

Consumption of ultra-processed foods is positively associated with levels of pro-inflammatory cytokines in postmenopausal women: a cross-sectional study

Giana Zarbato Longo

giana.zarbato@gmail.com

Federal University of Rio Grande do Norte

Danielle Cristine Guimarães da Silva

Universidade Federal de Ouro Preto

Fabrica Geralda Ferreira

Institutional Affiliation: Air Force Cadet Preparatory School

Denise Maria de Lima e Silva Galvão

Federal University of Rio Grande do Norte

Elias Kelvin Severiano de Araújo

Federal University of Rio Grande do Norte

Karla Danielly da S. Ribeiro Rodrigues

Federal University of Rio Grande do Norte

Karine Cavalcanti Maurício Sena Evangelista

Federal University of Rio Grande do Norte

Yara Maria Franco Moreno

Universidade Federal de Santa Catarina

Research Article

Keywords: Inflammatory markers, Menopause, ultra-processed food

Posted Date: October 19th, 2026

DOI: <https://doi.org/10.21203/rs.3.rs-10976704/v1>

License: © ⓘ This work is licensed under a Creative Commons Attribution 4.0 International License. [Read Full License](#)

Additional Declarations: No competing interests reported.

Abstract

Menopause represents the end of reproductive years due to the definitive decrease in follicular activity and is associated with changes in women's physiology and hormonal balance. Women's dietary habits during this period play a crucial role in preventing chronic diseases and mortality. We hypothesized that high consumption of UPF is associated with increased inflammatory markers, while MP is associated with decreased inflammatory markers. A population-based cross-sectional study was conducted with 565 women residing in the municipality of Viçosa/MG, of whom 25% self-reported being menopausal. Dietary consumption was collected using a previously validated 119-item food frequency questionnaire (FFQ). Inflammatory markers (TNF- α , IL-1 β , IL-6, IL-8, IL-10, and IL-12p70) were considered the dependent variables, and multiple linear regression models were developed to identify the association between these markers and the consumption of processed foods by women. A negative association was identified between women who consumed minimally processed foods and pro-inflammatory markers. Conversely, those with higher ultraprocessed food (UPF) consumption were positively associated with average levels of pro-inflammatory cytokines. A diet rich in ultra-processed foods was associated with increased levels of the inflammatory cytokines IL-6, IL-1 β , IL-12p70, and TNF- α , while the consumption of minimally processed foods showed a negative association with TNF- α , IL-6, and IL-1 β , suggesting that dietary patterns play a determining role in the inflammatory profile of menopausal women.

Introduction

Menopause marks the end of the reproductive years and the fundamental process of menopause is related directly to ovarian aging and is characterized by irreversible follicular decline, leading to changes in female physiology and hormone levels(1). Cessation of menstruation and a sharp drop in sex hormones, especially estrogen, trigger these shifts(2). The resulting increase in low-grade systemic inflammation contributes to the development of chronic disease (3).

This subclinical inflammatory process results from an imbalance between pro-inflammatory mediators and anti-inflammatory mechanisms and is exacerbated by reduced estrogen levels, which have anti-inflammatory properties (4). The reduction in estrogen appears to be associated with the activation of immune system cells, such as macrophages, which play a central role in modulating the inflammatory response (5).

Alongside hormonal, metabolic, and immunological changes during menopause, attention should be paid to women's eating habits in this period (6). Global studies show that diet plays a crucial role in preventing chronic diseases and mortality (7, 8). Diets high in energy density, fats, sodium, and low in fiber and micronutrients—especially ultra-processed foods (UPFs)—are linked to higher risks of adverse health outcomes (9). These include cardiometabolic and mental disorders, as well as increased mortality (10).

Juul et al. (2022) (11) examined secular trends in food intake by processing level among US adults, using NHANES survey data from 2001–2002 to 2017–2018. They found that the average intake of UPF foods rose from 53.5% kcal in 2001–2002 to 57.0% kcal in 2017–2018 (trend $p < 0.001$). In the same period, average consumption of minimally processed (MP) foods fell from 32.7 to 27.4% kcal (trend $p < 0.001$).

A review conducted by Tristan Asensi et al. (2023) (12) highlights the direct relationship between the intake of ultra-processed foods and the production of pro-inflammatory cytokines, not only because of their nutrient characteristics but also because of non-nutritive components that promote inflammation. This fact can aggravate the inflammatory state already present in menopausal women (13). Furthermore, evidence suggests that the consumption of UPFs is associated with the severity of somatic menopausal symptoms and a higher risk of osteoporosis and osteopenia in women over 50 years of age. A literature review suggests that, in addition to physical activity, a balanced diet is an important tool for mitigating osteoporosis and other bone health problems in postmenopausal women (14).

Understanding the relationship between menopause, cytokine-mediated inflammation, and contemporary diet constitutes an important axis of research to promote women's health, as elucidating the immunometabolic mechanisms underlying this

interaction is crucial for developing more effective preventive and therapeutic strategies (15). Furthermore, it provides a more integrated view of the physiological and environmental factors that influence women's health during menopause, highlighting the importance of dietary interventions as a complementary tool in managing the changes associated with this phase of life.

Therefore, we hypothesized that high consumption of UPF is associated with increased inflammatory markers, while MP is associated with decreased inflammatory markers.

Methods

1- Study design and Participants

This is a population-based cross-sectional study that employed a two-stage cluster sampling design (census tracts as first-stage units and households as second-stage units) without stratification (16). Of the 1,261 participants evaluated, 451 were men, and 245 had cytokine samples lost due to protein handling issues. The final sample consisted of 565 women, 141 (25.0%) of whom were postmenopausal (Fig. 1). Inclusion criteria were self-reported menopause (amenorrhea for 12 months) and age ≥ 40 years.

2 - Study Variables

Socio-demographic and behavioural variables.

Socio-demographic variables were collected using a structured form administered by pairs of previously trained interviewers. The variables were age (in completed years) and age at menopause. The woman self-reported that she had experienced menopause.

Behavioral variables included alcohol consumption (number of drinks per occasion: zero, one to seven, or eight or more) (17) and physical activity level, assessed with the International Physical Activity Questionnaire (IPAQ), version 6 – long format, validated for the Brazilian population (18). Leisure-time physical activity (LTPA) was measured by the time (minutes) spent on such activities in a typical week, as reported in the leisure domain.

Assessment of food consumption (independent variable)

Food consumption was assessed using a validated food-frequency questionnaire (FFQ) administered during an interview (19). To develop the FFQ, researchers compiled a detailed food list from population-specific 24-hour recall data, collected by trained interviewers during the pilot study using the USDA Five-Step Multiple Pass Method (19). This method guides participants through a 24-hour period (typically the previous day), prompting recall of all foods and beverages consumed during that period. The resulting food list informed questions about habitual intake of 119 foods for the FFQ. The FFQ reference period was the 12 months before the interview. Respondents reported frequency of consumption as 0–12 times per day, week, month, or year.

Foods with a consumption frequency of one or less were eliminated from the list. Information on food consumption was recorded in household measures and transformed into grams. Food and drink items were grouped by similarity in nutrient profiles and culinary uses, resulting in 35 food groups. Average absolute intakes (in grams/day) were computed for each food group.

For classifying foods, all food items were classified according to their degree of processing using the Nova food classification system into one of the following groups: fresh or minimally processed foods, processed culinary ingredients, processed foods, and ultra-processed foods, as detailed in (20). Food consumption data are presented as the contribution of each food item or degree of processing category to daily energy intake. To control for the effect of caloric intake on the

degree of processing groups, an adjustment was made using the residuals method proposed by Willett (21). This variable was categorized in tertiles (independent variable).

Collection of clinical data

Inflammatory markers (outcomes variables)

Blood samples frozen at -80°C were retrieved only on the day of analysis, despite having been collected on the same day as the other protective markers. TNF-α, IL-1β, IL-6, IL-8, IL-10, and IL-12p70 were selected as serum markers and analyzed using the *Human Inflammatory Cytokines* CBA kit according to the manufacturer's instructions, with readings performed via flow cytometry (FacsVerse®). Values were expressed in picograms per milliliter (pg/mL) and represent the mean of 300 beads, without replicates.

25(OH) Vitamin D levels

Serum 25(OH) vitamin D levels were evaluated by chemiluminescence (with an ARCHITECT 25(OH)D kit, using ARCHITECT/AABBOTT equipment (Abbott Architect I Instrument, IL, USA) (22).

3 - Statistical analyses

Normality was assessed using the Shapiro-Wilk test and graphical analysis. Values are reported as proportions, means, and 95% confidence intervals (CI 95%). Analyses were conducted in Stata® version 17.0.

Cytokine and food group variables were transformed into z-scores to enable comparison across different scales and identify outliers, facilitating analysis and decision-making. Food group scores were then categorized into tertiles.

Linear regression models were used to determine associations between food groups in tertiles (independent variable) and the inflammatory markers (outcomes). The models were adjusted for potential confounders selected from the existing literature: physical activity level (PAL) in minutes; alcohol consumption (doses); 25-hydroxyvitamin D (ng/mL); time since menopause; and percentage of calories from food groups. A significance level of 95% was considered. The linearity assumption of the multiple model was evaluated through residual analysis, using plots of residuals versus fitted values. All models yielded p-values > 0.05 for the Breusch-Pagan-Koenker heteroscedasticity test.

To determine the statistical power achieved in the analyses, a post-hoc analysis was conducted in Stata (power rsquared). The sampling plan accounted for weighted adjustments exclusively for female participants.

Results

This study included 565 women. Of these, 25% (95% CI: 21.5–28.7%) were menopausal. The mean age was 52.2 years (95% CI: 51.4–53.1). Physically active women made up 14.9% (95% CI: 9.9–21.7%). Their mean 25OHD concentration was 25.49 mg/dL (95% CI: 24.13–26.84). About 28% consumed alcohol (Table 1).

Table 1: Descriptive characteristics of menopause women according to food consumption.

Variables	All (n=141)	Tertile 1 (n=21)	Tertile 2 (n=42)	Tertile 3 (n=78)	Tertile 1 (n=76)	Tertile 2 (n=44)	Tertile 3 (n=21)
		Minimally Processed Foods,			Ultraprocessed Foods		
Citokines (pg/ml)							
IL10#	0.12 (-0.09;0.33)	0.39 (-0.10; 0.89)	-0.20 (-0.49; 0.09)	0.25 (-0.06; 0.56)	0.29 (-0.30; 0.63)	-0.07 (-0.39; 0.25)	0.01 (-0.35; 0.38)
IL6#	0.19 (-0.01; 0.39)	0.46 (-0.11; 1.02)	-0.05 (-0.40; 0.31)	0.29 (0.01; 0.56)	0.36 (0.07; 0.66)	-0.20 (-0.46; 0.06)	0.64 (-0.01; 1.28)
IL1β#	0.07 (-0.15;0.28)	0.18 (-0.33;0.69)	-0.17 (-0.57; 0.23)	0.20 (-0.09; 0.51)	0.29 (-0.03; 0.62)	-0.30 (-0.55; -0.07)	0.30 (-0.50; 1.10)
IL8#	0.06 (-0.14;0.27)	0.18 (-0.37;0.72)	-0.17 (-0.57; 0.23)	0.19 (-0.1; 0.50)	0.30 (-0.04; 0.63)	-0.30 (-0.55; -0.07)	0.19 (-0.47; 0.86)
TNF-α#	0.13 (-0.06;0.32)	0.51 (0.12; 0.89)	-0.18 (-0.51; 0.16)	0.22 (-0.06; 0.51)	0.30 (0.002; 0.61)	-0.29 (-0.48; -0.10)	0.19 (-0.14; 0.52)
Energy Intake (kcal)	0.03 (-0.19;0.25)	0.02 (-0.46;0.51)	-0.18 (-0.45; 0.08)	0.12 (-0.19; 0.43)	0.31 (-0.07;0.69)	-0.15 (-0.42; 0.11)	-0.03 (-0.47; 0.40)2481.5 (2093.7; 2869.2)
Percentual of food energy	2469.3 (2421.2;2555.1)	2657.5 (2144.6; 3170.4)	-0.29 (-0.57; -0.01)	0.12 (-0.19; 0.43)	2613.2 (2368.3; 2858.0)	-0.34 (-0.58; -0.11)	37.41 (33.57; 41.25)
Vitamin D (mg/dL)	25.49 (24.13; 26.84)	48.55 (43.91; 53.19)	2031.4 (1820.7; 2241.9)	2481.4 (2256.6; 2706.2)	14.37 (13.11; 15.61)	1909.2 (2368.3; 2858.0)	23.17 (19.74; 26.59)
PAL (%) (physically active)	25.04 (22.48; 27.79)	23.12 (19.88; 26.36)	61.29 (59.46; 63.11)	73.67 (72.09; 75.25)	25.82 (24.32; 27.34)	23.14 (21.69; 24.58)	10.00 (2.48; 32.66)
Alchool consumption (doses)	14.89 (9.88; 21.83)	20.00 (7.64; 43.02)	24.65 (22.13; 27.16)	26.41 (24.54; 28.28)	11.68 (6.16; 21.08)	25.51 (22.33; 28.68)	40.1 (34.8;45.5)
None	71.94 (63.89;78.79)		16.67 (8.10; 31.20)	18.73 (14.88; 23.30)		19.51 (10.00; 34.57)	49.4 (43.9;54.8)
1 a 7	24.46 (18.01;32.30)	32.5 (27.6;37.8)			45.6 (40.2; 51.0)		10.5 (7.6; 14.4)
8 or more	3.59 (1.50;8.36)	49.8 (44.4; 55.3)		56.2 (50.7; 61.5)	37.3 (32.3;42.7)		
		17.6 (13.9;22.2)	45.1 (39.8; 50.4)	35.2 (30.2;40.5)	17.1 (13.4;21.6)	48.0 (42.7;53.4)	
			43.8 (38.6;49;3)	8.6 (6.0; 12.2)		42.3 (37.1;47.7)	
			11.0 (8.1;14.9)			9.6 (6.9;13.3)	

PAL: Physical activit level.

Regarding the food classification groups by degree of processing, we observed lower averages in the third tertile of the MP group for interleukins IL-10, IL-6, IL-8. In the highest tertile of MP, 25OHD showed higher mean levels and a lower proportion of high-dose alcoholic beverage consumption (Table 1).

For the UPF group, a dose-response was observed for the cytokines IL6 and IL1- β and for the consumption of high doses of alcohol, and lower levels of 25OH D (Table 1).

After adjusting for confounders, we found that being in the third tertile of MP consumption was linked to lower mean levels of IL1- β (β = -1.69 pg/mL, 95% CI = -3.10; -0.28), IL-6 (β = -1.16 pg/mL, 95% CI = -2.50; 0.17), and TNF- α (β = -1.79 pg/mL, 95% CI = -3.45; -0.14). See Fig. 2 and **Supplementary Material 1**.

For UPF consumption, higher levels were linked to increased inflammatory markers: TNF- α (β = 2.30 pg/mL, 95% CI = 0.57; 4.02), IL-6 (β = 2.58 pg/mL, 95% CI = 1.25; 3.91), IL12p-70 (β = 2.56 pg/mL, 95% CI = 1.17; 3.95), and IL1- β (β = 3.13 pg/mL, 95% CI = 1.68; 4.59). See Fig. 3 and **Supplementary Material 1**.

Discussion

This study highlights data from postmenopausal women and identifies the association between food consumption by degree of processing and inflammatory markers. The main findings indicate that women who consumed foods from the minimally processed group showed a negative association with pro-inflammatory markers. In contrast, the highest tertiles of UPF consumption were positively associated with average levels of pro-inflammatory cytokines. These results confirm our initial hypothesis, suggesting that dietary patterns dominated by ultra-processed foods are associated with a heightened pro-inflammatory state in this population.

Hormonal reduction is one of the main physiological changes associated with menopause. Estradiol is a hormone with extensive metabolic effects that affects the central nervous system and increases food intake and caloric energy consumption(6, 23), increasing visceral fat, with a consequent increase in the secretion of pro-inflammatory signaling molecules, inducing local and low-grade systemic inflammation(24).

This persistent inflammatory state can be aggravated by external factors, especially by dietary patterns characterized by high consumption of ultra-processed foods (UPFs). Higher consumption of these foods is frequently associated with elevated levels of systemic inflammatory biomarkers, particularly CRP/hs-CRP (C-reactive protein/high-sensitivity C-reactive protein) in adults and in some pediatric contexts. Relationships with IL-6 and TNF- α are observed in specific populations, while IL-1 β , MCP-1, PAI-1, and leptin show inconsistent patterns (7, 11).

The inflammatory state promoted by high consumption of UPFs can be explained by both their low nutritional quality and non-nutritional factors, such as additives (25). A diet rich in UPFs often implies a higher intake of added sugars, saturated fats, and trans fatty acids, contributing to inflammatory reactions by the immune system (26).

In addition, the high intake of simple sugars, present in UPFs as sucrose and high-fructose syrup, can lead to a rapid increase in postprandial blood glucose (27). In response, increased insulin secretion can trigger inflammatory effects in the body (12, 28). Despite the scarcity of literature on UPF consumption in postmenopausal women and on inflammatory biomarkers, a study evaluating middle-aged to elderly adults observed significant effects of ultra-processed foods and beverages on higher levels of TNF α , IL-6, leukocyte and neutrophil counts, basophil constituents, and the neutrophil-to-lymphocyte ratio (29).

Regarding lipid content, in addition to their higher consumption compared to other foods, their low nutritional quality contributes to inflammatory responses (12, 26). A study conducted in elderly individuals with metabolic syndrome observed that high UPF consumption was associated with higher intake of saturated and trans fatty acids and a pro-oxidant and pro-

inflammatory state, with elevated levels of TNF α , IL-6, IL-15, and leptin, and lower CAT and SOD activity, compared to those with lower intake (30).

In addition, UPFs typically replace the consumption of MP foods, such as fruits and vegetables, which are sources of vitamins C, D, E, selenium, carotenoids, and phytochemicals that act as antioxidants in reducing reactive species, molecules capable of causing inflammatory reactions, and the development of chronic diseases (12, 31, 32).

Other mechanisms of action of UPFs involve the presence of additives (such as emulsifiers, artificial colors, non-nutritive sweeteners), compounds derived from processing (e.g., acrylamide, advanced glycation end products) and contaminants from packaging (e.g., bisphenols), which exert their effects through disruption of the microbiota, impairment of the intestinal barrier, endotoxemia and activation of the innate immune system (33, 34).

This study has potential limitations. First, our findings should be interpreted with caution because this was a cross-sectional study, and establishing a causal relationship between consumption and cytokines was not possible; in particular, it is not possible to exclude reverse causation. In addition to the method's inherent limitations, certain issues were observed in the use of FFQ. The pre-established list of foods was not designed with the NOVA classification in mind; thus, it was difficult to classify food preparations, since a single preparation can contain ingredients from all NOVA food groups. We opted to classify preparations by the major ingredient's category, which may have led to underestimation or overestimation of ultra-processed food consumption.

According to this study's findings, the menopausal period is characterized by an inflammatory and metabolic vulnerability, mainly due to hormonal changes, which can be aggravated by inadequate dietary patterns, such as high consumption of UPFs. In view of this, the importance of encouraging reductions in UPF consumption and increases in MP as a strategy to promote the health of menopausal women is highlighted, with the aim of modulating systemic inflammation and preventing chronic diseases.

Declarations

Acknowledgment

The authors thank all the volunteers who contributed to the study and National Council for Scientific and Technological Development (CNPq process n. 481418/2011-3) for financially funding the project and also Foundations for Supporting Research in the states of Minas Gerais (FAPEMIG process n. APQ-00296-12).

Author Contributions

The authors' responsibilities were as follows GZL: designed research; GZL, DCGS, FGF conceived the study; DCGS, FGF: managed data collection; LLO: performed laboratory tests; GZL: analyzed the data; GZL, DCGS, FGF, EKSA: wrote paper; GZL, DCGS, FGF, DMLSG, EKSA, KDSRR, KCMSE; Had primary responsibility for final content: GZL, YMFM; Postdoctoral advisor: YMFM. All authors have read and approved the final manuscript.

Data availability

The data supporting the findings of this study are available in the database of the principal investigator; however, restrictions apply to the availability of these data, which were used under license for the current study and are therefore not publicly available. The data are, nevertheless, available upon request and with the permission of the principal investigator.

Funding Sources

This study was not funded by any agency or entity. However, it was conducted using the data from a project called "Metabolic syndrome and associated factors: a population-based study in adults from Viçosa, MG". The project was funded

by the National Council for Scientific and Technological Development, grant number 481418/2011-3 and by Minas Gerais State Research Support Foundation grant number APQ-00296-12.

Ethics approval and consent to participate

This study was conducted in accordance with the Declaration of Helsinki. The research project was approved by the Research Ethics Committee of the Federal University of Viçosa (Official Letter 02/2013). All participants signed the informed consent form.

Consent for publication

Not applicable. (*No identifying patient images or personal details are included in this manuscript*).

Author Declarations

The authors declare no conflicts of interest

Declaration of generative ai and ai-assisted technologies

During the preparation of this work, the authors used Chat- GPT (OpenAI) and Grammarly (Grammarly Inc.) to improve the manuscript's readability and language. All content was subsequently reviewed and edited by the authors, who take full responsibility for the final version.

References

1. Santoro N, Roeca C, Peters BA, Neal-Perry G. The Menopause Transition: Signs, Symptoms, and Management Options. *Journal of Clinical Endocrinology and Metabolism*. Endocrine Society; 2021. pp. 1–15. 10.1210/clinem/dgaa764 . PubMed PMID: 33095879.
2. Davis SR, Lambrinoudaki I, Lumsden M, Mishra GD, Pal L, Rees M, et al. Menopause *Nat Rev Dis Primers*. 2015;1:1–19. 10.1038/nrdp.2015.4 . PubMed PMID: 27188659.
3. Raj A, Chakole S, Agrawal S, Gupta A, Khekade H, Prasad R, et al. The Impact of Menopause on Cardiovascular Aging: A Comprehensive Review of Androgen Influences. *Cureus*. 2023 Aug;16. 10.7759/cureus.43569.
4. Bochynska S, García-Pérez MÁ, Tarín JJ, Szeliga A, Meczekalski B, Cano A. The Final Phases of Ovarian Aging: A Tale of Diverging Functional Trajectories. *Journal of Clinical Medicine*. Multidisciplinary Digital Publishing Institute (MDPI); 2025. 10.3390/jcm14165834.
5. Nappi RE, Simoncini T. Menopause transition: a golden age to prevent cardiovascular disease. *Lancet Diabetes Endocrinol*. 2021;9(3):135–7. 10.1016/S2213-8587(. 21)00018-8 PubMed PMID: 33485472.
6. Erdélyi A, Pálfi E, Túú L, Nas K, Szűcs Z, Török M, et al. The Importance of Nutrition in Menopause and Perimenopause—A Review. *Nutrients*. Multidisciplinary Digital Publishing Institute (MDPI); 2024. 10.3390/nu16010027 . PubMed PMID: 38201856.
7. Furman D, Campisi J, Verdin E, Carrera-Bastos P, Targ S, Franceschi C, et al. Chronic inflammation in the etiology of disease across the life span. *Nat Med*. 2019;25(12):1822–32. 10.1038/s41591-019-0675-0 . PubMed PMID: 31806905.
8. James SL, Abate D, Abate KH, Abay SM, Abbafati C, Abbasi N, et al. Global, regional, and national incidence, prevalence, and years lived with disability for 354 diseases and injuries for 195 countries and territories, 1990–2017: a systematic analysis for the Global Burden of Disease Study 2017. *Lancet*. 2018;392(10159):1789–858. 10.1016/S0140-6736(18)32279-7.
9. Noel SE, Mangano KM, Mattei J, Griffith JL, Dawson-Hughes B, Bigornia S, et al. Dietary Approaches to Stop Hypertension, Mediterranean, and Alternative Healthy Eating indices are associated with bone health among Puerto

- Rican adults from the Boston Puerto Rican Osteoporosis Study. *Am J Clin Nutr.* 2020;111(6):1267–77. 10.1093/ajcn/nqaa090 . PubMed PMID: 32386194.
10. Lane MM, Gamage E, Du S, Ashtree DN, McGuinness AJ, Gauci S, et al. Ultra-processed food exposure and adverse health outcomes: Umbrella review of epidemiological meta-analyses. *BMJ.* 2024. 10.1136/bmj-2023-077310 . PubMed PMID: 38418082.
 11. Juul F, Parekh N, Martinez-Steele E, Monteiro CA, Chang VW. Ultra-processed food consumption among US adults from 2001 to 2018. *Am J Clin Nutr.* 2022;115(1):211–21. 10.1093/ajcn/nqab305 . PubMed PMID: 34647997.
 12. Tristan Asensi M, Napoletano A, Sofi F, Dinu M. Low-Grade Inflammation and Ultra-Processed Foods Consumption: A Review. *Nutrients.* Multidisciplinary Digital Publishing Institute (MDPI); 2023. 10.3390/nu15061546 . PubMed PMID: 36986276.
 13. Esposito S, Gialluisi A, Di Castelnuovo A, Costanzo S, Pepe A, Ruggiero E, et al. Ultra-processed food consumption is associated with the acceleration of biological aging in the Moli-sani Study. *Am J Clin Nutr.* 2024;120(6):1432–40. 10.1016/j.ajcnut.2024.10.006 . PubMed PMID: 39500680.
 14. Ilesanmi-Oyelere BL, Kruger MC. Nutrient and dietary patterns in relation to the pathogenesis of postmenopausal osteoporosis—a literature review. *Life.* MDPI AG; 2020. pp. 1–13. 10.3390/life10100220.
 15. Ilesanmi-Oyelere BL, Kruger MC. Associations between dietary patterns and an array of inflammation biomarkers and plasma lipid profile in postmenopausal women. *BMC Womens Health.* 2023;23(1):1–8. 10.1186/s12905-023-02417-w PubMed PMID: 37173732.
 16. Segheto W, da Silva DCG, Coelho FA, Reis VG, Morais SHO, Marins JCB, et al. *Nutr Hosp.* 2015;32(1):101–9. PubMed PMID: 26262703. Índice De Adiposidad Corporal Y Factores Asociados En Adultos: Método Y Logística De Un Estudio Poblacional.
 17. Valmorbida A, Longo GZ, Nascimento GM, De Oliveira LL, De Moraes Trindade EBS. Association between cytokine levels and anthropometric measurements: A population-based study. *Br J Nutr.* 2023;129(7):1119–26. doi:10.1017/S0007114522002148 PubMed PMID: 35856255.
 18. Pardini R, Matsudo S, Araújo T, Matsudo V, Andrade E, Braggion G et al. Validação do questionário internacional de nível de atividade física (IPAQ-versão 6): estudo piloto em adultos jovens brasileiros Validation of the International Physical Activity Questionnaire (IPAQ version 6): pilot study in Brazilian young adults. Report.
 19. Blanton CA, Moshfegh AJ, Baer DJ, Kretsch MJ. The USDA Automated Multiple-Pass Method Accurately Estimates Group Total Energy and Nutrient Intake 1,2. *The Journal of Nutrition* Nutritional Epidemiology J. Nutr. 2006. Report.
 20. da Silva DCG, Fiates GMR, Botelho AM, Vieira FGK, Medeiros KJ, Willecke RG, et al. Food consumption according to degree of food processing, behavioral variables, and sociodemographic factors: Findings from a population-based study in Brazil. *Nutrition.* 2022;93. 10.1016/j.nut.2021.111505 . PubMed PMID: 34763312.
 21. WILLETT W. *Nutritional epidemiology.* 2dt ed. New York: Oxford University Pres. 1998. 1–514 p.
 22. Alves M, Bastos M, Leitão F, Marques G, Ribeiro G, Carrilho F. Vitamina D—importância da avaliação laboratorial. *Revista Portuguesa de Endocrinologia Diabetes e Metabolismo.* 2013;8(1):32–9. 10.1016/j.rpedm.2012.12.001.
 23. Crewe C, An YA, Scherer PE. The ominous triad of adipose tissue dysfunction: Inflammation, fibrosis, and impaired angiogenesis. *Journal of Clinical Investigation.* American Society for Clinical Investigation; 2017. pp. 74–82. 10.1172/JCI88883 PubMed PMID: 28045400.
 24. Juul F, Martinez-Steele E, Parekh N, Monteiro CA. The role of ultra-processed food in obesity. *Nat Reviews Endocrinol Nat Res.* 2025;672–85. 10.1038/s41574-025-01143-7 . PubMed PMID: 40659796.
 25. Christ A, Lauterbach M, Latz E. Western Diet and the Immune System: An Inflammatory Connection. *Immunity. Cell;* 2019. pp. 794–811. 10.1016/j.immuni.2019.09.020 PubMed PMID: 31747581.
 26. Atkinson FS, Brand-Miller JC, Foster-Powell K, Buyken AE, Goletzke J. International tables of glycemic index and glycemic load values 2021: a systematic review. *American Journal of Clinical Nutrition.* Oxford University Press; 2021.

- pp. 1625–32. 10.1093/ajcn/nqab233 PubMed PMID: 34258626.
27. Dror E, Dalmas E, Meier DT, Wueest S, Thévenet J, Thienel C, et al. Postprandial macrophage-derived IL-1 β stimulates insulin, and both synergistically promote glucose disposal and inflammation. *Nat Immunol.* 2017;18(3):283–92. 10.1038/ni.3659 . PubMed PMID: 28092375.
 28. Millar SR, Harrington JM, Perry IJ, Phillips CM. Associations between ultra-processed food and drink consumption and biomarkers of chronic low-grade inflammation: exploring the mediating role of adiposity. *Eur J Nutr.* 2025;64(4). 10.1007/s00394-025-03666-1 . PubMed PMID: 40205185.
 29. Quetglas-Llabrés MM, Monserrat-Mesquida M, Bouzas C, Mateos D, Ugarriza L, Gómez C, et al. Oxidative Stress and Inflammatory Biomarkers Are Related to High Intake of Ultra-Processed Food in Old Adults with Metabolic Syndrome. *Antioxidants.* 2023;12(8). 10.3390/antiox12081532.
 30. Brauer M, Roth GA, Aravkin AY, Zheng P, Abate KH, Abate YH, et al. Global burden and strength of evidence for 88 risk factors in 204 countries and 811 subnational locations, 1990–2021: a systematic analysis for the Global Burden of Disease Study 2021. *Lancet.* 2024;403(10440):2162–203. 10.1016/S0140-6736(24)00933-4.
 31. Millar SR, Navarro P, Harrington JM, Shivappa N, Hébert JR, Perry IJ, et al. Dietary score associations with markers of chronic low-grade inflammation: a cross-sectional comparative analysis of a middle- to older-aged population. *Eur J Nutr.* 2022;61(7):3377–90. 10.1007/s00394-022-02892-1 . PubMed PMID: 35511284.
 32. Mignogna C, Costanzo S, Di Castelnuovo A, Ruggiero E, Shivappa N, Hebert JR, et al. The inflammatory potential of the diet as a link between food processing and low-grade inflammation: An analysis on 21,315 participants to the Moli-sani study. *Clin Nutr.* 2022;41(10):2226–34. 10.1016/j.clnu.2022.08. .020 PubMed PMID: 36081297.
 33. Mariath AB, Machado AD, Ferreira L do, Ribeiro NM. The possible role of increased consumption of ultra-processed food products in the development of frailty: A threat for healthy ageing? *Br J Nutr.* 2022;128(3):461–6. doi:10.1017/S0007114521003470 PubMed PMID: 34503590.
 34. Seto T, Grondin JA, Khan WI. Food Additives: Emerging Detrimental Roles on Gut Health. *FASEB J* John Wiley Sons Inc. 2025. 10.1096/fj.202500737. R PubMed PMID: 40622070.

Figures

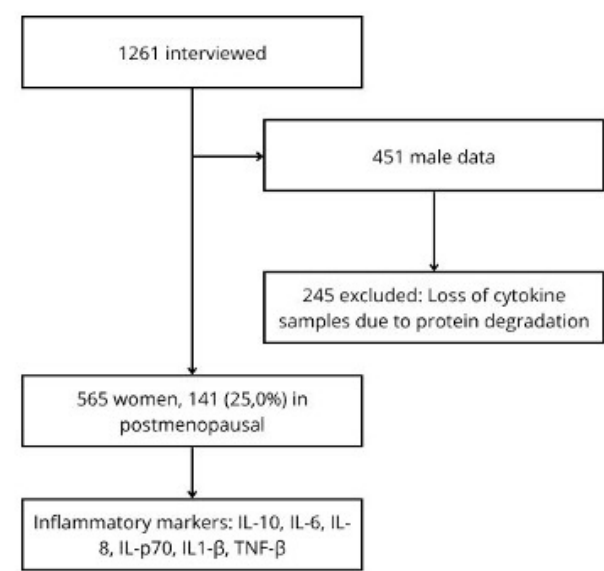


Figure 1

Flowchart of participant selection. City of Viçosa, State of Minas Gerais, Brazil.

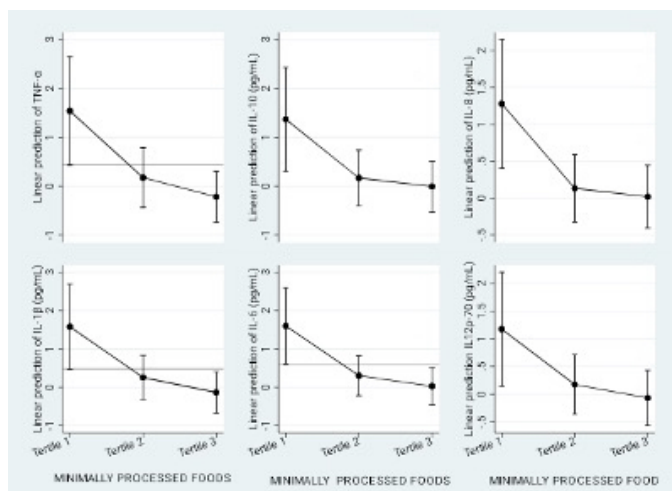


Figure 2

Multivariate regression between minimally processed food (main independent variables) with inflammatory markers (dependent variable): in menopausal women. Study on Health and Food (SHF), Viçosa (MG), Brazil, 2012-2014.

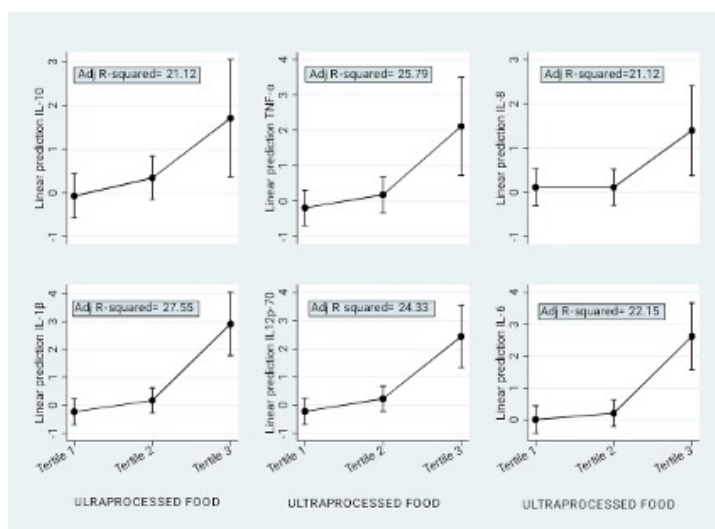


Figure 3

Multivariate regression between ultraprocessed food (main independent variables) with inflammatory markers (dependent variable): in menopausal women. Study on Health and Food (SHF), Viçosa (MG), Brazil, 2012-2014.

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- [Supplementarymaterial.docx](#)
- [graphicalabstract.tiff](#)