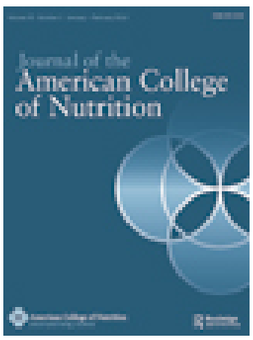

Journal of the American College of Nutrition

ISSN: 0731-5724 (Print) 1541-1087 (Online) Journal homepage: http://www.tandfonline.com/loi/uacn20

Postprandial Lipid Response to High-Saturated and High-Monounsaturated Fat Meals in Normal-Weight or Overweight Women

Lílian L. Lopes, Daniela Mayumi U. P. Rocha, Alessandra da Silva, Maria do Carmo G. Peluzio, Josefina Bressan & Helen Hermana M. Hermsdorff

To cite this article: Lílian L. Lopes, Daniela Mayumi U. P. Rocha, Alessandra da Silva, Maria do Carmo G. Peluzio, Josefina Bressan & Helen Hermana M. Hermsdorff (2018): Postprandial Lipid Response to High-Saturated and High-Monounsaturated Fat Meals in Normal-Weight or Overweight Women, Journal of the American College of Nutrition, DOI: 10.1080/07315724.2017.1399835

To link to this article: https://doi.org/10.1080/07315724.2017.1399835

Published online: 09 Feb 2018.

JOURNAL OF THE AMERICAN COLLEGE OF NUTRITION
https://doi.org/10.1080/07315724.2017.1399835

Taylor & Francis
Taylor & Francis Group

Postprandial Lipid Response to High-Saturated and High-Monounsaturated Fat Meals in Normal-Weight or Overweight Women

Lílian L. Lopes, MSc, Daniela Mayumi U. P. Rocha, MSc, Alessandra da Silva, RD, Maria do Carmo G. Peluzio, PhD, Josefina Bressan, PhD, and Helen Hermana M. Hermsdorff, PhD

Department of Nutrition and Health, Universidade Federal de Viçosa, Viçosa, Minas Gerais, Brazil

ABSTRACT

Purpose: We evaluated postprandial response of the lipid metabolism markers after the intake of a high-saturated fat (HSM) or high-monounsaturated fat meal (HMM).

Methods: A randomized, controlled and acute intervention study included 63 women (age 26.9 ± 6.1 years): 35 normal weight (NW) and 28 overweight (OW) (total body fat [TBF] 24.7 ± 3.9% and 36.6 ± 3.9%, respectively). After 12 hours of fasting, each subject was given one of the two test meals standardized, including 2 muffins and water (HSM, 42.1% of saturated fat acid, or HMM, 34.5% of monounsaturated fat acid). Plasma fatty acid profile and concentrations of apolipoproteins A1 and B100, complement C3, and triacylglycerols were analyzed during fasting and at 2, 3, and 5 postprandial hours.

Results: Among the markers studied, the triacylglycerol (TAG) and complement C3 were significantly higher in the OW group, compared to NW. The increment in the C3 concentration was higher after HSM intake, compared with HMM (iAUC = 4365.5 ± 5477.4 vs. 1215.2 ± 882.4; $p = 0.006$), with no differences between groups. After 5 hours postprandial, plasma oleic acid values remained high compared with the fasting value in the NW group, but not in the OW group (26.0 ± 4.2 vs 23.7 ± 3.9%; $p < 0.001$). Women with high percentage of total plasma saturated fatty acids (SFA) at the beginning of the intervention had higher incremental area under the curve (iAUC) for the palmitic, stearic, and total fatty acids ($p < 0.005$). Those women with a high percentage of monounsaturated fatty acids (MUFA) showed lower iAUC values for the same fatty acid profile ($p < 0.005$).

Conclusion: This study demonstrated the effect of the HSM on postprandial increment of C3 concentration, suggesting another mechanism for saturated fat metabolism. The postprandial response to HSM appears to be the mediated by baseline lipid profile of the individuals, while the response to HMM was correlated to the weight status.

Abreviations: APO, Apolipoprotein; CVD, Cardiovascular disease; HDL-C, High density lipoprotein; HMM, High-monounsaturated fatty acid meal; HSM, High-saturated fatty acid meal; iAUC, Incremental area under the curve; LDL-C, Low density lipoprotein; MUFA, Monounsaturated fatty acids; NW, Normal weight; OW, Overweight; TAG, Triacylglycerol; TBF, Total body fat; VLDL-C, Very low density lipoprotein; UFV, Universidade Federal de Viçosa

ARTICLE HISTORY
Received 29 March 2017
Accepted 29 October 2017

KEYWORDS
Lipid metabolism; complement C3; postprandial period; obesity; monounsaturated fatty acids and saturated fatty acids

Introduction

Healthy food habits can reduce the risk of chronic diseases via modulation of the lipid metabolism, the inflammatory state, and the oxidative stress (1,2), all related to the risk for cardiovascular disease (CVD). In light of this, the fatty acid profile of diet has been discussed in detail due to the potential role of the lipid metabolism modulation and the occurrence of related chronic diseases (3–5). Saturated fatty acids (SFA) consumed in excess are associated with the development of CVD and high lipoprotein concentrations like low-density lipoprotein (LDL-C), although it is essential for lipid metabolism (6–9). Monounsaturated fatty acids (MUFA), however, reduce LDL-C concentrations, without decreasing high-density lipoprotein (HDL-C) concentration and causing lipid oxidation (9,10).

Postprandial studies investigate the physiological effects caused by the intake of specific nutrients, postprandial changes in glucose and lipid profiles, energy metabolism, and hormonal and inflammatory markers (11,12). Postprandial lipemia is linked to a series of events related to the increase in the triacylglycerol-rich lipoproteins (TRL), chylomicrons and their remnants, and very-low-density lipoprotein (VLDL-C) (13), as well as to inflammation and oxidative stress markers (14), after a high-fat meal, which are risk factors for CVD (13). However, the effects of postprandial lipemia are still controversial, warranting further studies to assess lipid metabolism markers (15–19). In addition, obese subjects had higher concentrations of lipid and inflammation biomarkers (6,20–22) and their postprandial response appears to be different according to weight status,

CONTACT Helen H. M. Hermsdorff helenhermana@ufv.br Department of Nutrition and Health, Universidade Federal de Viçosa, Av. P.H. Rolfs s/n, Campus Universitário, 36570-900, Viçosa, Minas Gerais Brazil.

Color versions of one or more of the figures in the article can be found online at www.tandfonline.com/uacn.

although the response to different fatty acids in obese individuals is not fully understood (19,23).

Therefore, we evaluated the acute effect (postprandial) of a high-SFA (HSM) and high-MUFA (HMM) meal on the markers of lipid metabolism in apparently healthy normal-weight and overweight women.

Methodology

Subject

The volunteers were recruited via local media and the Universidade Federal de Viçosa (UFV) webpage. The inclusion criteria for the selection of the participants in the study were as follows: women between 20 and 40 years; not pregnant, breastfeeding, or menopausal; nonalcoholic and nonsmokers; and having no inflammatory, hormonal, heart, respiratory, kidney. or liver disease, or gastrointestinal disease (acute or chronic) that may alter digestion and nutrient absorption. The health status was self-reported. In addition, they could not using any medication that affects metabolism or body composition; must have a total cholesterol value <240 mg/dl, triacylglycerol (TAG) <150 mg/dl, and fasting glucose <100 mg/dl (9); no nutritional treatment for weight loss and have maintained a stable weight for 3 months prior to the study; and not an athlete (according to the International Physical Activity Questionnaire, short version [IPAQ]). On the test day, the volunteers could not be in the menstrual period (7 days before or 7 days after the end of the menstruation).

All the study procedures were registered in the Brazilian Clinical Trials Registry (ReBEC-Id: RBR-2h3wjn and RBR-66jx7j) and approved by the Ethics Committee on Human Research of the Universidade Federal de Viçosa (UFV) (Of. Ref. No. 184/2011 and CAAE 542,585/2014) in line with the Resolution CNS 466/2012, concerning research involving humans. The volunteers signed free and informed consent in accordance with the principles of the Helsinki Declaration.

Study design

This study was a randomized and controlled acute intervention, conducted at the Laboratory of Energy Metabolism and Body Composition, in the Department of Nutrition and Health, UFV.

The selected volunteers received a specific food plan to follow in the 2 days before the test day in order to standardize their dietary and lipid intake. The food plan was adapted according to individual daily energy requirements (24). In addition, the volunteers completed a 24-hour food recall in those 2 days to control the lipid intake.

On the test day, each subject was given previously randomly (through the website www.random.org) one of the two test meals standardized containing HSM or HMM, including 2 muffins (180 g) and 500 mL of water. Each muffin (90 g) for HMM was made with 72 g of olive oil, 5 g of milk powder, 50 g of water, 30 g of cashews, 20 g of wheat flour, 20 g of cornstarch, 10 g of egg, 1 g of salt, and 0.6 g of baking powder. Each muffin (90 g) for HSM was made with 72 g of bacon, 45 g of wheat flour, 45 g of milk, 36 g of mozzarella cheese, 9 g of natural yogurt, 7 g of butter, 4 g of sugar, 0.5 g of salt chemical, and 0.6 g of baking powder. The ingredients were mixed and baked at 200°C for 30 minutes. The macronutrient composition of the muffins is described in Table 1.

Table 1. Macronutrient composition of meals tests offered to study participants

Composition	Meals tests	
	HSM	HMM
Energy (kcal)	1008.0	1008.0
Carbohydrate (g) (% EI)	36.7 (14.6)	44.2 (17.5)
Proteins (g) (% EI)	15.6 (6.2)	10.7 (4.3)
Total fat (g) (% EI)	88.7 (79.2)	87.6 (78.2)
Saturated fatty acids (g) (% total fat)	42.1 (47.5)	13.5 (15.4)
Monounsaturated fatty acids (g) (% total fat)	34.5 (38.9)	63.1 (72.0)
Polyunsaturated fatty acids (g) (% total fat)	6.1 (6.9)	9.5 (10.8)

Note: EI = energy intake, HSM = high-saturated fatty acid meal, HMM = high-monounsaturated fatty acid meal.

The anthropometric measures were assessed in 12-hour fasting. The stature (m) was measured using a stadiometer (Seca 206 model, Hamburg, Germany). Body weight (kg) and body fat (%) were obtained by tetrapolar bioimpedance (InBody Composition Analyzer, model Y230), in accordance with the manufacturer's protocol. The body mass index (BMI) was calculated as the ratio between body weight (kilograms) and squared height (meters). The waist circumference was measured at the midpoint between the last rib and the iliac crest, using a flexible and inelastic measuring tape, subdivided in millimeters.

Intravenous blood samples were drawn from the median antecubital vein using the vacuum system VACUETTE®, K3EDTA. Four samples were taken at 0 (after fasting for 12 hours), 2, 3, and 5 hours, respectively, after intake of each test meal. The samples (plasma) were clearly identified and stored in a freezer at −80°C until analysis.

Determination of the lipid metabolism markers

In order to determine the plasma fatty acid profile the total lipids were extracted according to the Folch method (1957), saponified and esterified using the method of Hartmann and Lago (1973) (25,26).The fatty acid profile was analyzed via the gas chromatography model CG Solution (Shimadzu®), equipped with flame ionization detection (FID). The compounds were then identified and separated on a capillary column (30m × 0.25mm). For chromatographic separation, 1 μl sample was injected using a 10-μl syringe (Hamilton®) in a Split system. Nitrogen gas was used as a carrier at a linear speed of 43.2cm/s, as well as the hydrogen gas and synthetic air formed in the flame detector.

Temperatures of the injector and detector were controlled isothermally at 200°C and 220°C. The initial column temperature was 100°C (maintained for 5 minutes), and increased at 4°C per minute until 220°C (maintained for 20 minutes). The carrier gas flow in the column was 1.0 mL/minute. Identification and quantification of the fatty acids present in the samples was done by comparing the retention times of the samples with the standard mixture of the fatty acids (FAME mix, Supelco TM from C 4:0 to C 24:0, Sigma-Aldrich®, EUA), and the results were expressed as percentages.

Serum TAG (mg/dl) and complement C3 were analyzed employing the colorimetric and turbidimetric assay, respectively, in an automatic analyzer using specific assay kits (Labmax Pleno, Labtest, Brazil). Serum Apolipoprotein B100 was analyzed with the immunoturbidimetric test by the automatic analyzer Advia 1800 (Siemens®) and commercial kits from the same manufacturer. Serum apolipoproteins A1 and B100 were analyzed by the nephelometric test utilizing an automatic analyzer Immage (Beckamnn Coulter®) and commercial kits from the same equipment manufacturer.

Statistical analysis

Data were expressed as the mean ± SD and or median (interquartile range) according to the variable normality, which was assessed by the Shapiro–Wilk test.

The Mann–Whitney *U*-test or Student's *t*-test was used to assess the presence of any differences between the test meals (HSM × HMM) and between the groups (normal weight [NW] × overweight [OW]) for studied variables. A two-way analysis of variance (ANOVA) was used to compare the postprandial response of the lipid metabolism markers in relation to the test meal (HSM × HMM) and the group (NW × OW), at each time, and the possible interaction between the factors. The nonparametric variables were earlier transformed into log 10 before analysis. To identify the correlations between the lipid metabolism markers we used the Pearson correlation test (parametric variables) or the Spearman (nonparametric) test.

To investigate whether the postprandial response would differ according to the basal metabolic profile of the participants, the subjects were divided into two groups based on the median (low or high value) to the basal values of the total saturated and monounsaturated fatty acids, as well as the basal concentrations of the apolipoproteins (Apo) A1 and B100. The application of median as the cutoff point has been performed (20,27), according to the methodology of the identification of the two risk groups in the epidemiological studies (28).

Statistical analyses were performed using SPSS Statistics 22.0 software (IBM Corp., 2013). The incremental area under the curve (iAUC) was calculated using the trapeizodal method, with the program Prism, version 5 (GraphPad Software, 2011). The statistical significance level was considered as less than 5% probability.

Results

The study included 63 adult women (22 to 37 years of age), of whom 35 were normal weight (NW) (<30% of total body fat [TBF]) and 28 overweight (OW) (≥30% of TBF) (29). The baseline characteristics of participants in the study, by weight status, are shown in Table 2. Among the markers studied, TAG and complement C3 were significantly higher in the OW group, compared to the NW group.

As expected, the iAUC at 5hours postprandial for stearic acid and total SFA was higher after HSM intake, while the iAUC at 5hours postprandial for oleic acid was higher after HMM intake. This was an expected result; however, there was no difference in accordance with weight status. The iAUC for the complement C3 was significantly higher after HSM intake compared with HMM intake, with no difference between the groups (NW and OW) (Figure 1).

In addition, the change in the postprandial oleic acid was significant over time and there was a difference between groups (NW and OW) (Figures 2a and 2b) and for diet (HSM) (Figures 2c and 2d). Interestingly, the increase in oleic acid values remained until the fifth postprandial hour in the NW group (Figure 2a), but not in the OW group ($p < 0.001$) (Figure 2b). On analyzing the response of oleic acid after the meals over time, iAUC (Figure 2e) at 2 and 5 hours (Figure 2g) presented a significant difference in relation to the diet ($p = 0.005$ and $p = 0.003$, respectively), but there were no differences between the groups (NW front of the OW) and there was no interaction between diet and group (Figures 2e and 2f).

Furthermore, the postprandial rise of stearic acid and total SFA were positively correlated with the increase in the complement C3 among the NW women, while the total SFA and C3 correlated positively in the OW group (Figure 3).

Finally, when we categorized the volunteers according to the median of baseline plasma SFA, the 5-hour iAUC values for total SFA, palmitic acid, and stearic acid after HSM consumption were significantly greater in those participants with high

Table 2. Baseline characteristics of the volunteers, according to weight status[a]

	Normal weight (n = 35)	Overweight (n = 28)	*p* Value*
Characteristics			
Age (years)	25 (22–27)	29 (23–37)	0.014
BMI (kg/m^2)	21.4 (19.3–22.6)	28.2 (25.4–31.9)	< 0.001
Waist circumference (cm)	69.0 (66.5–74.0)	84.0 (73.9–98.5)	< 0.001
Plasma lipids			
Palmitic acid (16:0) (%)	25.2 (22.5–28.8)	25.2 (22.7–27.8)	0.926
Stearic acid (18:0) (%)	7.1 (5.9–10.0)	6.9 (5.9–10.5)	0.959
Total SFA (%)	58.4 (30.4–71.5)	33.4 (31.0–68.2)	0.611
Oleic acid (18:1) (%)	18.9 ± 4.1	21.2 ± 5.4	0.079
Total MUFA (%)	24.7 (21.7–27.8)	27.4 (23.1–30.2)	0.115
Apolipoprotein A1 (mg/dL)	131.2 ± 24.2	131.6 ± 23.7	0.953
Apolipoprotein B 100 (mg/dL)	78.7 ± 21.6	88.9 ± 18.2	0.066
Triacylglycerol (mg/dL)	92.2 ± 33.1	115.6 ± 53.5	0.042
`Complement C3 (mg/dL)	118.0 (107.1–137.5)	128.0 (118.9–165.7)	0.020

[a]Rating: Normal weight, <30.0% total body fat; overweight, ≥ 30.0% total body fat; BMI body mass index, SFA saturated fatty acids, MUFA monounsaturated fatty acids.
*These are *p* values by Student's *t*-test or Mann–Whitney test according to variable normality. Data expressed as mean ± standard deviation or median (CI 25–75th percentile), respectively.

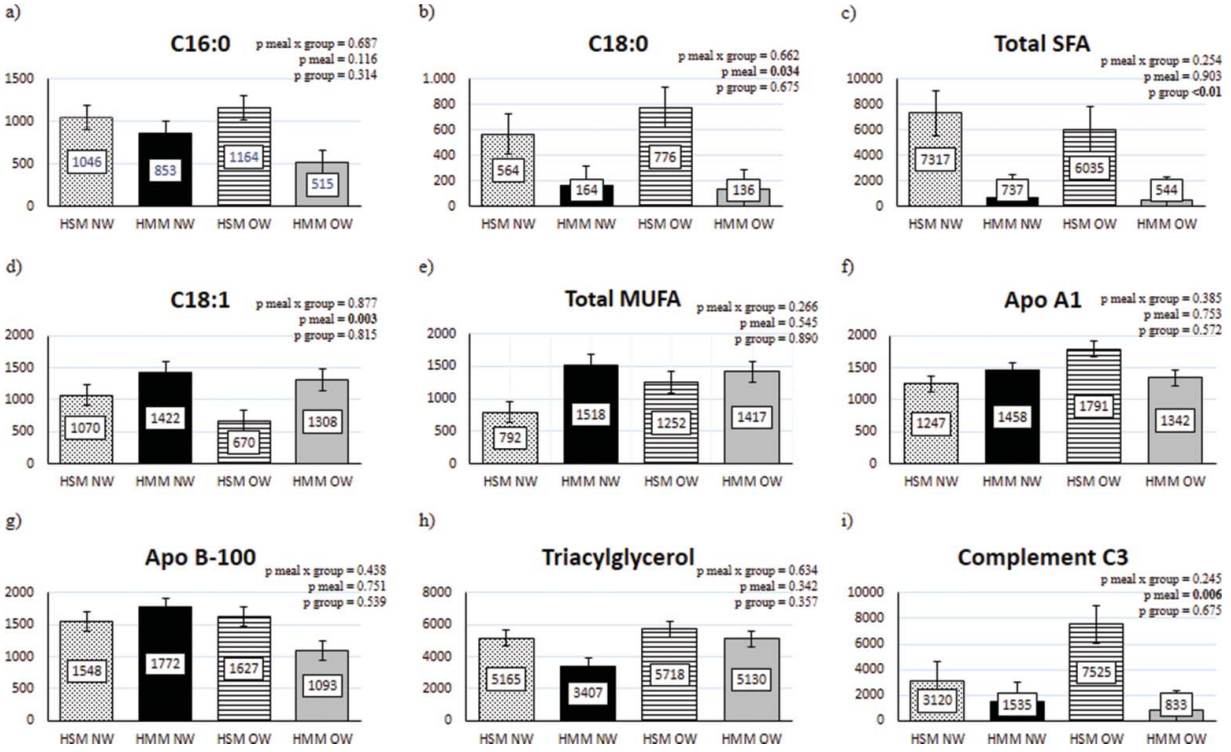

Figure 1. Incremental area under curve relating to lipid metabolism markers according to the test meals to NW and OW groups. Apo A1 apolipoprotein A1, Apo B-100 apolipoprotein B-100, HSM high-saturated fat meal, HMM high-monounsaturated fat meal, iAUC incremental area under the curve, MUFA monounsaturated fatty acids, NW normal weight (<30.0% total body fat, n = 35), OW overweight (≥30.0% total body fat, n = 28), SFA saturated fatty acids. *These are *p* values from two-way ANOVA. (a) C16:0, palmitic acid; (b) C18:0, stearic acid; (c) total saturated fatty acids; (d) C18:1, oleic acid; (e) total monounsaturated fatty acids; (f) apolipoprotein A1; (g) apolipoprotein B100; (h) triacylglycerol; (i) complement C3.

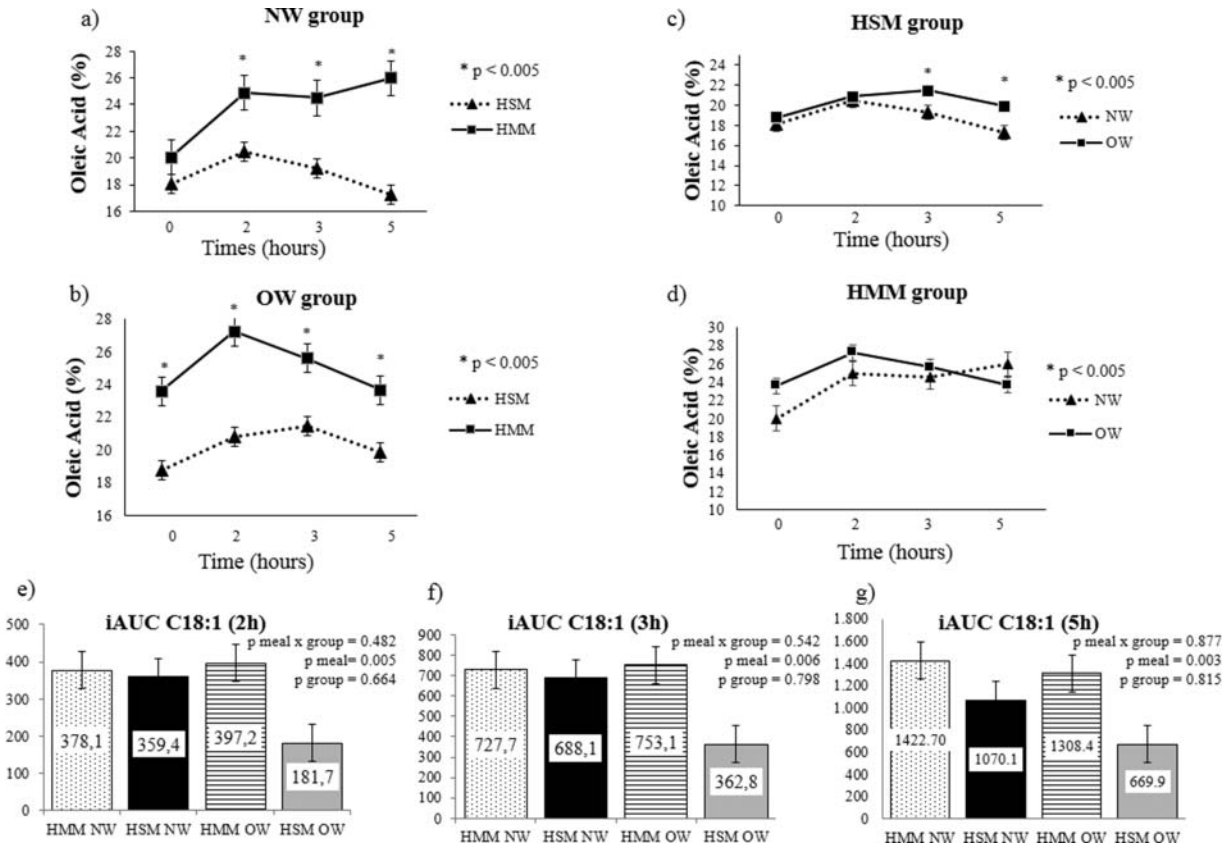

Figure 2. Postprandial responses of oleic acid (18:1), according to the weight status and test meal. Data represented as mean ± standard error. HSM high-saturated fat meal, HMM high-monounsaturated fat meal, iAUC incremental area under the curve, NW normal weight (<30.0% total body fat, n = 35), OW overweight (≥30.0% total body fat, n = 28). *These are *p* values from Student's *t*-test for each time (a, b, c, d) and two-way ANOVA (e, f, g). Postprandial response of oleic acid after meals, separated by NW (a) and OW (b) groups, separated by HSM (c) and HMM (d) meals over time. Response of oleic acid (iAUC) after meals over time: 2 (e), 3 (f), and 5 (g) postprandial hours.

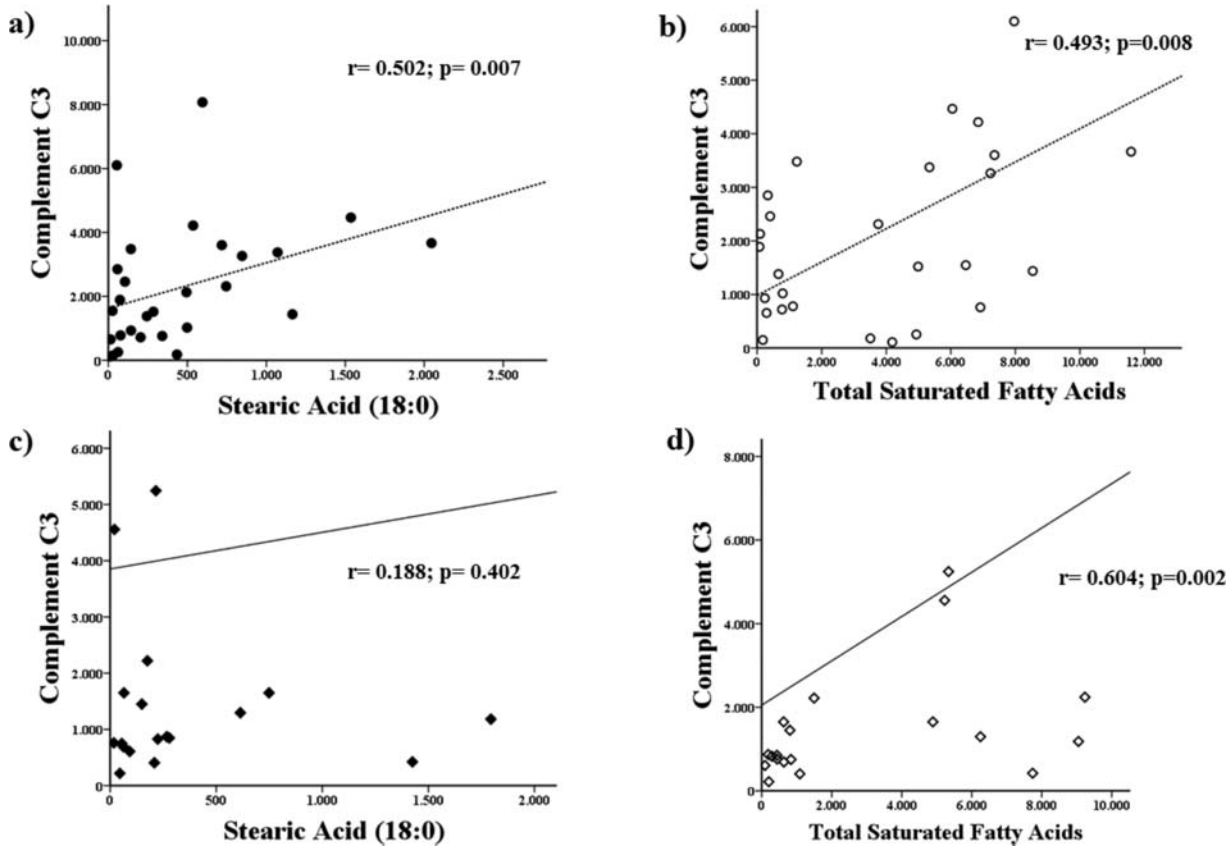

Figure 3. Correlations between incremental area under the curve (iAUC) for complement C3 with stearic acid and total saturated fatty acids. (a) Complement C3 and stearic acid in normal-weight group; (b) complement C3 and total saturated fatty acids in normal-weight group; (c) complement C3 and stearic acid in the overweight group; (d) complement C3 and total saturated fatty acids in the overweight group. The p values are by Pearson correlation or Spearman.

baseline plasma SFA (Table 3). When we categorized the volunteers according to the median of plasma baseline MUFA, the 5-hour iAUC values for complement C3 after HMM were lower than in those participants with high plasma MUFA (Table 3). When we categorized the volunteers according to the median of plasma baseline Apo A1, the 5-hour iAUC values for complement C3 after HMM were lower than in those participants with high plasma Apo A1 (Table 4).

Table 3. iAUC of the lipid metabolism markers after HSM and HMM, according to the median values of baseline total SFA and MUFA.

iAUC of variables	Total saturated fatty acids			Total monounsaturated fatty acids		
HSM	<69.3 (%)	≥69.3 (%)	*p* Values*	<25.1 (%)	≥25.1 (%)	*p* Values*
Palmitic acid (16:0) (%)[a]	384 (101–801)	1286 (666–1993)	0.006	1456 (909–2974)	587 (375–866)	0.003
Stearic acid (18:0) (%)[a]	215 (23–747)	782 (396–1508)	0.012	1071 (276–1665)	411 (35–739)	0.032
Total SFA (%)[a]	5224 (4184–.492)	7778 (6994–9353)	0.003	9048 (7228–9721)	5792 (4360–6899)	0.000
Oleic acid (18:1) (%)	1165 (1384)	693 (421)	0.205	1112 (1017)	736 (960)	0.316
Total MUFA (%)[a]	827 (328–4046)	999 (599–1957)	0.892	2141 (1126–4046)	544 (317–851)	0.000
Apolipoprotein A1 (mg/dL)	1678 (1346)	1319 (1074)	0.443	1233 (1051)	1728 (1323)	0.289
Apolipoprotein B100 (mg/dL)	1828 (1664)	1622 (1978)	0.768	1941 (1633)	1537 (1965)	0.562
Triacylglycerol (mg/dL)	7160 (7242)	5778 (3656)	0.504	6852 (5189)	6068 (6153)	0.705
Complement C3 (mg/dL)[a]	1650 (1028–4715)	3666 (1438–8073)	0.217	4467 (2753–7087)	1548 (760–4218)	0.058
HMM	<31.2	≥ 31.2	*p* Values	<26.7	≥26.7	*p* Values
Palmitic acid (16:0) (%)[a]	522 (257–1.078)	257 (137–1127)	0.264	510 (247–1203)	496 (150–1012)	0.362
Stearic acid (18:0) (%)[a]	149 (60–261)	105 (60–246)	0.801	204 (98–359)	65 (50–200)	0.044
Total SFA (%)[a]	772 (363–961)	290 (138–871)	0.169	660 (164–946)	435 (192–972)	0.920
Oleic acid (18:1) (%)	1538 (986)	1202 (1055)	0.411	1607 (1010)	1133 (1002)	0.241
Total MUFA (%)[a]	1260 (880–2311)	1069 (201–1814)	0.311	1622 (910–2441)	1069 (145–1264)	0.064
Apolipoprotein A1 (mg/dL)	1313 (1215)	1391 (1280)	0.874	1256 (1271)	1447 (1218)	0.699
Apolipoprotein B100 (mg/dL)	1356 (1006)	1810 (2200)	0.505	1838 (2159)	1327 (1077)	0.452
Triacylglycerol (mg/dL)	4755 (4535)	3697 (2232)	0.489	4852 (4562)	3688 (2453)	0.444
Complement C3 (mg/dL)[a]	848 (531–1550)	870 (620–2220)	0.531	975 (620–1780)	838 (706–2078)	0.932

Note: SFA saturated fatty acids, MUFA monounsaturated fatty acids. *These are *p* values by Student's *t*-test or Mann–Whitney test according to the normality of the variables. [a]Nonparametric variables. Data expressed as mean ± standard deviation or median (CI 25–75th percentile), respectively, the incremental area under the curve (iAUC).

Table 4. iAUC of the markers of lipid metabolism after HSM and HMM, according to the median values of baseline total apolipoproteins.

iAUC of variables	Apolipoprotein A1			Apolipoprotein B 100		
HSM	<127 (mg/dL)	≥127 (mg/dL)	*p* Values	<86.1 (mg/dL)	≥86.1 (mg/dL)	*p* Values*
Palmitic acid (16:0) (%)*	602 (199–1379)	950 (533–2263)	0.231	673 (101–2218)	817 (587–1.443)	0.614
Stearic acid (18:0) (%)*	566 (346–819)	501 (46–1323)	0.899	596 (278–1.118)	536 (23–1319)	0.614
Total SFA (%)*	7242 (6.370–9587)	6464 (4987–9048)	0.496	7242 (5337–8997)	6849 (4987–9232)	0.821
Oleic acid (18:1) (%)	1065 (1306)	839 (745)	0.587	1180 (1242)	707 (726)	0.247
Total MUFA (%)*	859 (353–2403)	1126 (633–2178)	0.440	871 (483–1746)	859 (437–2788)	1.000
Apolipoprotein A1 (mg/dL)	1234 (1123)	1727 (1.272)	0.290	1264 (1121)	1701 (1284)	0.350
Apolipoprotein B100 (mg/dL)	1813 (2357)	1648 (1.205)	0.813	921 (643)	2421 (2181)	0.025
Triacylglycerol (mg/dL)	5515 (4945)	7182 (6.663)	0.465	4669 (6463)	7915 (5068)	0.149
Complement C3 (mg/dL)*	4218 (761–8073)	2241 (1438–4557)	0.474	2844 (894–7498)	2922 (1436–4636)	0.860
HMM	<132	≥132	*p* Values*	<75.5	≥75.5	*p* Values
Palmitic acid (16:0) (%)[a]	522 (267–1207)	237 (137–851)	0.072	339 (204–1207)	510 (208–905)	0.880
Stearic acid (18:0) (%)[a]	149 (75–339)	92 (37–246)	0.169	77 (51–255)	149 (77–252)	0.511
Total SFA (%)[a]	772 (366–1099)	290 (181–734)	0.186	330 (181–1099)	633 (263–820)	0.579
Oleic acid (18:1) (%)	1560 (981)	1179 (1051)	0.349	1691 (1191)	1049 (710)	0.108
Total MUFA (%)[a]	1260 (910–2441)	1177 (201–1814)	0.287	1260 (702–2599)	1177 (349–1474)	0.287
Apolipoprotein A1 (mg/dL)	1016 (983)	1687 (1382)	0.166	1300 (1153)	1403 (1335)	0.835
Apolipoprotein B100 (mg/dL)	1319 (1278)	1847 (2044)	0.437	1165 (871)	2000 (2197)	0.215
Triacylglycerol (mg/dL)	4996 (4303)	3412 (2571)	0.297	3552 (1610)	5119 (5075)	0.303
Complement C3 (mg/dL)[a]	1450 (750–2.295)	750 (608–870)	0.041	870 (313–2010)	848 (720–1.450)	0.865

Note: SFA saturated fatty acids, MUFA monounsaturated fatty acids. *These are *p* values by Student's *t*-test or Mann–Whitney test according to the normality of the variables. [a]Nonparametric variables. Data expressed as mean ± standard deviation or median (CI 25–75th percentile), respectively, the incremental area under the curve (iAUC).

Discussion

The first important finding from this work was the significant increase in the complement C3 concentrations after the intake of HSM, compared with HMM. Such an increase in the concentration of this protein after a high-fat meal has been also reported in earlier studies (30–32). However, this is the first report in which postprandial response is differentiated by the type of dietary fat.

Complement C3, linked with TAG synthesis and storage, has been recognized as an independent marker of CVD. In addition, complement C3 plays role in inflammation and the development of nonfunctional apolipoprotein A1 particles, which favor the development of CVD (33). Moreover, high circulating C3 has been related to postprandial lipemia, fasting insulin concentrations, and thrombotic processes (30,31,34).

Thus, the findings from this study could indicate that the secretion of complement C3 is another pro-inflammatory and potentially pro-atherogenic mechanism of SFA. This study also revealed positive correlations between the complement C3 concentrations and plasma stearic acid and total SFA. The saturated fatty acids palmitic and stearic are the most abundant in natural fats; in addition, stearic acid has a neutral effect on cholesterol concentrations, but has been related to the change in biochemical markers predictive of the risk or the progression of cardiovascular diseases (35–37).

In turn, the complement C3 concentrations were higher in the OW group compared with the NW group. This result is supported by prior studies that reported a positive correlation between complement C3 and adiposity (34,38–40), as well as a reduction in their values after dietary intervention for weight loss (38,39,41,42). However, the positive relationship between SFA and the complement C3 was independent of weight status, which implies that this relationship may be established earlier than body fat accumulation. On the other hand, their relationship is closer to abdominal obesity (33,39,43). In this study, only 20 of the 63 participants had abdominal obesity (waist circumference of ≥80cm)(21).

The second significant finding of this study was the postprandial change in the plasma oleic acid after HMM consumption. Oleic acid values remained high after 5 hours postprandial in the NW compared with the OW group. This implies a protection in the normal-weight individuals compared with those who were overweight. In fact, excessive body fat accumulation may result in blood lipid imbalance that, in turn, may result in their storage in the liver, muscle, and adipose tissue itself (4,6,22).

Our third finding was that the postprandial response to the HSM was mediated by the basal fatty acid (FA) profile of the volunteers. Thus, those individuals with a high plasma total SFA had greater iAUC for palmitic acid, stearic acid, and total fatty acids, while those women with high MUFA showed less change in the iAUC for the same fatty acids profiles, suggesting a protection of the MUFA front of a saturated lipid load. This finding may indicate a cumulative effect of the acute response to a prior atherogenic lipid profile. However, in the group that ingested the HMM there were no differences in the postprandial fatty acids between the groups. In fact, the consumption of the high MUFA content meal compared with other fat sources has been shown to have a beneficial effect on postprandial lipid response in healthy adults (44–47).

Interestingly, those subjects with high values of Apo B100, a recognized atherogenic marker, showed a higher increase in Apo B100 after HSM intake, compared with those having low Apo B100 concentration at the start of intervention. After HMM intake no differences were observed between the increments after the meal. Therefore, our results suggest plasma total SFA and Apo B100 at baseline influence the postprandial response.

TAG concentrations showed no difference between the groups (NW and OW) and between the diets offered (HMM and HSM), although differences were expected. Previous studies have showed results similar to ours with respect to the TAG concentrations, with no difference between the lipid sources offered (15,18,48,49). In others, a relationship between meals and the weight status is observed, which is explained by the difference between the methodologies such as postprandial time or different SFA and MUFA sources (16,17,19).

In conclusion, the present study revealed, apparently for the first time, the effect of HSM in the postprandial increment in complement C3 concentrations, compared with the HMM. Further, the plasma oleic acid value remained high after 5hours from HMM consumption in the NW compared to the OW group, suggesting a protection marker in NW individuals. Finally, the postprandial response after HSM intake appears to be mediated by baseline FA profile, which suggests a cumulative effect of the acute response to a lipid profile previously established.

This study used a standard meal as a source of lipids for all volunteers. The use of a lipid load in place of the adequacy of the amount of lipids offered by body weight may be considered as a limitation of the study. However, this methodology is valid and widely used in postprandial studies with a design and objectives similar to ours.

Acknowledgments

We thank the volunteers who participated for the study. We also thank the CAPES Foundation and National Council for Scientific and Technological Development (CNPq/MCT/Brazil) for the scholarship granted to L. L. Lopes and D. M. U. P. Rocha, and to A. Silva, respectively. This work was supported by the Foundation for Research Support of the State of Minas Gerais (FAPEMIG) and the CNPq. M. C. G. Peluzio, J. Bressan, and H. H. M. Hermsdorff are CNPq Fellows.

Ethical standards

The Research Ethics Committee of the Universidade Federal de Viçosa (UFV) provided confirmation of fulfillment of the ethical standards affecting this research (Of. Ref. No. 184/2011 and CAAE 542,585/2014). Therefore, the survey was in accordance with the principles of the 1964 Declaration of Helsinki and its later amendments.

Conflict of interest

The authors declare that they have no conflict of interest.

Funding

Financial support was provided by the CAPES Foundation, National Council for Scientific, Technological Development (CNPq/MCT/Brazil), and Foundation for Research Support of the State of Minas Gerais (FAPEMIG).

ORCID

Daniela Mayumi U. P. Rocha http://orcid.org/0000-0001-6130-0179

References

1. Hermsdorff HHM, Zulet MA, Puchau B, Martínez JA. Fruit and vegetable consumption and proinflammatory gene expression from peripheral blood mononuclear cells in young adults: a translational study. Nutr Metab (Lond). 2010;7:42. doi:10.1186/1743-7075-7-42.
2. Hermsdorff HHM, Barbosa KBF, Volp ACP, Puchau B, Bressan J, Zulet MÁ, Martínez JA. Vitamin C and fibre consumption from fruits and vegetables improves oxidative stress markers in healthy young adults. Br J Nutr. 2012;107:1119–27. doi:10.1017/S0007114511004235.
3. Bressan J, Hermsdorff HHM, Zulet MA, Martínez JA. [Hormonal and inflammatory impact of different dietetic composition: emphasis on dietary patterns and specific dietary factors]. Arq Bras Endocrinol Metabol. 2009;53:572–81. doi:10.1590/S0004-27302009000500010.
4. Volp ACP, Bressan J, Hermsdorff HHM, Zulet MÁ, Martínez JA. Efeitos antioxidantes do selênio e seu elo com a inflamação e síndrome metabólica. Rev Nutr. 2010;23:581–90. doi:10.1590/S1415-52732010000400009.
5. Bhupathiraju SN, Tucker KL. Coronary heart disease prevention: nutrients, foods, and dietary patterns. Clin Chim Acta. 2011;412:1493–514. doi:10.1016/j.cca.2011.04.038.
6. Chow C, editor. Fatty acids in food and their health implications, 3rd ed. Boca Raton (FL): CRC Press; 2008.
7. Hermsdorff HHM, Barbosa KB, Volp ACP, Puchau B, Bressan J, Zulet MÁ, Martínez JA. Gender-specific relationships between plasma oxidized low-density lipoprotein cholesterol, total antioxidant capacity, and central adiposity indicators. Eur J Prev Cardiol. 2012;21:884–91. doi:10.1177/2047487312472420.
8. Santos R, Gagliardi A, Xavier H, Magnoni C, Cassani R, Lottenberg A. I Diretriz sobre o consumo de Gorduras e Saúde Cardiovascular. Arq Bras Cardiol. 2013;100:1–40.
9. Xavier HT, Izar MC, Faria Neto JR, Assad MH, Rocha VZ, Sposito AC, Fonseca FA, dos Santos JE, Santos RD, Bertolami MC, et al. V diretriz brasileira de dislipidemias e prevenção da aterosclerose. Arq Bras Cardiol 2013;101:1–20. doi:10.5935/abc.2013S010.
10. Arapostathi C, Tzanetakou IP, Kokkinos AD, Tentolouris NK, Vlachos IS, Donta I A, Perrea KN, Perrea DN, Katsilambros NL. A diet rich in monounsaturated fatty acids improves the lipid profile of mice previously on a diet rich in saturated fatty acids. Angiology. 2011;62:636–40. doi:10.1177/0003319711403734.
11. Hermsdorff HHM, Volp ACP, Santos RGC, Viana ML, Bressan J. [Effect of diet macronutrients profile on leptin concentration]. Arq Bras Endocrinol Metabol. 2006;50:934–43. doi:10.1590/S0004-27302006000500017.
12. Volp ACP, Hermsdorff HHM, Bressan J. Glycemia and insulinemia evaluation after high-sucrose and high-fat diets in lean and overweight/obese women. J Physiol Biochem. 2008;64:103–13. doi:10.1007/BF03168238.
13. Sahade V, França S, Badaró R, Fernando Adán L. Obesity and postprandial lipemia in adolescents: risk factors for cardiovascular disease. Endocrinol y Nutr. 2012;59:131–39. doi:10.1016/j.endonu.2011.08.004.
14. Adabag S, Huxley RR, Lopez FL, Chen LY, Sotoodehnia N, Siscovick D, Deo R, Konety S, Alonso A, Folsom AR. Obesity related risk of sudden cardiac death in the atherosclerosis risk in communities study. Heart. 2015;101:215–21. doi:10.1136/heartjnl-2014-306238.
15. Bouwens M, Grootte Bromhaar M, Jansen J, Müller M, Afman L. Postprandial dietary lipid-specific effects on human peripheral blood mononuclear cell gene expression profiles. Am J Clin Nutr. 2010;91:208–17. doi:10.3945/ajcn.2009.28586.
16. Hartwich J, Leszczynska-Golabek I, Kiec-Wilk B, Siedlecka D, Pérez-Martinez P, Marin C, López-Miranda J, Tierney A, Monagle JM, Roche HM, et al. Lipoprotein profile, plasma ischemia modified albumin and LDL density change in the course of postprandial lipemia. Insights from the LIPGENE study. Scand J Clin Lab Invest. 2010;70:201–8. doi:10.3109/00365511003663630.
17. Jiménez-Gómez Y, Marın C, Pérez-Martinéz P, Hartwich J, Malczewska-maleczewska-Malec M, Golabek I, Kiec-Wilk B, Cruz-Teno C, Rodríguez F, et al. A low-fat, high-complex carbohydrate diet supplemented with long-chain (n-3) fatty acids alters the postprandial lipoprotein profile in patients with metabolic syndrome. J Nutr. 2010;140(9):1595–601.
18. Lozano A, Perez-Martinez P, Marin C, Tinahones FJ, Delgado-Lista J, Cruz-Teno C, Gomez-Luna P, Rodriguez-Cantalejo F, Perez-Jimenez

F, Lopez-Miranda J. An acute intake of a walnut-enriched meal improves postprandial adiponectin response in healthy young adults. Nutr Res. 2013;33:1012–18. doi:10.1016/j.nutres.2013.08.010.
19. Teng KT, Nagapan G, Cheng HM, Nesaretnam K. Palm olein and olive oil cause a higher increase in postprandial lipemia compared with lard but had no effect on plasma glucose, insulin and adipocytokines. Lipids. 2011;46:381–88. doi:10.1007/s11745-010-3516-y.
20. Hermsdorff HHM, Puchau B, Zulet MA, Martínez JA. Association of body fat distribution with proinflammatory gene expression in peripheral blood mononuclear cells from young adult subjects. OMICS. 2010;14:297–307. doi:10.1089/omi.2009.0125.
21. Associação Brasileira para o Estudo da Obesidade e da Síndrome Metabólica Diretrizes brasileiras de obesidade 2009/2010/ABESO. Associação Brasileira para o Estudo da Obesidade e da Síndrome Metabólica, 3rd ed. Itapevi, São Paulo, Brazil: AC Farmacêutica; 2009.
22. Hermsdorff HHM, Monteiro JBR. Gordura visceral, subcutânea ou intramuscular: onde está o problema?. Arq Bras Endocrinol Metabol. 2004;48:803–11. doi:10.1590/S0004-27302004000600005.
23. Lozano A, Perez-Martinez P, Delgado-Lista J, Marin C, Cortes B, Rodriguez-Cantalejo F, Gomez-Luna MJ, Cruz-Teno C, Perez-Jimenez F, Lopez-Miranda J. Body mass interacts with fat quality to determine the postprandial lipoprotein response in healthy young adults. Nutr Metab Cardiovasc Dis. 2012;22:355–61. doi:10.1016/j.numecd.2010.07.013.
24. Dietary Reference Intakes for Energy, Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Protein, and Amino Acids (Macronutrients). Washington, DC: National Academies Press; 2005. Available from: http://www.nap.edu/catalog/10490/dietary-reference-intakes-for-energy-carbohydrate-fiber-fat-fatty-acids-cholesterol-protein-and-amino-acids-macronutrients
25. Folch J, Lees M, Stanley GHS. A simple method for the isolation and purification of total lipides from animal tissues. J. Biol. Chem. 1957;226(1):497–509.
26. Hartman L, Lago RC. Rapid preparation of fatty acid methyl esters from lipids. Lab Pract. 1973;22:475–76.
27. Hermsdorff HHM, Zulet MA, Puchau B, Bressan J, Martínez JA. Association of retinol-binding protein-4 with dietary selenium intake and other lifestyle features in young healthy women. Nutrition. 2009;25:392–99. doi:10.1016/j.nut.2008.09.015.
28. Willett W. Nutritional epidemiology (monographs in epidemiology and biostatistics), 3rd ed. New York, NY: Oxford University Press; 2012.
29. Bray G, Bouchard C, James WPT. Definitions and proposed current classifications of obesity. In: Bray G, Bouchard C, James WPT, editors. Handbook of obesity. New York, NY: Marcel Dekker; 1998. p. 31–40.
30. Halkes CJM, van Dijk H, de Jaegere PPT, Plokker HWM, van der Helm Y, Erkelens DW, Castro Cabezas M. Postprandial increase of complement component 3 in normolipidemic patients with coronary artery disease: effects of expanded-dose simvastatin. Arterioscler Thromb Vasc Biol. 2001;21:1526–30. doi:10.1161/hq0901.095276.
31. Meijssen S. Delayed and exaggerated postprandial complement component 3 response in familial combined hyperlipidemia. Arterioscler Thromb Vasc Biol. 2002;22:811–16. doi:10.1161/01.ATV.0000014079.98335.72.
32. van Oostrom AJHHM, Alipour A, Plokker TWM, Sniderman AD, Cabezas MC. The metabolic syndrome in relation to complement component 3 and postprandial lipemia in patients from an outpatient lipid clinic and healthy volunteers. Atherosclerosis. 2007;190:167–73. doi:10.1016/j.atherosclerosis.2006.01.009.
33. Barbu A, Hamad O A, Lind L, Ekdahl KN, Nilsson B. The role of complement factor C3 in lipid metabolism. Mol Immunol. 2015;67:101–7. doi:10.1016/j.molimm.2015.02.027.
34. Onat A, Can G, Rezvani R, Cianflone K. Complement C3 and cleavage products in cardiometabolic risk. Clin Chim Acta. 2011;412:1171–79. doi:10.1016/j.cca.2011.03.005.
35. Legrand P, Rioux V. The complex and important cellular and metabolic functions of saturated fatty acids. Lipids. 2010;45:941–46, doi:10.1007/s11745-010-3444-x.
36. Watts GF, Jackson P, Burke V, Lewis B. Dietary fatty acids and progression of coronary artery disease in men. Am J Clin Nutr. 1996;64:202–9.
37. Sundram K, Karupaiah T, Hayes KC. Stearic acid-rich interesterified fat and trans-rich fat raise the LDL/HDL ratio and plasma glucose relative to palm olein in humans. Nutr Metab (Lond). 2007;4:3. doi:10.1186/1743-7075-4-3.
38. Hermsdorff HHM, Zulet MÁ, Puchau B, Martínez JA. Central adiposity rather than total adiposity measurements are specifically involved in the inflammatory status from healthy young adults. Inflammation. 2011;34:161–70. doi:10.1007/s10753-010-9219-y.
39. Hermsdorff HHM, Volp ACP, Puchau B, Barbosa KBF, Zulet MA, Bressan J, Martínez JA. Contribution of gender and body fat distribution to inflammatory marker concentrations in apparently healthy young adults. Inflamm Res. 2012;61:427–35. doi:10.1007/s00011-011-0429-z.
40. Volp ACP, Barbosa KBF, Bressan J. Triacylglycerols and body fat mass are possible independent predictors of C3 in apparently healthy young Brazilian adults. Nutrition. 2012;28:544–50. doi:10.1016/j.nut.2011.08.014.
41. Hermsdorff HHM, Zulet MÁ, Abete I, Martínez JA. Discriminated benefits of a Mediterranean dietary pattern within a hypocaloric diet program on plasma RBP4 concentrations and other inflammatory markers in obese subjects. Endocrine. 2009; 36:445–51. doi:10.1007/s12020-009-9248-1.
42. Hermsdorff HHM, Zulet MÁ, Abete I, Martínez JA. A legume-based hypocaloric diet reduces proinflammatory status and improves metabolic features in overweight/obese subjects. Eur J Nutr 2011;50:61–69. doi:10.1007/s00394-010-0115-x.
43. Prestes J, Frollini AB, Borin JP, Moura NA, Nardo Júnior N, Perez SE de A. Efeitos de um treinamento de 16 semanas sobre a composição corporal de homens e mulheres. Rev Bras Atividade Física Saúde. 2012;11:19–28.
44. Gilmore LA, Walzem RL, Crouse SF, Smith DR, Adams TH, Vaidyanathan V, Cao X, Smith SB. Consumption of high-oleic acid ground beef increases HDL-cholesterol concentration but both high- and low-oleic acid ground beef decrease HDL particle diameter in normocholesterolemic men. J Nutr. 2011;141:1188–94. doi:10.3945/jn.110.136085.
45. Perez-Martinez P, Ordovas JM, Garcia-Rios A, Delgado-Lista J, Delgado-Casao N, Cruz-Teno C, Camargo A, Yubero-Serrano EM, Rodriguez F, Perez-Jimenez F, et al. Consumption of diets with different type of fat influences triacylglycerols-rich lipoproteins particle number and size during the postprandial state. Nutr Metab Cardiovasc Dis. 2011;21:39–45. doi:10.1016/j.numecd.2009.07.008.
46. Raz O, Steinvil A, Berliner S, Rosenzweig T, Justo D, Shapira I. The effect of two iso-caloric meals containing equal amounts of fats with a different fat composition on the inflammatory and metabolic markers in apparently healthy volunteers. J Inflamm (Lond). 2013;10:3. doi:10.1186/1476-9255-10-3.
47. Varela LM, Ortega-Gomez A, Lopez S, Abia R, Muriana FJG, Bermudez B. The effects of dietary fatty acids on the postprandial triglyceride-rich lipoprotein/apoB48 receptor axis in human monocyte/macrophage cells. J Nutr Biochem. 2013;24:2031–39. doi:10.1016/j.jnutbio.2013.07.004.
48. Lopez S, Bermudez B, Ortega A, Varela LM, Pacheco YM, Villar J, Abia R, Muriana FJ. Effects of meals rich in either monounsaturated or saturated fat on lipid concentrations and on insulin secretion and action in subjects with high fasting triglyceride concentrations. Am J Clin Nutr. 2011;93:494–99. doi:10.3945/ajcn.110.003251.
49. Dijk SJ, Van Feskens EJ, Bos MB, de Groot LC, de Vries JH, Müller M, Afman LA. Consumption of a high monounsaturated fat diet reduces oxidative phosphorylation gene expression in peripheral blood mononuclear cells of abdominally overweight men. J Nutr. 2012;142(7):1219–25. doi:10.3945/jn.111.155283.