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ORIGINAL ARTICLE

Association of Dietary Inflammatory Potential in Metabolically Healthy and Metabolically Unhealthy Obese Individuals

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Abstract

Introduction: Currently, two types of phenotypes have been recognized in individuals who are obese. Among the factors related to lifestyle, diet has a relevant influence, although there is no consensus regarding the role of diet in metabolic phenotypes; furthermore, diet is a strong moderator of chronic systemic inflammation.

Objective: Investigate dietary inflammatory potential between metabolic phenotypes and to compare the differences between anti-inflammatory and pro-inflammatory diets in individuals with the same phenotype.

Methods: This is a cross-sectional observational study that utilized the database of 533 individuals divided into 4 groups, according to metabolic phenotype and dietary inflammatory characteristic. Sociodemographic, clinical, anthropometric and biochemical characteristics were evaluated and the inflammatory index of the diet was calculated.

Results: The mean Dietary Inflammatory index (DII) of the total sample was 0.974±1.02, with a maximum of 4.34 and a minimum of –1.74. In the metabolically unhealthy groups, we found a statistical difference in relation to systolic blood pressure when comparing the anti-inflammatory [median 120 (110.0-130.0)] and pro-inflammatory diets [median 130 (120.0-140.0); p = 0.022], and mean isoprostane concentrations were lower in the metabolically healthy group with anti-inflammatory diet. In regression analysis, the only variable that demonstrated a higher risk of alterations in all groups when compared to the metabolically healthy and anti-inflammatory group were isoprostane concentrations.

Conclusion: We are able to conclude that an anti-inflammatory diet is associated with lower oxidative stress in metabolically healthy obese, and a pro-inflammatory diet is associated with higher systolic blood pressure values.

Keywords: Obesity; Metabolic Syndrome; Inflammation.

Introduction

Obesity has become a major health problem worldwide, and it is strongly associated with a series of diseases such as insulin resistance, type 2 diabetes, asthma, arterial hypertension, atherosclerosis, certain forms of neoplasia, and ischemic heart disease, which reduce life expectancy and, together, have enormous economic and social consequences.[1,2]

Metabolic syndrome has been recognized as a complication of obesity, and it is associated with increased risk of diabetes, hypertension, and dyslipidemias. Insulin resistance, chronic inflammation, and increased oxidative stress play an essential role in the development of pathogenesis.[3,4] Nevertheless, there is a group of individuals who, in spite of the risks, do not develop metabolic syndrome.[4] This phenotype is called metabolically healthy obese. These individuals have a lower amount of visceral fat, smaller adipocytes, and lower levels of inflammation when compared to metabolically unhealthy obese individuals.[4]

Oxidative stress is referred to as excessive production of reactive oxygen species that the antioxidant system cannot neutralize.[5] Inflammation, on the other hand, is the response to threats received by the organism, which can be the entry of pathogens, chronic and autoimmune diseases, tobacco use, alcohol consumption, hypercaloric diet, and

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Central Illustration: Association of Dietary Inflammatory Potential in Metabolically Healthy and Metabolically Unhealthy Obese Individuals

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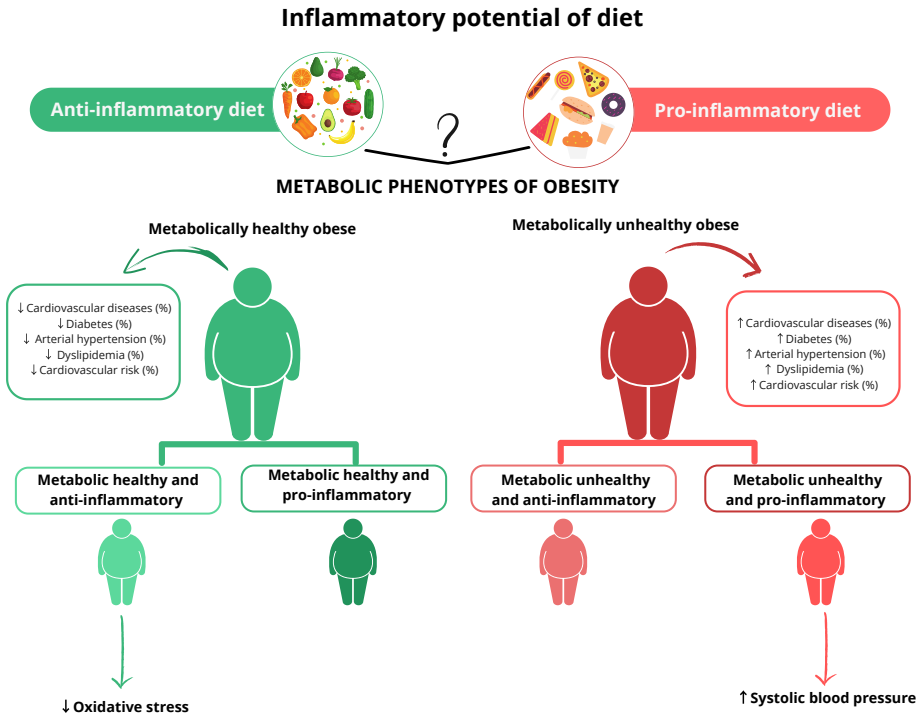

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others.[5] Many of these conditions favor the production of reactive oxygen species, resulting in oxidative stress and increased signaling of inflammatory molecules.[5]

Among the factors related to lifestyle, diet has a relevant influence, although there is no consensus regarding the role of diet in metabolic phenotypes.[6] Current evidence suggests that metabolically healthy individuals have similar intake of calories and macronutrients compared to metabolically unhealthy obese individuals.[6] Furthermore, diet is a strong moderator of chronic systemic inflammation; for instance, "unhealthy" dietary patterns (Western-style diets with high fat, refined carbohydrate, and protein content) are typically associated with higher concentrations of inflammatory markers, whereas "healthier" diets (for example, the Mediterranean diet high in fruits, vegetables, and fish) are associated with lower levels of inflammation.[7]

The Dietary Inflammatory Index (DII) was developed by researchers at the University of South Carolina to estimate overall dietary inflammatory potential based on extensive literature research that incorporates cell culture, animal, and epidemiological studies on the effects of diet on inflammation.[8] The DII has been shown to be associated with inflammation, specifically in concentrations of C-reactive protein, interleukin (IL)-6, and tumor necrosis factor alpha in adults.[9] The association between dietary inflammatory potential and metabolic syndrome continues to be highly controversial. In two systematic reviews, the results were contradictory.[10,11]

Thus, the objective of this study was to investigate dietary inflammatory potential between metabolic phenotypes and to compare the biochemical, anthropometric, clinical, and socioeconomic differences, as well as differences between anti-inflammatory and pro-inflammatory diets in individuals with the same metabolic phenotype.

Methods

Study design

This is a cross-sectional observational study that utilized the database of individuals treated from 2010 to 2019 at the Center for Research and Extension in Clinical Nutrition of the Clementino Fraga Filho University Hospital of the

Federal University of Rio de Janeiro. The study received approval from the Research Ethics Committee of the same institution, and it was registered under number Nº 062/10. All participants signed a free and informed consent form.

Population

The 533 individuals in the study were selected by convenience sample, according to the following eligibility criteria: individuals of both sexes between 20 and 59 years of age, body mass index (BMI) ≥25kg/m^2, with all clinical, socioeconomic, anthropometric, and dietary information available. Individuals with any of the following factors were excluded: elderly (≥60 years); healthy weight, younger than 20 years, triglycerides ≥400mg/dL with no history of genetic disease, neoplasms, AIDS, or any immunological disease. Women with energy consumption <500 kcal and >5000 kcal and men with energy consumption <800kcal and >6000kcal were also excluded in order to avoid inadequate reports regarding dietary consumption. Participants were divided into 4 groups, according to metabolic phenotype and dietary inflammatory characteristic, as follows: metabolic healthy and pro-inflammatory (MHPI); metabolic healthy and anti-inflammatory (MHAI); metabolic unhealthy and pro-inflammatory (MUPI); and metabolic unhealthy and anti-inflammatory (MUAI).

Socioeconomic and clinical data

Self-reported information on skin color, marital status, level of education, income, tobacco use, and clinical conditions such as hypertension and cardiovascular disease were provided by means of a standardized questionnaire developed by the research center and monitored by the researchers. Participants were classified as having diabetes if their blood glucose results met the diagnostic criteria of the 2019–2020 *Brazilian Diabetes Guidelines*[12] or if they were using hypoglycemic agents. Participants were classified as having dyslipidemia if their lipidogram results met the diagnostic criteria of the *Brazilian Guidelines on Dyslipidemias and Prevention of Atherosclerosis*.[13] Income was expressed per capita, and the values were updated to the minimum wage for the year 2020 in Brazil (1045.00 Brazilian reals).

Anthropometry and blood pressure

Weight and height were measured using an electronic scale with a capacity of 200kg and accuracy of 50g, with a stadiometer attached. The measurements were used to calculate BMI.[14] Waist circumference (WC) was measured from the midpoint between the last rib and the iliac crest, using an inelastic tape measure; participants were in orthostatic position, with abdomen relaxed and arms and feet close to the body.[14] Neck circumference (NC) was measured with participants standing, with their head positioned in the horizontal plane, encircling the neck with an inelastic tape measure at the midpoint, at the level of the cricothyroid cartilage, between the midpoint of the cervical spine to the mid-anterior of the neck. In men, the measurement was performed below the laryngeal prominence and applied perpendicular to the long axis of the neck.[15] Blood pressure was measured using a sphygmomanometer (Missouri, aneroid), an obese arm cuff, and a stethoscope (Missouri,duoscopic) by auscultation, after participants had remained seated for at least 5 minutes.[12,16]

Biochemical data

Blood samples were collected in the morning, after a fasting period of at least 12 hours and at most maximum 14 hours, and serum was obtained by centrifugation (4000rpm, 15 minutes). Aliquots were stored in a freezer at an appropriate temperature until lipid profile, blood glucose, insulin, and isoprostane analysis were analyzed. Biochemical analyses were performed in duplicate, by means of an automated method (Automatic Analyzer A25, BioSystems), using commercial BioSystems kits. Serum concentrations of glucose, triglycerides, total cholesterol, and HDL (high-density lipoprotein) were evaluated. LDL values were calculated following the formula proposed by Friedewald et al.,[17] which is only valid if triglyceride concentration is below 400mg/dL. Insulin was obtained from serum and analyzed by the ELISA method (Ultrasensitive Insulin ELISA Kit, DRG) on a BRIO 2 Radim device. Plasma concentrations of 8-isoprostane were determined by competitive assay with a Cayman kit (USA).

Dietary data

The dietary survey was performed using a 3-day food record. Dietary data were analyzed using Food Processor software, version 7.2, obtaining average consumption of macro- and micronutrients and bioactive compounds, while flavonoids were taken from the USDA Database for the Flavonoid Content of Selected Foods.[2]

DII

This tool was developed and validated by Shivappa et al.,[18] based on extensive literature research, where 45 dietary components were selected from 1943 articles that demonstrated pro-inflammatory, anti-inflammatory, and neutral effects through measurement of inflammatory cytokines, such as IL-1β, IL-4, IL-6, IL-10, tumor necrosis factor alpha α, and C-reactive protein. When developing the calculation, the authors established an inflammatory score for each component, the global dietary index being the sum of this score. The present study used the following 36 components: alcohol, vitamin B_{12}, B_6, B_9, B_3, B_2, B_1, beta-carotene, caffeine, carbohydrates, cholesterol, energy, total fat, fiber, garlic, iron, magnesium, mono- and polyunsaturated fatty acids, n-3 and n-6 fatty acids, onions, protein, saturated fat, selenium, trans fat, vitamin A, vitamin C, vitamin D, vitamin E, zinc, flavan-3-ol, flavones, flavonols, flavonones, and anthocyanidins. Dietary inflammatory potential ranges between the values +7.98 (strongly pro-inflammatory) and –8.87 (strongly anti-inflammatory). In this study, we classified "anti-inflammatory diet" as index equal to or less than zero and "pro-inflammatory diet" as index value greater than zero.[19]

Classification of metabolic phenotypes

Individuals were divided into metabolically healthy and metabolically unhealthy phenotypes according to the NCEP-ATP III classification, 2002.[20] The metabolically unhealthy phenotype has at least 3 of the following 5 criteria: central obesity (WC ≥88cm for women and ≥102cm for men), triglycerides ≥150mg/dL, HDL-c <50mg/dL for women and <40mg/dL for men, blood pressure ≥130/85mmHg, and fasting blood glucose >100mg/dL (include diagnosis of diabetes).

Cardiovascular risk classification

The Framingham risk score[21] was used to calculate cardiovascular risk, which was stratified as low, intermediate, high, and very high risk.[22]

Statistical analysis

Data are presented as frequency, mean, and standard deviation, as appropriate. The normality of quantitative variables was evaluated using the Kolmogorov-Smirnov test. For categorical variables, the chi-square test was used. For parametric quantitative variables, the t test and the ANOVA test were used, whereas, for non-parametric data, the Mann-Whitney test and the Kruskal-Wallis test with post-hoc Bonferroni were used. Multivariate logistic regression analysis was used to better evaluate the associations of variables and to control the effects of the variables of age, BMI, systemic arterial hypertension, dyslipidemia, others, level of education, skin color, marital status, and income. All statistical analyses were performed using the Statistical Package for Social Sciences for Windows, version 22.0 (SPSS Inc,Chicago, Illinois, USA). Results with p values ≤0.05 were considered statistically significant.

Results

We evaluated 533 volunteers, 237 in the MHPI, 44 in the MHAI group, 202 in the MUPI group, and 50 in the MUAI group. In the supplementary material we display the characteristics of the study groups. The groups with metabolically unhealthy phenotype presented higher frequency of illiteracy, higher per capita income and higher frequency of cardiovascular diseases, diabetes, systemic arterial hypertension, dyslipidemia and higher cardiovascular risk in relation to metabolically healthy phenotypes. Regarding energy intake, we did not observe a significant difference between the groups studied, however, a higher consumption of antioxidant vitamins (A, C and E) and flavonoids was verified in the MHAI and MUAI groups, respectively.

The mean DII of the total sample was 0.974±1.02, with a maximum of 4.34 and a minimum of –1.74. By separating the groups according to the metabolic phenotype and inflammatory characteristic of the diet, we observed a significant difference between the groups with the same metabolic phenotype (MHAI versus MHPI and MUPI versus MUAI; p<0.05) and between the MHAI versus MUPI and MHPI versus MUAI groups (Table 1).

When comparing the groups of the same metabolic phenotype, we found that there was no statistical difference in relation to anthropometric, biochemical, and blood pressure measurements between the metabolically healthy groups with the pro-inflammatory and anti-inflammatory diets. In the metabolically unhealthy groups, we found a statistical difference in relation to systolic and diastolic blood pressure. When comparing the 4 groups, we observed a difference between the anthropometric (BMI, WC and NC) and biochemical (Glycemia, HDL and triglycerides) parameters between the groups with unhealthy metabolic phenotype in relation to the healthy metabolic phenotype and pro-inflammatory

Table 1 – Comparison of DII, anthropometry, biochemistry, and blood pressure between groups.

Characteristics/ metabolic phenotype and DII	MHAI (n = 43)	MHPI (n = 237)	p[1]	MUAI (n = 50)	MUPI (n = 203)	p[1]	p[2]
DII	-0.52 ± 0.42 [a]	1.29 ± 0.80 [d]	< 0.001	–0.55 ± 0.45	1.30 ± 0.79	< 0.001	< 0.001
BMI (kg/m²)	34.23 (31.53-37.87)	32.67 (29.43-37.12) [c,d]	0.293	35.94 (31.95-39.95)	36.03 (32.64 - 40.79)	0.562	< 0.001
WC (cm)	105.75 (92.12-112.75)	100.0 (91.75-110.00) [c,d]	0.453	106.0 (97.45-117.25)	107.75 (99.37-116.50)	0.883	< 0.001
NC (cm)	36.0 (34.62-38.5) [a,b]	36.3 (34.5-39.0) [c,d]	0.653	38.5 (36.0-42.12)	38.0 (36.0- 40.0)	0.311	< 0.001
Blood glucose (mg/dL)	87.5 (83-97.25)[a]	88.0 (80.0-95.0) [c,d]	0.992	98.0 (91.75 -117.50)	98.0 (88-110.25)	0.492	< 0.001
Total cholesterol (mg/dL)	202.0 (166.5 – 227.0)	193.0 (164.0 – 226.5)	0.648	209.0 (169.50 – 227.75)	205 (178.0- 235.5)	0.262	0.081
HDL-c (mg/dL)	50.50 (43.0 – 54.0) [a,b]	51.0 (42.0 -57.0) [c,d]	0.650	43.0 (37.50 – 47.25)	41.0 (38.0 – 47.0)	0.672	< 0.001
LDL-c (mg/dL)	114.5 (97.25 158.25)	122.0 (96.5 152.0)	0.995	118.0 (95.50 – 149.0)	127.0 (101.75 – 152.25)	0.193	0.365
Triglycerides (mg/dL)	101.5 (77.5 – 136.75) [a,b]	106.0 (76.5 – 130.5) [c,d]	0.583	167.0 (127.75 – 224.25)	162.0 (119.0 – 213.25)	0.633	< 0.001
Isoprostane (pg/mL)	13.50 (6.57 – 21.62)	20.70 (9.8 – 28.6)	0.023	22.00 (7.35 -27.9)	18.15 (11.25 -29.45)	0.827	0.138
Systolic blood pressure (mmHg)	110 (100.0 – 120.0) [a]	120.0 (110.0 – 120.0) [c]	0.097	120 (110.0 130.0)	130 (120.0 – 140.0)	0.001	< 0.001
Diastolic blood pressure (mmHg)	80 (70.0 – 80.0) [a]	80.0 (70.0 – 80.0) [c]	0.277	80.0 (70.0 – 80.0)	80.0 (80.0 – 90.0)	0.022	< 0.001

Values are shown as mean and standard deviation, median and interquartile range. [1]p – value - Comparison of means between groups of the same metabolic phenotype (MHPI versus MHAI or MUPI versus MUAI): T test for independent samples for parametric variables and Mann Whitney test for nonparametric variables. [2] p – value. Comparison of averages between groups studied: Kruskal-Wallis for non-parametric quantitative variables, and ANOVA for parametric variables, with post hoc Bonferroni test. Considering significant difference for p<0.05. [a] p < 0.05 between MHAI and MUPI; [b] p < 0.05 between MHAI and MUAI; [c] p < 0.05 between MHPI and MUPI; [d] p < 0.05 between MHPI and MUAI. BMI: body mass index; HDL-c: high-density lipoprotein; LDL-C: low-density lipoprotein; MHAI: metabolically healthy and anti-inflammatory; MHPI; metabolically healthy and pro-inflammatory; MUAI; metabolically unhealthy and anti-inflammatory; MUPI: metabolically unhealthy and pro-inflammatory; DII: dietary inflammatory index; WC: waist circumference; NC: Neck circumference.

diet. The group with healthy metabolic phenotype and anti-inflammatory diet also had significantly lower concentrations of glucose and triglycerides and higher concentrations of HDL cholesterol compared to the 2 groups with unhealthy metabolic phenotype (Table 1).

When evaluating the serum concentrations of isoprostanes, we found that the metabolically healthy and anti-inflammatory group had significantly lower values when compared to the metabolically healthy and pro-inflammatory (p = 0.023), however, we did not observe statistical difference when comparing the four study groups, as displayed in Table 2.

In regression analysis (Table 2), the only variable that demonstrated a higher risk of alterations in all groups when compared to the metabolically healthy and anti-inflammatory group were isoprostane concentrations. When comparing the unhealthy groups, we found that participants with a pro-inflammatory diet had a greater likelihood of having higher systolic blood pressure values.

Discussion

This study evaluated the association of the metabolic phenotypes of obesity with dietary inflammatory potential. We demonstrated that metabolically healthy individuals on an anti-inflammatory diet had lower concentrations of isoprostanes. Furthermore, when compared to the metabolically healthy and the anti-

Table 2 – Logistic regression analysis of clinical, anthropometric, and biochemical variables between groups.

Variable characteristics	MHAI (n = 43)	MHPI (n = 237)	MUAI (n = 50)	MUPI (n = 203)	MHAI *versus* MHPI
WC	1 (Reference)	1.009(0.962-1.059)	0.981 (0.919-1.047)	0.975 (0.921-1.031)	0.994 (0.950-1.040)
Blood glucose	1 (Reference)	1.016(0.985- 1.048)	1.054 (1.018-1.091)*	1.058 (1.022-1.094)*	1.003 (0.994-1.013)
Triglycerides	1 (Reference)	0.996(0.989-1.003)	1.021 (1.011-1.030)*	1.021 (1.012-1.029)*	1.000 (0.995-1.005)
HDL-c	1 (Reference)	0.999(0.973-1.025)	0.926 (0.884-0.970)*	0.905 (0.870-0.942)*	0.978 (0.939-1.017)
Isoprostane	1 (Reference)	1.089(1.013-1.170)*	1.094 (1.009-1.186)*	1.089 (1.009-1.175)*	0.995 (0.961-1.030)
SBP	1 (Reference)	1.021(0.984-1.059)	1.007 (0.960-1.057)	1.049 (1.006-1.093)*	1.041 (1.008-1.076)*
DBP	1 (Reference)	1.006(0.961-1.052)	1.023 (0.962-1.087)	1.023 (0.972-1.078)	1.001 (0.959-1.045)

Multinomial logistic regression with adjustment for the variables of age, BMI, systemic arterial hypertension, dyslipidemia, others, level of education, skin color, marital status, and income. Values with (*) showed statistical significance with p<0.05. DBP: diastolic blood pressure; HDL-c: high-density lipoprotein; SBP: systolic blood pressure. MHAI: metabolically healthy and anti-inflammatory; MHPI; metabolically healthy and pro-inflammatory; MUAI; metabolically unhealthy and anti-inflammatory; MUPI: metabolically unhealthy and pro-inflammatory; WC: waist circumference.

inflammatory groups, the other groups showed a higher risk of showing changes in this marker. In addition, individuals with unhealthy metabolic phenotype and pro-inflammatory diet were more likely to have higher systolic blood pressure values.

As expected, the groups with unhealthy metabolic phenotypes had higher abdominal adiposity, blood glucose, blood pressure, and lipid profile alterations when compared to groups with healthy metabolic phenotypes. These results are consistent with previous studies that showed differences in these individuals' anthropometric and biochemical characteristics.[23,24] Some factors have been suggested to explain the profile of metabolically healthy obesity. For instance, the smaller size of adipocytes, lower ectopic fat accumulation, and greater intestinal integrity are associated with reduced systemic inflammation, and greater insulin sensitivity.[25]

Dietary pattern has also been associated with the development of obesity phenotypes. In 2019, Kouvari et al. evaluated data from the ATTICA study in 1514 men and 1528 women over 18 years of age in Athens, Greece and observed that 52% of participants developed an unhealthy metabolic state during the 10-year follow-up, and metabolically healthy obesity was independently associated with increased risk of cardiovascular disease in participants with low adherence to the Mediterranean diet. Subsequently, the same researchers showed an inverse association between DII and transition from a healthy to unhealthy metabolic state and diabetes, concluding that a diet with a high anti-inflammatory load appears to be an effective preventive measure to maintain a metabolically healthy state.[26,27]

Systematic reviews evaluating the relationship between DII and metabolic syndrome have reported a high degree of heterogeneity.[10,11] These results may be due to the difference in the study populations, with different habits, lifestyles, and cultures.

Few studies in the literature have associated DII with phenotype of obesity, which makes comparisons difficult. A study carried out in Iran with 300 obese individuals, mostly female, with a mean age of 43 years, mostly classified as metabolically unhealthy (63.5%) according to the criteria used in our study, showed that the mean DII of this sample was –0.33 ± 1.60,[28] and they observed an increased likelihood for the unhealthy metabolic phenotype as the diet became more pro-inflammatory.[28] In general, our sample showed a DII with more pro-inflammatory characteristics, but, when comparing individuals with the same metabolic phenotype according to dietary inflammatory potential, we did not observe differences in the metabolic characteristics of these individuals. Park et al., investigating the association of DII scores, metabolic phenotypes, and mortality risk in overweight/obese individuals from a representative sample of the United States, showed that a more pro-inflammatory diet is associated with an increased risk of all-cause mortality and cardiovascular mortality, only in the metabolically unhealthy obese phenotype,[29] which leads us to question whether this association is influenced by a less healthy lifestyle and not only by the dietary inflammatory characteristic.

In order to expand knowledge on this topic, we divided the volunteers into 4 groups according to metabolic phenotype and dietary inflammatory. To our knowledge, this is the first study in Brazil to evaluate this association. When evaluating dietary inflammatory potential, we observed a variation of –1.74 to +4.34, different from other studies carried out in Brazil, which found variations of –4.77 to +5.98,[30] –5.48 to +4.55,[31] and –4.69 and +5.28.[32] This can be explained by the fact that our population was composed of individuals with obesity, while others were from a general population. According to the results of the study by Pereira et al.,[30] the Brazilian population consumes a diet with high inflammatory potential, especially adolescents who are overweight and obese, white people, and people with higher income and education. The fact that these researchers included adolescents in their study may justify the higher DII values when compared to our results. According to the authors, this may be due to the fact that adults and elderly individuals with chronic diseases tend to improve the quality of their diet as they become more concerned about health.[30] Other studies carried out in young adults in Brazil found that pro-inflammatory dietary patterns were associated with a higher prevalence of overweight and obesity in both men and women,[31] whereas the study conducted by Carvalho et al., with adults 23 to 25 years of age in Ribeirão Preto, Brazil, did not find the association of the DII with insulin resistance and metabolic syndrome, probably because these individuals were young and the effects of a pro-inflammatory diet on the development of these outcomes had not yet manifested.[32]

When comparing the unhealthy phenotype groups, we found a significant difference in relation to blood pressure and, when moving to regression analysis, we continued to observe this difference in systolic blood pressure, which leads us to suggest that dietary inflammatory capacity may influence the increase in blood pressure among unhealthy individuals. In fact, a 2018 systematic review concluded that the prevalence of hypertension and systolic blood pressure measurements are higher in more pro-inflammatory dietary categories, which was not seen in diastolic blood pressure.[33] However, other studies have found a weak association between DII and the prevalence of hypertension in adult women,[34] as well as a weak association between DII and blood pressure in a sample of adolescents.[35]

Finally, we verified whether the concentrations of isoprostanes, a marker of oxidative stress, were different between groups according to the metabolic phenotype and according to the inflammatory capacity of the diet. Isoprostane has been considered a good marker of oxidative stress, given that it is specific for lipid peroxidation, chemically stable, found in detectable amounts in tissues and biological fluids, and not affected by the lipid content of the diet. Elevated concentrations of this marker were found in patients with several cardiovascular risk factors such as diabetes, obesity, hypercholesterolemia, and smoking.[36] An article published analyzing the Framingham study volunteers reached the conclusion that obesity is associated with a state of excessive oxidative stress, which may contribute to the prevalence of cardiovascular disease in this population.[37] When evaluating the association between obesity phenotypes and oxidative stress markers, Lejawa et al., found that total oxidation state and total antioxidant capacity (TAC), as well as concentrations of lipid hydroperoxides were significantly related to metabolically unhealthy obesity.[38] Jakubiak et al., evaluated young individuals, between 18 and 36 years of ages, without any history of chronic diseases, also observing that metabolically unhealthy obese patients had higher oxidative stress parameters compared to patients with normal weight and no metabolic disorders.[39]

Our results showed that individuals with healthy metabolic phenotypes and who consumed an anti-inflammatory diet had lower concentrations of isoprostanes, which leads us to suppose that the anti-inflammatory diet may possibly protect obese individuals from exacerbation of oxidative stress.[39] We did not find in the literature studies that made this assessment in individuals with different metabolic phenotypes of obesity, which limits us to make comparisons. However, we verified two studies that compared DII with oxidative stress. Zhang et. al observed a significant and positive correlation between the Children's Dietary Inflammatory Index (C-DII) with indicators of oxidative stress (serum bilirubin, albumin and iron) when evaluating children and adolescents, and this association was more pronounced in those who were overweight and obese.[40] Moradi et al., evaluated the relationship between DII and serum markers of oxidative stress in a case-control study with 121 patients with non-alcoholic fatty liver disease (NAFLD) and 119 healthy individuals and observed a correlation between DII with malondialdehyde (MDA) and total antioxidant capacity only in the group of healthy individuals.[41] Despite the few studies on the association of IDD with oxidative stress, it has been shown that healthy dietary patterns are composed of foods that are sources of

bioactive compounds, such as antioxidants, polyphenols, micronutrients, and polyunsaturated fatty acids; in this manner, they can attenuate inflammation, reduce oxidative stress, and reduce the risk of metabolic syndrome.[42]

This study has some limitations. As it is an observational study, causality cannot be inferred. Because the dietary characteristics of this sample were more pro-inflammatory, the number of individuals in the anti-inflammatory diet groups was much smaller, which may have influenced the results. That notwithstanding, this study has some strengths, including the facts that it is the only study in Brazil to evaluate obese individuals according to their phenotype and the first to evaluate dietary characteristics according to each metabolic phenotype, in addition to evaluation of oxidative stress.

Conclusion

We are able to conclude that an anti-inflammatory diet is associated with lower oxidative stress in metabolically healthy obese, and a pro-inflammatory diet is associated with higher systolic blood pressure value.

Author Contributions

Conception and design of the research, acquisition of data and writing of the manuscript: Pinto LR, Aranha LN, Oliveira GMM, Rosa G; analysis and interpretation of the data and statistical analysis: Pinto LR, Aranha LN, Luiz RR, Oliveira GMM, Rosa G; critical revision of the manuscript for intellectual content: Aranha LN, Oliveira GMM, Rosa G.

Potential Conflict of Interest

No potential conflict of interest relevant to this article was reported.

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Study Association

This article is part of the thesis of master submitted by Lara Ribeiro Pinto, from Universidade Federal do Rio de Janeiro.

Ethics Approval and Consent to Participate

This study was approved by the Ethics Committee of the Clementino Fraga Filho University Hospital under the protocol number 062/10. All the procedures in this study were in accordance with the 1975 Helsinki Declaration, updated in 2013. Informed consent was obtained from all participants included in the study.

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