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Red but not white meat consumption is associated with metabolic syndrome, insulin resistance and lipid peroxidation in Brazilian middle-aged men

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Abstract

Background: The influence of diet on metabolic syndrome and oxidative stress are not completely known.

Design: This cross-sectional study assessed the association of red meat and white meat consumption with metabolic syndrome, insulin resistance and lipid peroxidation in Brazilian middle-aged men.

Methods: A total of 296 subjects (age: 50.5 ± 5.0 years, body mass index: $25.8 \pm 3.5\,kg/m^2$) were evaluated. Anthropometry, lifestyle features, blood biochemical parameters, diagnosis of metabolic syndrome, homeostatic model assessment for insulin resistance, a lipid peroxidation marker (oxidized low-density lipoprotein) and triglycerides:high-density lipoprotein cholesterol ratio were assessed. Dietary intake was estimated by a food frequency questionnaire.

Results: The subjects included in the highest tertile red meat ($\geq$81.5 g/d) and saturated fatty acid from red meat consumption ($\geq$4.3 g/d) had higher occurrence of central obesity (nearly 60%, $p < 0.01$), hypertriglyceridaemia (nearly 43%, $p < 0.01$) and metabolic syndrome (35%, $p < 0.01$). They also had higher values of homeostatic model assessment for insulin resistance, oxidized low-density lipoprotein, and triglycerides:high-density lipoprotein cholesterol ratio, regardless of interfering factors. There were no associations of highest white meat tertile ($\geq$39.4 g/d) and saturated fatty acid from white meat ($\geq$1.0 g/d) consumption with the assessed parameters ($p > 0.05$).

Conclusions: Red meat consumption was cross-sectionally associated with the occurrence of central obesity, hypertriglyceridaemia, and metabolic syndrome as well as with higher homeostatic model assessment for insulin resistance, oxidized low-density lipoprotein concentrations and triglycerides:high-density lipoprotein cholesterol ratio. The content of saturated fatty acid from red meat consumption may be a factor that contributed to this relationship, while white meat consumption was not associated with metabolic syndrome and the assessed biomarkers.

Keywords

Meat, abdominal obesity, metabolic syndrome, oxidized LDL

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Introduction

Metabolic syndrome (MetS) is characterized by an aggregation of metabolic abnormalities such as central obesity, high blood pressure, high fasting blood glucose and dyslipidaemias, which are considered relevant risk factors for cardiovascular diseases.[1] Moreover, oxidative stress is a state where the production of free radicals and/or reactive species exceeds the antioxidant

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defence favouring the oxidation of biomolecules such as lipids, resulting in loss of its biological functions[2] and aggravation of cardiovascular diseases.[3]

Among the behavioural risk factors associated with MetS, oxidative stress and/or cardiovascular disease is an unhealthy dietary pattern.[4–6] In this context, two major prospective cohort studies (The Health Professionals Follow-up Study and The Nurses' Health Study) stated that high red meat (RM) consumption increased the risk for cardiovascular mortality and all-causes mortality.[7]

The last Household Budget Survey 2008–2009[8] held by the Brazilian Institute of Geography and Statistics showed a reduction in carbohydrate and an increase in fat and protein consumption, especially of animal source protein. Beef was considered one of the foods with the highest average consumption per capita[8] and this food group (meat) contributed mostly to saturated fatty acid (SFA) intake[9], which has been associated with increased adiposity, inflammation and insulin resistance (IR).[10]

In this sense, the high contribution of RM to daily energy consumption could be a potential harmful component of the Brazilian dietary pattern.[11] However, the number of studies concerning the relationships of white meat (WM) and/or RM consumption with the occurrence of MetS[11–14] and IR[15,16] is still modest. Moreover, the relationships of RM and WM consumption with lipid peroxidation, to our knowledge, have yet to be clarified.

Thus, this cross-sectional study assessed the potential associations between RM and WM consumption and MetS, IR and lipid peroxidation in Brazilian middle-aged men.

Methods

Study population

This cross-sectional study was carried out between March and December 2011. The sample size was calculated[17] considering the total number of male staff at the Federal University of Viçosa, Viçosa, Brazil in February 2011, aged between 40 and 59 years (1744 individuals), a confidence level of 95%, an expected MetS prevalence of 24.4% in Brazilian middle-aged men[18] and 4.5% sampling error, resulting in 293 participants required.

Participants were recruited by systematic sampling. We excluded those individuals who self-declared the following: body weight alterations greater than 3 kg in the 3 months preceding the study; altered levels of physical activity and eating habits in the 3 months preceding the study; thyroid diseases; heart failure; cerebrovascular diseases; infectious diseases; inflammatory diseases; diseases of the gastrointestinal tract; liver disease; chronic kidney disease and/or a history of kidney stones; cancer in the previous 10 years; eating disorders (anorexia and bulimia); food allergies. Individuals using vitamin supplements and those using diuretics or drugs that alter food intake and/or the metabolism of nutrients, pacemaker and/or prosthetic limbs users and elite athletes were also excluded.

We interviewed 848 men and 548 were eliminated by the exclusion criteria.[19] Of 300 selected, four did not answer the food frequency questionnaire (FFQ), so the final sample comprised 296 individuals.

The study was conducted according to the Declaration of Helsinki guidelines and all procedures involving human subjects were approved by the Ethics Committee in Human Research of the Federal University of Viçosa (Reference n°069/2010). Written informed consent was obtained from all subjects.

Dietary intake assessment

A FFQ, validated for the Brazilian population, was used to assess the usual dietary intake of the participants.[20] Daily food consumption was estimated as frequency × portion × size for each consumed food item. Nutrient intake was assessed using the software Dietpro® version 5.5i (AS Systems, Viçosa, Brazil), using mainly two Brazilian nutritional composition tables.[21,22] When the required nutritional information was not observed in these tables, the USDA table[23] was used.

The meat consumption assessed from data in the FFQ included 12 food-items: lean beef; high-fat beef; ground beef; lean pork; high-fat pork; bacon/pork rinds; poultry with skin; skinless poultry; fish; sausage; ham; hamburger. In the present study we considered for the RM group the consumption of lean beef, high-fat beef, ground beef, lean pork, high-fat pork and bacon/pork rinds. For the WM group the intake of poultry with skin, skinless poultry and fish was considered. The consumption of sausage, ham and hamburger was not considered in the statistical analysis due to the fact the FFQ did not discriminate the use of RM or WM in the production of these foods.

Blood pressure, anthropometric and body composition assessments

Systolic and diastolic blood pressures were measured following VI Brazilian Guidelines on Hypertension,[24] while anthropometric determinations such as weight, height and waist circumference were taken using standard measurement procedures, as previously described.[19] Body mass index was calculated as weight (kg) divided by height squared (m^2). Total body fat percentage was

determined by total body scanning with a dual energy X-ray absorptiometry (enCORE software version 13.31; GE/Lunar, Madison, WI, USA).

Lifestyle co-variables

The participants were asked about their current smoking status and alcohol consumption (yes/no). High alcohol consumption was also defined as a daily ingestion above 21 units per week.[25]

Habitual physical activity was estimated by the mean number of daily steps (7 consecutive days)[26,27] measured by a Digi-Walker SW-200 pedometer (Yamax Corporation, Tokyo, Japan), according to the instructions previously described.[19]

Sample collection and analysis

Blood samples were collected from the antecubital vein after 12-h overnight fasting. Serum concentrations of glucose, insulin, high-density lipoprotein (HDL-c) and triglycerides were measured by standard methods as previously described.[19] IR was estimated by the Index of Homeostasis Model Assessment of Insulin Resistance (HOMA-IR) using the Matthews et al. equation[28] and the atherogenic index was calculated by the ratio between triglycerides and HDL-c.[29] MetS was diagnosed by Alberti et al. criteria.[1]

Finally, plasma oxidized low-density lipoprotein (ox-LDL) concentrations were determined by a commercially available enzyme-linked immunosorbent assay kit from Mercodia (Uppsala, Sweden).

Statistical analysis

Data distribution was determined by the Shapiro–Wilk test. Non-normally distributed variables were log-transformed before statistical analysis. To evaluate the associations among consumption of meats and SFA with MetS occurrence, metabolic and lipid peroxidation markers, the participants were categorized into tertiles based on food-group consumption, which was adjusted by daily energy intake using the residual method. A comparison of nutrient consumption and lifestyle co-variables among tertiles of RM intake was performed by analysis of variance followed by Bonferroni's post-hoc test or by chi-square test for linear trend according to continuous and categorical variables, respectively.

The prevalence ratio was determined by Poisson regression with a confidence interval of 95% to assess the associations among MetS and tertiles RM, SFA from RM, WM and SFA from WM consumption. The chi-square test for linear trend was used to compare proportions among food-group consumption and MetS and its components.

Linear trends were assessed by assigning the average value to each tertile of RM, SFA from RM, WM and SFA from WM consumption, modelling those values as a continuous variable to assess its association with HOMA-IR, ox-LDL concentrations and triglycerides:HDL-c ratio. Multivariate regression models were controlled by confounding variables.

Calorie consumption outliers were defined by dispersing interquartile according to Vittinghoff et al.[30] Outliers were excluded (five individuals with caloric intake $\geq$2.640 kcal/d) followed by all statistical analyses previously described. After that the results maintained the same trend and statistical significance, where the results include all study participants. Data processing and analysis were performed using the software STATA version 9.1 (Stata Corp, College Station, TX, USA), considering p-values < 0.05 as statistically significant.

Results

Anthropometric and clinical characteristics of study participants are shown in Table 1. The occurrences of MetS and central obesity in the study sample were 24.7% and 47.3%, respectively.

Regarding dietary habits, protein, total fat, monounsaturated fatty acid, SFA and cholesterol intakes were higher in the third tertile of RM consumption compared with the second and first tertiles. Sausage, ham and hamburger consumption and iron intake were higher and fibre consumption was lower in subjects included in the third tertile compared to those in the first tertile of RM consumption (Table 2). Moreover, regarding the lifestyle co-variable, there were no statistical differences of current smoking status (number of smokers) and of habitual physical

Table 1. Anthropometric and clinical characteristics of participants ($n = 296$)

Variables	Values
Age (years)	50.5 ± 5.0
Body mass index (kg/m^2)	25.8 ± 3.5
Total body fat (%)	22.7 ± 7.2
HOMA-IR	1.4 ± 1.1
HDL-c (mg/dL)	46.9 ± 12.7
Triglycerides (mg/dL)	142.4 ± 95.1
ox-LDL (U/L)	55.6 ± 16.8
Central obesity *n* (%)	140 (47.3)
Metabolic syndrome *n* (%)	73 (24.7)

HOMA-IR, homeostasis model assessment of insulin resistance; HDL-c, high density lipoprotein; ox-LDL, oxidized low density lipoprotein; Values are mean $\pm$ SD or *n* (%).

Table 2. Food, nutrient consumption and lifestyle characteristics according to tertiles (T) of energy-adjusted red meat intake

	T1 <56.0 g/d ($n = 98$)	T2 56.0 – 81.5 g/d ($n = 98$)	T3 ≥81.5 g/d ($n = 100$)	*p*-value
White meat (g/d)	36.9 ± 28.5	37.2 ± 23.0	37.7 ± 26.2	0.564
Sausage, hamburger and ham (g/d)	11.6 ± 10.7[a]	14.9 ± 14.9	17.9 ± 14.1	0.005*
Energy (kcal/d)	1463.2 ± 443.5	1429.4 ± 475.7	1475.7 ± 555.4	0.749
Protein (g/d)	59.9 ± 10.2[b]	67.4 ± 8.0[c]	79.5 ± 13.2	<0.001*
Carbohydrate (g/d)	208.7 ± 28.2[b]	199.5 ± 26.6[c]	175.6 ± 31.8	<0.001*
Fat (g/d)	42.4 ± 11.7[a]	43.2 ± 10.6[c]	48.4 ± 10.4	<0.001*
SFA (g/d)	14.3 ± 5.2[a]	14.7 ± 4.0[c]	16.9 ± 4.6	<0.001*
PUFA (g/d)	6.9 ± 2.5	6.9 ± 2.3	7.5 ± 2.6	0.268
MUFA (g/d)	12.7 ± 3.6[b]	14.1 ± 3.6[c]	17.1 ± 4.2	<0.001*
Cholesterol (g/d)	189.5 ± 89.9[b]	211.9 ± 85.3[c]	261.1 ± 88.4	<0.001*
Fibre (g/d)	23.6 ± 6.3[a]	21.8 ± 5.3	20.4 ± 6.3	<0.001*
Sodium (mg/d)	1,320.7 ± 559.7	1,449.5 ± 634.1	1,301.4 ± 527.2	0.090
Iron (mg/d)	7.0 ± 1.7[a]	7.1 ± 1.2	7.3 ± 1.2	0.030*
Alcohol (g/d)	7.3 ± 13.8[b]	16.0 ± 18.6	16.9 ± 19.4	<0.001*
Habitual physical activity (steps numbers/d)	11,586 ± 4,052	11,138 ± 3,388	10,587 ± 4,168	0.200
Current smoker *n* (%)	10.0 (25.0)	15.0 (37.5)	15.0 (37.5)	0.326

SFA, saturated fatty acid; PUFA, polyunsaturated fatty acid; MUFA, monounsaturated fatty acid; Values are mean ± SD or *n* (%); [a]Significantly different from T3 (*p-value < 0.05, post hoc Bonferroni's test); [b]Significantly different from T2 and T3 (*p-value < 0.05, post hoc Bonferroni's test); [c]Significantly different from T3 (*p-value < 0.05, post hoc Bonferroni's test).

activity (steps number/d) according to tertile RM intake (Table 2).

Interestingly, there was higher MetS occurrence in those subjects included in the highest tertiles of RM and of SFA from RM consumption compared to those in the first tertile, regardless of interfering factors (Table 3). In addition, central obesity occurrence also was higher in the third tertile compared with the first tertiles of RM and SFA from RM consumption (first tertile: 39.8%, 40.8%; second tertile: 41.8%, 39.8%; third tertile: 60.0%, 61.0% respectively, $p < 0.01$). Similar findings were seen with the hypertriglyceridaemia (first tertile: 23.5%, 22.4%; second tertile: 23.5%, 26.5%; third tertile: 43.0%, 44.0% respectively, $p < 0.01$). There were no significant associations of MetS and its components with WM and SFA from WM consumption ($p > 0.05$).

Moreover, HOMA-IR, ox-LDL concentrations and the triglycerides:HDL-c ratio were positively associated with RM and with SFA from RM consumption, regardless of interfering factors. However, HOMA-IR, ox-LDL levels and the triglycerides:HDL-c ratio were not associated with WM and SFA from WM consumption (Table 4).

Given the role of central fat accumulation on cardiometabolic risk, we replaced the total body fat by central obesity indicator (waist circumference ≥90 cm), as an adjustment variable, and thus the statistical significances for the associations of RM consumption with HOMA-IR (p for trend = 0.448) and triglycerides:HDL-c ratio (p for trend = 0.208), as well as of SFA from RM consumption with HOMA-IR (p for trend = 0.169), ox-LDL concentrations (p for trend = 0.092) and triglycerides:HDL-c ratio (p for trend = 0.142) disappeared.

Discussion

The first important finding of the current study was the positive association of RM and SFA from RM consumption with the occurrence of MetS, central obesity and hypertriglyceridaemia. The relationship between MetS and RM consumption was also reported in a cohort study involving migrants and Japanese descendants living in Brazil, where the highest tertile of RM consumption (mean = 144.2 g/d) was associated with a 4.7 times higher risk of MetS, regardless of interfering factors.[11] In turn, the *Prevención Dieta Mediterránea* trial study found that RM consumption was positively associated with a risk of central obesity and MetS incidence, having a tendency to influence RM consumption in hypertriglyceridaemia.[12]

Regarding the positive associations of the occurrence of MetS, central obesity, and hypertriglyceridaemia with SFA from RM consumption, it is important to highlight that the consumption of a diet with high

Table 3. Prevalence ratio (PR) and 95% confidence intervals (95% CI) of metabolic syndrome (MetS) according to tertiles (T) of energy-adjusted red meat (RM), white meat (WM) and saturated fatty acid (SFA) from RM and WM intake

	MetS *n* (%)a	Model 1 PR (95% CI)b	Model 2 PR (95% CI)b	Model 3 PR (95% CI)b
RM (g/d)				
T1: <56.0	17 (17.3)	1.00	1.00	1.00
T2: 56.0–81.5	21 (21.4)	1.23 (0.65–2.34)	1.25 (0.66–2.38)	1.15 (1.06–3.44)
T3: ≥81.5	35 (35.0)	2.01 (1.13–3.60)*	2.00 (1.12–3.57)*	1.90 (1.06–3.44)*
p-value	0.004*	0.013*	0.015*	0.023*
SFA from RM (g/d)				
T1: <2.7	19 (19.4)	1.00	1.00	1.00
T2: 2.7–4.3	19 (19.4)	1.00 (0.66–2.38)	1.02 (0.54–1.93)	0.95 (0.50–1.83)
T3: ≥4.3	35.0 (35.0)	1.80 (1.03–3.15)*	1.82 (1.04–3.18)*	1.79 (1.01–3.15)*
p-value	0.011*	0.028*	0.026*	0.033*
WM (g/d)				
T1: <24.0	23 (23.5)	1.00	1.00	1.00
T2: 24.0–39.4	23 (23.5)	1.00 (0.56–1.78)	0.94 (0.52–1.68)	0.95 (0.53–1.71)
T3: 39.4	27.0 (27)	1.15 (0.66–2.00)	1.14 (0.65–1.99)	1.12 (0.64–1.97)
p-value	0.564	0.616	0.625	0.682
SFA from WM (g/d)				
T1: <0.6	23 (23.5)	1.00	1.00	1.00
T2: 0.6–1.0	22 (22.4)	0.95 (0.53–1.71)	0.91 (0.50–1.64)	0.86 (0.47–1.55)
T3: 1.0	28.0 (28)	1.19 (0.68–2.07)	1.14 (0.65–1.99)	1.10 (0.63–1.93)
p-value	0.460	0.519	0.609	0.712

Values are *n* (%) to MetS occurrence and prevalence ratio (95% CI) to Models 1, 2 and 3. Model 1, unadjusted model; Model 2, adjusted for age; Model 3, adjusted for age (years), habitual physical activity (steps number/d), smoking habit (yes/no), excessive alcohol intake (yes/no) and daily caloric intake (kcal); [a]*p*-value from chi-square for linear trend; [b]*p*-value from Poisson regression; *Significant prevalence ratio and *p*-value < 0.05.

Table 4. Homeostasis model assessment of insulin resistance (HOMA-IR), oxidized LDL (ox-LDL) and triglycerides:HDL-c ratio according to tertiles (T) of energy-adjusted red meat (RM), white meat (WM) and saturated fatty acid (SFA) from RM and WM intake

		HOMA-IR	ox-LDL (U/L)	Triglycerides: HDL-c ratio
RM (g/d)	T1: <56.0	1.3 ± 0.9	52.4 ± 14.7	3.1 ± 2.5
	T2: 56.0–81.5	1.4 ± 1.1	53.8 ± 16.3	3.0 ± 2.2
	T3: ≥81.5	1.6 ± 1.3	60.7 ± 18.2	4.1 ± 3.7
	p for trend[a]	0.039*	0.009*	0.029*
SFA from RM (g/d)	T1: <2.7	1.3 ± 0.8	52.5 ± 15.2	3.0 ± 2.2
	T2: 2.7–4.3	1.3 ± 1.0	54.7 ± 16.3	3.0 ± 2.3
	T3: ≥4.3	1.7 ± 1.3	59.7 ± 18.1	4.2 ± 3.8
	p for trend[a]	0.019*	0.049*	0.032*
WM (g/d)	T1: <24.0	1.4 ± 1.1	56.7 ± 18.1	3.6 ± 3.0
	T2: 24.0–39.4	1.4 ± 1.0	56.7 ± 16.6	3.5 ± 3.4
	T3: 39.4	1.5 ± 1.2	53.6 ± 15.6	3.1 ± 2.2
	p for trend[a]	0.580	0.212	0.154
SFA from WM (g/d)	T1: <0.6	1.3 ± 1.0	56.2 ± 17.8	3.7 ± 3.4
	T2: 0.6–1.0	1.4 ± 1.1	55.8 ± 16.4	3.3 ± 2.6
	T3: 1.0	1.5 ± 1.2	55.0 ± 16.4	3.3 ± 2.6
	p for trend[a]	0.778	0.355	0.168

HDL-c, high density lipoprotein; Values are mean ± SD; [a]*p* for trend from the linear regression model, adjusted for age (years), total body fat (%), habitual physical activity (steps number/d), smoking habit (yes/no), fibre consumption (g/d), sausage, ham and hamburger consumption (g/d) and daily caloric intake (kcal/d); *Significant *p*-value < 0.05.

amounts of this type of fatty acid favour the occurrence of hypertriglyceridaemia by stimulating hepatic secretion of lipoproteins containing apoB-100[31] as well as a possible increase in weight gain by its lower thermogenic effect compared with unsaturated fat from vegetable sources.[32] In fact, RM is rich in SFA[33] and, accordingly, we verified a linear increase of SFA intake with an increment of RM consumption. Babio et al.[12] also verified this direct association between SFA and RM consumption, suggesting the relationship of this nutrient with central obesity. Although a recent study has proposed another RM compound (L-carnitine) as a risk factor for metabolic complications,[34] our results indicate the important role of SFA from increased RM consumption on the occurrence of metabolic disorders.

We also showed that RM and SFA from RM consumption were positive predictors of HOMA-IR. In a recent meta-analysis including nine prospective cohort studies, six of them verified a significant positive relationship between unprocessed RM consumption and incidence of type-2 diabetes mellitus (DM2).[35] In fact, RM provides significant amounts of SFA, particularly palmitic acid.[33] In excess, palmitate may inhibit the activation of insulin receptor substrate-1, fosfatidilinisitol-3-kinase or protein kinase B in adipocytes causing impaired IR, as well as reducing muscle cell insulin sensitivity by stimulating the secretion of inflammatory cytokines.[10]

Furthermore, RM is a relevant source of iron.[36] If 100 g of RM has roughly 1.1 mg of haeme iron,[37] our participants probably consumed 1.26 mg/d of haeme iron from RM (last tertile). In this context, prospective studies have shown that haeme iron consumption from RM, but not from poultry/fish, is positively associated with a higher incidence of MetS[38] and DM2.[39] In addition, beta cells are susceptible to oxidative stress induced by iron and the deposition of this nutrient in these cells may lead to apoptosis and insulin deficiency.[40]

Interestingly, we found a positive association of RM and SFA from RM consumption with ox-LDL concentrations. It is possible that an excess of palmitate expands white adipose tissue, thus increasing the reactive oxygen species and the expression of inflammatory genes in human macrophages by a nuclear factor kappa β-dependent mechanism.[10] Inflammatory cytokine production may further stimulate the production of free radicals, causing the occurrence of oxidative stress.[41] Iron ions also induce the oxidation of polyunsaturated fatty acid in macrophages and this lipid peroxidation may lead to LDL oxidation.[42] In addition, since that increased formation of ox-LDL has been associated with endothelial dysfunction related to atheromatous plaque and atherosclerosis,[41] our findings suggest that RM intake could also be a possible risk factor for atherosclerosis. Reinforcing this hypothesis, we also observed positive associations between RM consumption and the triglycerides:HDL-c ratio, another potential indicator of atherogenesis.[29]

Moreover, no relationships of WM consumption with MetS occurrence and metabolic/oxidative stress markers were noted in this study. Our findings are in accordance with those from previous studies, in which no associations of poultry consumption with DM2[43] and MetS[11] were verified. All findings together reinforce the idea that, although a protective effect of WM consumption on metabolic and oxidative stress markers has not been proven, moderate consumption of WM could be an interesting dietary strategy to be adopted over the life course.

Since central obesity has an extremely important role in metabolic disorders[44,45] and oxidative stress,[46] it could affect the relationship between meat consumption and the variables studied here. The loss of statistical significance in the associations tested, by the inclusion of central obesity as an adjustment variable, corroborates this hypothesis.

The main limitation of the present study is its cross-sectional nature. Thus, the results shown here must be cautiously considered as we cannot guarantee that the observed associations show a cause/effect relationship, although we controlled potential interfering variables. Future studies involving the participation of women should be conducted before considering the application of these results at the population level.

In conclusion, RM consumption was cross-sectionally associated with the occurrence of MetS and its components, central obesity and hypertriglyceridaemia as well as with higher values of HOMA-IR, triglyceride:HDL-c ratio, and ox-LDL in Brazilian middle-aged men. Its SFA content appears to contribute to the harmful effects. Additionally, WM consumption was not associated with MetS and with the assessed biomarkers, indicating that WM consumption is a more secure dietary strategy to be recommended in terms of healthy eating habits.

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Conflict of interest

The authors declare that they have no conflicts of interest.

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