/mL)	224,97 \pm 232,49	188,49 \pm 217,65	0,548

Legend: Student's t-test was used for variables with normal distribution, and the Mann–Whitney U test was used for variables with non-normal distribution. BMI: body mass index; DII: Dietary Inflammatory Index; IL-6: interleukin-6; IL-10: interleukin-10; IL-1 β : interleukin-1 beta; IL-1ra: interleukin-1 receptor antagonist; IFN- γ : interferon-gamma; TNF- α : tumor necrosis factor-alpha. Source: authors.

Table 2

Correlation between DII and plasma cytokines in road runners and sedentary individuals.

Cytokine	Runners r (Spearman) n=55	p-value	Sedentary individuals r (Spearman) n=25	p-value
IL-6 (pg/mL)	0,44	0,009	0,26	0,241

IL-10 (pg/mL)	-0,18	0,218	0,39	0,044
IL-1 β (pg/mL)	0,35	0,028	0,21	0,314
IL-1ra (pg/mL)	0,19	0,203	0,42	0,031
IFN- γ (pg/mL)	0,13	0,331	0,22	0,284
TNF- α (pg/mL)	0,12	0,351	0,41	0,036

Legend: IL-6: interleukin-6; IL-10: interleukin-10; IL-1 β : interleukin-1 beta; IL-1ra: interleukin-1 receptor antagonist; IFN- γ : interferon-gamma; TNF- α : tumor necrosis factor-alpha. Source: authors.

Table 3

Correlation between DII and body composition variables in road runners and sedentary individuals.

Group	Body composition variable	r (Spearman)	p-value
Runners n=55	BMI	0,298	0,058
	Body Fat (%)	0,110	0,492
	Fat mass (kg)	0,146	0,363
	Lean mass (kg)	0,044	0,786
Sedentary individuals n=25	BMI	0,275	0,183
	Body Fat (%)	0,472	0,017
	Fat mass (kg)	0,321	0,118
	Lean mass (kg)	-0,100	0,636

Legend: BMI: body mass index. Source: authors.

Table 4

DII scores according to tertiles of training parameters among road runners.

Training parameters	Category	Mean \pm SD	Mean \pm SD DII	n	p-value
Distance (km/week)	Low	24,3 \pm 5,8	-0,12 \pm 0,98	19	0,061
	Moderate	42,5 \pm 6,7	0,55 \pm 1,23	10	
	High	65,7 \pm 8,4	-0,63 \pm 0,96	10	
Time (minutes/week)	Low	200,5 \pm 54,2	0,43 \pm 0,95	16	0,042
	Moderate	340,7 \pm 61,5	-0,22 \pm 1,09	10	
	High	510,3 \pm 80,6	-0,61 \pm 1,13	12	

Legend: km: kilometers; SD: standard deviation; DII: Dietary Inflammatory Index. Source: authors.

Table 5

Correlation between dietary components of the DII and plasma cytokines in road runners and Sedentary individuals

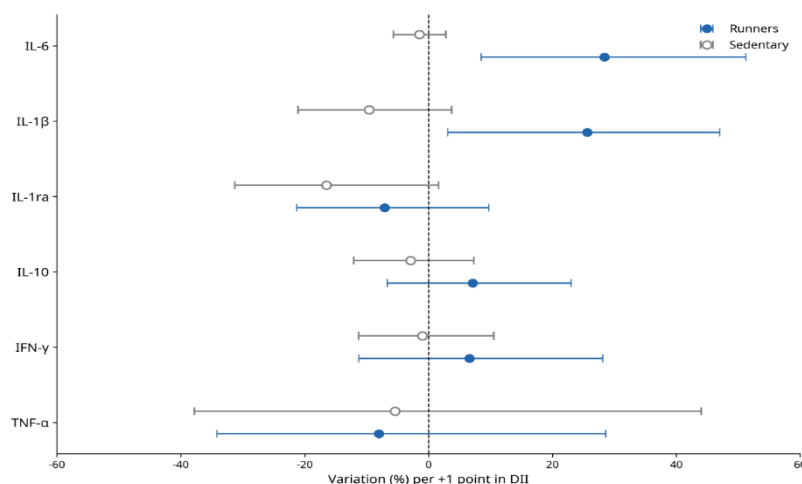
	Nutrient (DII)	Cytokine	ρ (Spearman)	p-value
Runners n=55	Ômega-3	IL-6	-0.45	0,018
	Fiber	IL-1 β	-0.38	0,041

	Saturated fat	IL-10	-0.43	0,027
	Vitamin C	IL-1ra	0.42	0,031
Sedentary individuals n=25	Trans fat	IL-6	0.46	0,022
	Ômega-6	TNF- α	0.41	0,036
	Polyunsaturated fats	IL-10	0.44	0,029

Legend: IL-6: interleukin-6; IL-10: interleukin-10; IL-1 β : interleukin-1 beta; IL-1ra: interleukin-1 receptor antagonist; IFN- γ : interferon-gamma; TNF- α : tumor necrosis factor-alpha; DII: Dietary Inflammatory Index. Source: authors.

Figure 1

Effect of DII on cytokines (adjusted models)



Legend: IL-6: interleukin-6; IL-10: interleukin-10; IL-1 β : interleukin-1 beta; IL-1ra: interleukin-1 receptor antagonist; IFN- γ : interferon-gamma; TNF- α : tumor necrosis factor-alpha; DII: Dietary Inflammatory Index. Source: authors.

4 DISCUSSION

This study investigated the association between DII and plasma cytokine levels in road runners and sedentary individuals, considering aspects of body composition and physical training. The main finding was the positive association between the Dietary Inflammatory Index (DII) and plasma concentrations of IL-6 and IL-1 β among recreational runners. This association was observed both in the correlation analyses and in the models adjusted for age, sex, and body fat percentage, indicating that the inflammatory potential of the diet was related to the profile of these cytokines even after controlling for relevant confounding factors. Among sedentary individuals, correlations were observed between DII and IL-10, IL-1ra, and TNF- α ; however, these associations did not remain after adjustment, suggesting different behavior between the groups.

Runners presented higher concentrations of IL-6 and IL-1 β compared with sedentary individuals, despite having a lower DII score and lower body fat percentage. This result should be interpreted with caution. In individuals who exercise regularly, especially in endurance modalities, higher basal concentrations of some cytokines do not necessarily

indicate pathological inflammation and may reflect physiological adaptations to chronic training. IL-6, for example, acts as a myokine released by skeletal muscle in response to repeated contraction, with metabolic and immunoregulatory functions already described in the literature (Gleeson et al., 2011; Reihmane; Dela, 2014). In this context, the association between IL-6, IL-1 β , and DII only among runners suggests that, in this group, diet quality was related to variation in the basal inflammatory profile even in the presence of regular training.

This finding indicates that exercise alone does not eliminate the influence of diet on inflammatory biomarkers. The higher levels of IL-6 and IL-1 β observed among runners may be related to repeated exposure to acute inflammatory stimuli induced by training (Chamberlin et al., 2022; Via et al., 2024). At the same time, the positive association between DII and these cytokines indicates that a more pro-inflammatory dietary pattern may coexist with regular running practice and be associated with a less favorable inflammatory profile. This interpretation is consistent with findings in athletes and runners linking poorer dietary intake quality to higher concentrations of inflammatory markers (Almeida-Neto et al., 2025; Passos et al., 2019). It is also consistent with studies associating anti-inflammatory dietary patterns with lower concentrations of IL-6 and IL-1 β , in addition to higher concentrations of IL-10 (Ndiema, 2023; Shin et al., 2020; Pietrzak et al., 2023; Kęska et al., 2022). Even among physically active individuals, diet seems to contribute to the organization of the basal inflammatory profile.

Among sedentary individuals, the pattern was different. This group presented higher concentrations of IL-10 and IL-1ra, cytokines traditionally associated with regulatory responses. As the associations between DII and cytokines did not remain significant in the adjusted models, the relationship between diet and inflammation in this group appears to depend more on other characteristics, especially body adiposity. Since sedentary individuals also had a higher body fat percentage, it is plausible that the inflammatory environment was influenced by broader metabolic and body composition factors, and not only by the inflammatory potential of the diet. This interpretation is in line with studies showing a relationship between greater adiposity, higher dietary inflammatory scores, and a worse systemic inflammatory profile (Shivappa et al., 2015; Hass et al., 2022; Millar et al., 2022; Godala et al., 2025; Camargo-Ramos et al., 2017; Phillips et al., 2017; Wang et al., 2025).

Among sedentary individuals, the positive association between DII and body fat percentage suggests that diets with greater inflammatory potential coexisted with higher adiposity, a condition linked to the production of inflammatory mediators and immunometabolic dysfunction. Among runners, no significant correlations were observed

between DII and body composition variables. This result may reflect the lower adiposity of this group and, possibly, the modulating effect of regular exercise on adipose tissue and cytokine secretion (Dave et al., 2025; Doustmohammadian, et al., 2024; Zhang et al., 2023; Li et al., 2025; Shi et al., 2023). As this was a cross-sectional study, it is not possible to establish the direction of these associations.

Regarding training variables, a significant difference in DII was observed according to weekly training time, whereas weekly mileage showed only a trend in the same direction. This result suggests that runners who are more engaged in training may present a less inflammatory dietary pattern. This hypothesis is plausible and may reflect greater awareness of healthy lifestyle habits and greater attention to diet among individuals more engaged in sports practice (Muros et al., 2021; Carey et al., 2024). It may also reflect behavioral differences among recreational practitioners, in whom eating habits and nutritional knowledge seem to vary according to the degree of involvement with the modality (Kosendiak et al., 2023; Dion et al., 2024).

The analysis of the individual DII components added a complementary perspective. Among runners, omega-3 intake was negatively correlated with IL-6, and fiber intake was negatively correlated with IL-1 β . These findings are biologically plausible and reinforce the role of nutrients with recognized anti-inflammatory potential in the regulation of the immune response (Shin et al., 2020; Akbari-Fakhrabadi et al., 2024; Badenhorst et al., 2016). Among sedentary individuals, trans fat intake showed a positive correlation with IL-6, whereas omega-6 intake was positively correlated with TNF- α . These results follow the expected direction for dietary components associated with a more pro-inflammatory profile. The association between polyunsaturated fats and IL-10 should be interpreted with caution, but it is biologically plausible based on experimental and mechanistic evidence describing increased IL-10 and anti-inflammatory effects for certain polyunsaturated fatty acids, especially those from the n-3 series (Bradley et al., 2008; Rocha et al., 2017).

The results do not allow this pattern, by itself, to be interpreted as greater pathological inflammation in runners. The findings point to distinct inflammatory profiles, probably influenced by different mechanisms. Among runners, the combination of chronic training and diet quality seems to be more relevant to variation in IL-6 and IL-1 β . Among sedentary individuals, higher body adiposity may have had a greater influence on the organization of this inflammatory profile. This interpretation seems more consistent with the multifactorial nature of the inflammatory response to exercise, in which training load, timing of sample collection, body composition, and individual characteristics influence the interpretation of biomarkers (Barros et al., 2017).

Among the study limitations, it should be noted that the cross-sectional design prevents causal inferences. The use of only a single blood collection does not allow the temporal variability of cytokines to be assessed. Sampling was performed after 24 to 48 hours without vigorous exercise, an interval that may still be heterogeneous for markers sensitive to recent training, since exercise-induced inflammatory changes may return to baseline within about 24 hours in many protocols, but may also persist longer depending on the intensity, duration, and characteristics of the effort (Alves et al., 2022; Proschinger et al., 2023). There was also no standardization of pre-collection dietary intake, which may have increased the variability of plasma concentrations. In addition, DII was calculated using 25 of the 45 original components, which may reduce the comprehensiveness of the estimate of the inflammatory potential of the diet. Differences between the groups in age and body composition, together with the smaller sample size among sedentary individuals, also require caution in the direct comparison of inflammatory profiles. Even so, the study shows that, among recreational runners, DII was independently associated with IL-6 and IL-1 β in a group that remains little explored in this context.

5 CONCLUSION

In this study, the inflammatory potential of the diet was associated with IL-6 and IL-1 β in recreational runners, but not in sedentary individuals after adjustment. The results show distinct inflammatory profiles between the groups and indicate that, in physically active individuals, diet quality may be related to the basal inflammatory profile. The cross-sectional design and the single blood collection require caution in interpreting the findings. Even so, the data highlight the importance of considering diet quality in the evaluation of inflammatory biomarkers in recreational runners and support the need for longitudinal studies with greater control of training and dietary variables.

ACKNOWLEDGMENTS

This study was supported by the Federal University of Health Sciences of Porto Alegre (UFCSPA), the National Council for Scientific and Technological Development (CNPq), the Research Support Foundation of the State of Rio Grande do Sul (FAPERGS), and the Coordination for the Improvement of Higher Education Personnel (CAPES) – Finance Code 001. Alessandra Peres holds a CNPq PQ2 Research Productivity Fellowship.

REFERENCES

- Akbari-Fakhrabadi, M., et al. (2024). Diet and exercise-induced inflammation. *Frontiers in Nutrition*, 11, 1438832.
- Almeida-Neto, P. F. D., et al. (2025). The effect of repeated sprints on immunological modulation. *Frontiers in Sports and Active Living*, 7, 1662761.
- Alves, M. D. J., et al. (2022). Changes in cytokines concentration following long-distance running. *Frontiers in Physiology*, 13, 838069.
- Badenhorst, C. E., et al. (2016). High carbohydrate ingestion does not attenuate post-exercise IL-6. *European Journal of Applied Physiology*, 116(9), 1715–1724.
- Barros, E. S., et al. (2017). Acute and chronic effects of endurance running on inflammatory markers. *Frontiers in Physiology*, 8, 779.
- Bernhart, J. A., et al. (2022). The IMAGINE intervention. *Translational Journal of the American College of Sports Medicine*, 7(1), e000181.
- Bradley, R. L., et al. (2008). Dietary fatty acids regulate TNF-alpha and IL-10. *Obesity*, 16(5), 938–944.
- Camargo-Ramos, C. M., et al. (2017). Dietary inflammatory index and cardiometabolic risk. *International Journal of Environmental Research and Public Health*, 14(10), 1104.
- Carey, C. C., et al. (2024). Exploring food choice influences in athletes. *Proceedings of the Nutrition Society*, 83(OCE4), E324.
- Chamberlin, M., et al. (2022). Inflammation variability and physical activity. *Medicine & Science in Sports & Exercise*, 54(9S), 356–357.
- Dave, A., et al. (2025). Evaluating anthropometric indices and inflammation. *Indian Journal of Public Health*.
- Dion, S., et al. (2024). Diet quality of athletes. *Nutrients*, 16(24), 4317.
- Doustmohammadian, A., et al. (2024). Dietary inflammatory index and CRP. *Clinical Nutrition ESPEN*, 60, 156–164.
- Fisberg, R. M., & Villar, B. S. (2002). *Manual de receitas e medidas caseiras*. Signus.
- Gleeson, M., et al. (2011). The anti-inflammatory effects of exercise. *Nature Reviews Immunology*, 11(9), 607–615.
- Gleeson, M. (2013). Anti-inflammatory effects of exercise. Em A. Dannenberg & N. Berger (Eds.), *Obesity, inflammation and cancer*. Springer.
- Godala, M., et al. (2025). Pro-inflammatory diet and inflammatory bowel disease. *Nutrients*, 17(17), 2858.

- Hass, U., et al. (2022). Dietary inflammatory index and muscle function. *Journal of Nutrition Health & Aging*, 26(4), 346–351.
- Jackson, A. S., & Pollock, M. L. (1978). Generalized equations for predicting body density of men. *British Journal of Nutrition*, 40(3), 497–504.
- Jackson, A. S., et al. (1980). Generalized equations for predicting body density of women. *Medicine and Science in Sports and Exercise*, 12(3), 175–181.
- Kęska, A., et al. (2022). Dietary inflammatory index in active men. *International Journal of Environmental Research and Public Health*, 19(11), 6884.
- Kosendiak, A., et al. (2023). Eating habits among ultrarunners. *Frontiers in Nutrition*, 10, 1137412.
- Li, S., et al. (2025). Dietary inflammatory index and abdominal adipose tissue. *Journal of Health, Population and Nutrition*, 44, 254.
- Matsudo, S., et al. (2001). Questionário internacional de atividade física. *Revista Brasileira de Atividade Física e Saúde*, 5, 5–18.
- Millar, S. R., et al. (2022). Dietary score and chronic inflammation. *European Journal of Nutrition*, 61(7), 3377–3390.
- Molina, M. C., et al. (2013). Validade do questionário de frequência alimentar. *Cadernos de Saúde Pública*, 29(6), 379–389.
- Muros, J. J., et al. (2021). Dietary habits in cyclists and triathletes. *Scientific Reports*, 11(1), 15193.
- Ndiema, K. E. (2023). Mediterranean diet and inflammatory index. *Nutrients*, 15(2), 366.
- Passos, B. N., et al. (2019). Dietary intake and marathon inflammation. *Mediators of Inflammation*, 1537274.
- Phillips, C. M., et al. (2017). Physical activity and inflammatory status. *International Journal of Behavioral Nutrition and Physical Activity*, 14(1), 1–12.
- Pietrzak, A., et al. (2023). Diet inflammatory index in active individuals. *Nutrients*.
- Proschinger, S., et al. (2023). Interval and continuous exercise effects on inflammation. *European Journal of Applied Physiology*.
- Reihmane, D., & Dela, F. (2014). Interleukin-6 roles during exercise. *European Journal of Sport Science*, 14(3), 242–250.
- Rocha, D. M., et al. (2017). Dietary fatty acids and inflammatory gene expression. *São Paulo Medical Journal*, 135(2), 157–168.
- Shi, J., et al. (2023). Physical activity and dietary inflammatory index. *Environmental Health and Preventive Medicine*, 28, 40.

- Shin, P. K., et al. (2020). Korean diet and inflammatory markers. *Nutrients*, 12(8), 2468.
- Shivappa, N., et al. (2015). Dietary inflammatory index and inflammatory markers. *British Journal of Nutrition*, 113(4), 665–671.
- Shivappa, N., et al. (2014). Designing a dietary inflammatory index. *Public Health Nutrition*, 17(8), 1689–1696.
- Tabela Brasileira de Composição de Alimentos. (2023). *Versão 7.2*. Universidade de São Paulo.
- Siri, W. E. (1961). Body composition from fluid spaces and density. Em *Techniques for measuring body composition* (pp. 223–244).
- Via, J., et al. (2024). Exercise effects on immune function. *Journal of Physiology*.
- Wang, H., et al. (2025). Anti-inflammatory diets and mortality. *Nutrition & Metabolism*, 22, 11.
- Wang, X., et al. (2023). Dietary inflammatory index and depressive symptoms. *Journal of Affective Disorders*, 335, 332–339.
- Zhang, S., et al. (2023). Central obesity and inflammatory markers. *International Journal of Environmental Research and Public Health*, 20(5), 3781.
- Zhou, J., et al. (2025). Physical activity, inflammatory diet and diabetes. *Nutrients*, 17(1), 47.