

# Effects of coconut oil consumption on energy metabolism, cardiometabolic risk markers, and appetitive responses in women with excess body fat

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## Abstract

**Purpose** Virgin coconut oil (VCO) is a medium-chain fatty acid source with popularly attributed benefits on obesity management. However, its role on obesity requires elucidation due to its saturated nature. In the study herein, we investigated acute effects of VCO consumption on energy metabolism, cardiometabolic risk markers, and appetitive responses in women with excess body fat.

**Methods** Fifteen adult women with excess body fat ( $37.43 \pm 0.83\%$ ) participated in this randomized, crossover, controlled study. Two isocaloric mixed breakfasts containing 25 mL of VCO or control (extra-virgin olive oil-C) were evaluated. Resting energy expenditure (REE), fat oxidation rate (FOR), diet induced thermogenesis (DIT) and appetitive subjective responses were assessed at fasting and postprandial periods (up to 240 min). Cardiometabolic risk markers were assessed at fasting and up to 180 min postprandially.

**Results** VCO did not affect REE, FOR, and DIT compared to C. In addition, VCO did not cause deleterious change in triglycerides, total cholesterol, HDL-c, LDL-c, triglycerides/HDL-c ratio, uric acid, glucose and Homeostasis Model Assessment of Insulin Resistance Index (HOMA-IR)

( $P_{\text{time} \times \text{treatment}} > 0.05$ ). However, VCO suppressed less hunger ( $P_{\text{time} \times \text{treatment}} = 0.003$ ), total satiety ( $P_{\text{iAUC}} = 0.021$ ) and total fullness ( $P_{\text{iAUC}} = 0.035$ ) responses than C.

**Conclusions** VCO consumption did not acutely change energy metabolism and cardiometabolic risk markers when added to a mixed breakfast but promoted less appetitive responses.

**Keywords** Coconut oil · Energy metabolism · Fat oxidation · Cardiometabolic risk markers · Appetite

## Introduction

Obesity and overweight remain to be a serious public health problem despite of international efforts to combat them. Excess body fat is related to non-communicable diseases (NCDs) such as cardiovascular diseases, hypertension, diabetes, and some types of cancer [1]. Data from the latest World Health Organization report [2] showed that NCDs have been responsible for around 38 million deaths per year since 2012, accounting for 68% of all deaths worldwide. Cardiovascular diseases were the leader in mortality, which claimed 17.5 million lives in 2012 (46% of all NCDs deaths), 6 million of which were premature.

Virgin coconut oil (*Cocos nucifera* L.) (VCO) is a high quality source of medium-chain fatty acids (MCFA) with commercial weight loss claims. It has been attributed to coconut oil thermogenic effects, increased fullness responses and HDL-c improvement [3, 4]. These are based on the fact that MCFA could be absorbed and metabolized faster than others fatty acids [5]. The theory is that faster metabolism could boost energy and fat metabolism without promoting fat storage and dyslipidemia and could enhance satiety [6, 7] thus favoring weight loss. These

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claims attract consumers' attention [8] but it lacks scientific confirmation.

While some studies reported beneficial effects of coconut oil or isolated MCFA on energy metabolism [9], fat oxidation [10, 11], food intake [12, 13], and no detrimental effect on serum cardiometabolic risk markers [14–17], others demonstrated negative results on lipid profile [18], and conflicting results on metabolic rates [11, 19], and satiety [20, 21]. Contradictory results could be partly explained by the distinct fatty acid composition of VCO compared to the synthetic source of MCFA adopted in the majority of studies [9, 11–13, 16, 18, 19], enforcing the need for studies conducted with VCO. Furthermore, randomized controlled clinical trials about VCO are scarce, present methodological limitations such as unusual doses of oil consumption, and/or exhibit inconclusive results [22].

Because women are the main coconut oil consumers due to its weight loss claims and previous studies have lead us to question the applicability of MCFA-rich oil claims in women [23], the aim of this study was to evaluate acute effects of reasonable amounts of VCO intake on energy metabolism, serum cardiometabolic risk markers, and subjective appetitive responses in women with excess body fat.

## Methods

### Study population

Seventeen women aged 19–42 years with BMI between 25 and 31 kg/m<sup>2</sup> and total body fat >32% were recruited through local advertisement (Supplementary Figure 1). Exclusion criteria were: known chronic illnesses except obesity, fasting glucose >5.5 mmol/L, hypertension (>140/90 mmHg), smoking, alcohol consumption (>2 doses/d), pregnancy or lactation, recent changes (previous three months) in diet or physical activities habits, elite athletes (>10 h/week), use of dietary supplements or drugs except oral contraceptive, food allergies or intolerances, and aversion to the tested ingredients. Based on published values of fasting fat oxidation and estimated change of 3.6 g during the postprandial state [24], a sample size of 12 subjects was estimated for this crossover study design using the formula by Mera et al. [25]. Accounting for dropouts (30%), seventeen subjects have been enrolled. Participants gave written consent after receiving verbal and written information. The study was performed at Laboratory of Energy Metabolism and Body Composition of Nutrition and Health Department, Universidade Federal de Viçosa, Brazil. The study protocol was approved by the Ethics Committee of Universidade Federal de Viçosa (protocol number: 541.836/2014), conducted in

accordance with Helsinki declaration and its later amendments and registered at <http://www.ensaiosclinicos.gov.br/> (RBR-8NGPQ9).

### Study design

This was a randomized single-blinded crossover design study in which two different isocaloric breakfasts were tested in non-consecutive days. Subjects were randomly assigned by simple draw to either control (extra-virgin olive oil—EVOO) or VCO breakfast, with washout period of one week.

Prior to test days, participants were instructed to maintain their usual dietary intake and to abstain from alcohol and strenuous physical activity. On the night before each test day, all participants consumed standardized dinner (2.5 MJ; 62E% carbohydrate; 8.5E% protein; 29.4E% fat) and water intake was allowed until 4 h before starting the test. Immediately before dinner consumption, participants emptied their bladder and thenceforth all urine was collected and carried to laboratory.

In each test day, participants attended the laboratory from 7 a.m. to 1 p.m. after 11 h overnight fasting with minimal physical effort possible. The remaining 1 h fasting was completed at laboratory, totaling 12 h of fasting before test drink consumption. Anthropometric measurements were recorded by trained appraiser. Body weight was measured using digital platform scale with resolution of 0.5 kg (Toledo<sup>®</sup>, Model 2096PP/2, São Paulo, Brazil), while participants were barefoot and wearing lightweight clothing. Height was measured with wall-mounted stadiometer (Wiso, Chapecó, SC, Brazil) to the closest value to 0.1 cm. Total body fat was assessed by bioelectrical impedance (model Y230, InBody Co. Lta., Gyeonggi, Korea). Then, subjects remained in lay supine for mandatory 15 min rest period. After that, fasting resting energy expenditure (REE) and substrate oxidation rates (SOR) were assessed by indirect calorimetry (Carefusion Vmax<sup>®</sup> Series, California, EUA) for 30 min and participants remained awake and motionless as much as possible. This protocol comprises fasting REE not very different from a basal energy expenditure measurement obtained immediately on awaking, after an overnight stay in the laboratory [26]. Urine was collected to complete 12 h of urine collection. Fasting (12 h) antecubital blood sample was drawn and subjects consumed one of two mixed breakfasts containing 25 mL of control (EVOO) or test (VCO) oils within 15 min. Meal palatability questionnaire was completed in this period.

Indirect calorimetry followed intermittent protocol previously developed [27], in which measurements were made every half for each hour during 4 h following breakfast. During protocol intervals, subjects remained awake but inactive and they could not leave the laboratory. Water at

room temperature (200 mL) was given in each interval and urine collections were made during all postprandial period. Antecubital blood samples were collected at 60, 120, and 180 min and subjective appetitive responses were taken immediately before/after breakfast consumption and immediately before/after each indirect calorimetry until 240 min.

### Test breakfasts

Participants were assigned to consume 30 to 40% of their daily energy requirements as test breakfasts. It consisted of standard breakfasts containing white bread with 25 mL of control (EVOO) or test (VCO) oils and 300 mL of milk-derived fat-free drink (Table 1). Virgin coconut oil (FidBö, São Paulo, Brazil) and extra-virgin olive oil (Bunge Alimentos, Santa Catarina, Brazil) were maintained protected from light and heat until consumption and fat-free drink was offered at ~10 °C.

Fatty acid compositions of EVOO and VCO were assessed after esterification [29] by gas chromatography (GC). Chromatographic analysis was carried out using Shimadzu GC Solution instrument (Shimadzu Seisakusho Co., Kyoto, Japan) equipped with flame ionization detector (FID) and Carbowax capillary column (30 m × 0.25 mm). Briefly, 1 µL of esterified sample was injected in GC with split ratio of 10. Nitrogen was supplied as carrier gas at flow rate of 43.2 cm/s. The initial oven temperature was 100 °C, maintained for 5 min, then increased to 220 °C at 4 °C/minutes and held for 20 min. Flow rate over column was 1.0 mL/minute. FID and injection port temperature was 200 and 220 °C, respectively. Data handling was carried out using the software GC Solution package (Shimadzu Seisakusho Co., Kyoto, Japan). Fatty acid methyl esters (FAME) were identified by direct comparison of retention time with FAME standard mix (Supelco 37 Component FAME Mix; Sigma-Aldrich®, EUA).

### Energy metabolism measurement and calculation

Flow meter and flow sensor were calibrated daily by a 3.0-L syringe. Analyzers were calibrated with gases of known concentration before each testing as recommended by manufacturer's instructions. The first gas was 26% O<sub>2</sub> with N<sub>2</sub> balance, the second gas was 4% CO<sub>2</sub> 16% O<sub>2</sub>, with N<sub>2</sub>, and the last gas was ambient air.

During measurements, subjects had their head covered with ventilated canopy to quantify oxygen consumed and carbon dioxide produced. They stayed in quiet room with stable temperature (22 °C) and humidity (55%) during measurements. The first 10 min (adaptation phase), and individual outlier values of oxygen and carbon dioxide were excluded from the analyses [25, 30]. Means of oxygen

**Table 1** Nutritional composition of control and coconut oil breakfasts

	Control	Coconut oil
White bread (g)	50	50
Olive oil (mL)	25	–
Coconut oil (mL)	–	25
Powered skim milk (g)	40	40
Strawberry flavoring powder (g)	1	1
Water (mL)	280	280
Energy content (kJ)	494.0	494.0
Carbohydrate (g)	49.0	49.0
(E%)	39.7	39.7
Protein (g)	18.0	18.0
(E%)	14.6	14.6
Total fat (g)	25.0	25.0
(E%)	45.7	45.7
Fatty acid profile (%)		
C8:0	–	5.2
C10:0	–	5.4
C12:0	–	51.6
C14:0	–	19.9
C16:0	10.4	8.8
C18:0	2.7	3.0
C18:1 ω9	79.5	5.1
C18:2 ω6	5.6	0.7
C18:3 ω3	0.5	–
Total MCFA	–	62.3
Total LCFA	100	37.6
Total SFA	13.1	94.0
Total MUFA	80.1	5.1
Total PUFA	6.5	0.7
Fiber (g)	0.0	0.0

Nutritional information was obtained from manufacturer's product information and Brazilian Food Composition Table [28]. Fatty acid profile was obtained after esterification [29] by gas chromatography *MCFA* medium chain fatty acids, *LCFA* long-chain fatty acids, *SFA* saturated fatty acids, *MUFA* monounsaturated fatty acids, *PUFA* polyunsaturated fatty acids

and carbon dioxide volumes (L/min) from the remaining data were used in the calculations.

REE and SOR (carbohydrate, protein and fat oxidation) from fasting and postprandial states were calculated using oxygen and carbon dioxide volumes and urinary nitrogen excretion of each period of time, as described previously [31]. Values of non-protein respiratory quotient (NPRQ) were also calculated [31]. Diet induced thermogenesis (DIT) was assessed [27] and expressed as percentage of breakfasts energy. Total urinary nitrogen excretion was estimated by Kjeldahl technique. Changes between fasting and postprandial carbohydrate and fat oxidation were calculated by subtracting the total postprandial values over 4 h from

fasting value multiplied by duration of measurement (4 h) [24].

### Cardiometabolic risk markers

Serum samples were separated from whole blood by centrifugation (3500 rpm, 4 °C, 15 min) and immediately frozen at −80 °C until analyses. Triglycerides (TG), total cholesterol, high-density lipoprotein cholesterol (HDL-c), low-density lipoprotein cholesterol (LDL-c), glucose, and uric acid were measured by standard colorimetric kits (K082, K117, K083, K071, K088, and K139; Bioclin®, Minas Gerais, Brazil) by automatic biochemical analyzer BS-200 (Mindray Medical International Ltd., Shenzhen, China). Insulin was assessed by chemiluminescence method. Very low-density lipoprotein (VLDL) was estimated by Friedewald et al. [32] formula. Insulin resistance was estimated by Homeostasis Model Assessment Index of Insulin Resistance (HOMA-IR) using Matthews et al. [33] equation. Atherogenic Index (TG/HDL ratio) [34] and total area under the curves (tAUC) of each cardiometabolic risk marker were also calculated [35].

The normal ranges used for fasting parameters were: TG (<1.7 mmol/L), total cholesterol (<5.17 mmol/L), HDL-c (>1.3 mmol/L), LDL-c (<2.6 mmol/L), glucose (<5.55 mmol/L), uric acid (89.22–475.84 µmol/L), insulin (<174 pmol/L), and HOMA-IR (<2.7) [36, 37].

### Appetitive responses

Hunger (“How hungry do you feel?”), fullness (“How full do you feel?”), satisfaction (“How satisfied do you feel?”), and desire to eat specific food types (“Would you like to eat something sweet?”; “Would you like to eat something salty?”; “Would you like to eat something savoury?”; “Would you like to eat something fatty?”) were assessed using 10 unit visual analog scales (VAS) with words anchored at their ends, expressing the most positive and the most negative rating [38]. Incremental area under the curve (iAUC) was determined for fullness and satiety, and incremental area above the curve (iAAC) was determined for hunger and desire to eat specific food types. Incremental areas were assessed by trapezoidal method [35]. VAS were also used to rate palatability by the following questions: visual appeal, smell, taste, aftertaste, and palatability [38].

### Statistical analysis

Statistical analyses were carried out with SPSS 17 for Windows (SPSS, Inc., Chicago, IL, USA). Data are expressed as means and standard errors of the mean (SEM). Individual outlier values of each variable were excluded before analyses. Data normality and homoscedasticity were assessed by Shapiro–Wilk and Levene

tests, respectively. Non-parametric data were log transformed prior to the analysis. Paired *t* test was used to assess significant differences between dietary treatments for areas under (AUC) or above (AAC) the curve and palatability responses. Repeated-measures ANOVA in mixed model setting, considering time as within-subject factor and treatment (breakfast) as between-subject factor were conducted to verify treatment effects on all variables. Paired *t* test with Bonferroni’s correction was performed to assess differences in individual time-points in which differences were expected to arise. The  $\alpha$  level of 5% was considered statistically significant.

## Results

From the seventeen recruited participants two of them failed to complete the study protocol due to personal reasons. Both breakfasts were well tolerated by the 15 participants. There were no differences between breakfasts related to palatability questions ( $P > 0.05$ ). For subjects’ characteristics see Table 2.

### Metabolic rates

There were no differences in fasting metabolic rates between the test days. As expected, there was significant time effect in REE, NPRQ, and all SOR (carbohydrate, protein, and fat oxidation) ( $P_{\text{time}} < 0.001$ ). However, there were no treatment effect or time  $\times$  treatment interaction for any metabolic rate analyzed (Table 3). Similarly, comparisons of fasting and total postprandial states for all metabolic parameters showed only effect of time ( $P_{\text{time}} \leq 0.001$ ). Absence of significant results was

**Table 2** Baseline characteristics of subjects ( $n = 15$ )

Characteristics	Subjects
Age (years)	26.8 $\pm$ 1.37
BMI (kg/m <sup>2</sup> )	27.66 $\pm$ 0.44
Waist circumference (cm)	89.89 $\pm$ 1.59
Hip circumference (cm)	105.66 $\pm$ 0.82
Body fat percentage (%)	37.43 $\pm$ 0.83
Lean mass (kg)	24.42 $\pm$ 0.54
Fat-free mass (kg)	44.1 $\pm$ 0.90
REE (kJ/day)	5392.16 $\pm$ 92.56
REE/kg (kJ/day)	76.55 $\pm$ 1.08

Data are presented as mean  $\pm$  SEM. REE are averaged from baseline values of each participant at control and VCO test days

*BMI* body mass index, *REE* resting energy expenditure

**Table 3** Fasting and postprandial energy expenditure and substrate oxidation rates of subjects consuming control or coconut oil ( $n = 15$ )

Meal induced change	Control		Coconut oil		<i>P</i>
	Fasting state	Postprandial state	Fasting state	Postprandial state	
REE (kJ/day)	5276.08 ± 86.41	5971.99 ± 147.78	5422.52 ± 68.65	5989.97 ± 110.8	Time effect <0.001 Meal effect 0.762 Time × meal 0.227
NPRQ	0.83 ± 0.01	0.86 ± 0.02	0.83 ± 0.01	0.86 ± 0.01	Time effect <b>0.001</b> Meal effect 0.816 Time × meal 0.751
Carbohydrate oxidation (g/h)	4.88 ± 0.36	8.06 ± 0.59	4.92 ± 0.34	8.20 ± 0.39	Time effect <0.001 Meal effect 0.555 Time × meal 0.555
Fat oxidation (g/h)	2.59 ± 0.12	2.88 ± 0.19	2.72 ± 0.16	2.70 ± 0.16	Time effect <b>0.001</b> Meal effect 0.113 Time × meal 0.117
Protein oxidation (g/h)	2.11 ± 0.18	2.50 ± 0.18	2.14 ± 0.20	2.39 ± 0.20	Time effect <0.001 Meal effect 0.898 Time × meal 0.714
Diet induced thermogenesis (% energy intake)	–	4.29 ± 0.51	–	3.88 ± 0.45	0.483*
Change in carbohydrate oxidation (g/4 h) <sup>a</sup>	–	12.24 ± 1.66	–	12.79 ± 1.46	0.801*
Change in fat oxidation (g/4 h) <sup>a</sup>	–	11.34 ± 0.52	–	10.36 ± 0.34	0.240*

Data were expressed as mean ± SEM. Bold letters indicated significant differences (RM-ANOVA in mixed model setting with time as within-subject factor and meal as between-subject factor,  $P < 0.05$ )

REE resting energy expenditure, NPRQ non-protein respiratory quotient

\* Data was analyzed by paired  $t$  test

<sup>a</sup> Calculated as postprandial values over 4 h – (fasting value per h × 4)

maintained when DIT and total postprandial carbohydrate and fat oxidation were assessed (Table 3).

### Cardiometabolic risk markers

All participants had normal uric acid fasting range (minimal/maximal: 130.86/285.50  $\mu\text{mol/L}$ ). For other cardiometabolic markers, 13 participants showed normal fasting range for glucose (minimal/maximal, 4.05/6.49  $\text{mmol/L}$ ), 13 for insulin (25.7/260.44  $\text{pmol/L}$ ), 8 for HOMA index (0.75/10.82), 10 for triglycerides (0.46/2.09  $\text{mmol/L}$ ), 13 for total cholesterol (3.13/5.72  $\text{mmol/L}$ ), 8 for HDL-c (0.59/1.99  $\text{mmol/L}$ ), and 12 for LDL-c (1.06/3.41  $\text{mmol/L}$ ). Blood assays indicated typical middle-aged obesity profile with some individuals exhibiting disruption in cardiometabolic risk markers.

There was significant time × treatment interaction for uric acid concentrations between breakfasts ( $P_{\text{time} \times \text{treatment}} = 0.006$ ); however, such interaction did not persist after post hoc analysis ( $P > 0.050$ ). Furthermore, there was trend to time × treatment interaction in HDL-c levels at time 60 min ( $P_{\text{time} \times \text{treatment}} = 0.055$ ).

There were no significant changes in total postprandial responses between treatments (Table 4).

### Appetitive responses

Analyses of subjective hunger responses showed significant time × treatment interaction ( $P_{\text{time} \times \text{treatment}} = 0.003$ ) with VCO breakfast presenting lesser hunger suppression at 240 min when compared to control breakfast ( $P = 0.019$ ). Further, VCO had significantly lesser total satiety ( $P_{\text{IAUC}} = 0.021$ ) and total fullness ( $P_{\text{IAUC}} = 0.035$ ) responses when compared to control (Fig. 1). Additional questions regarding desire to eat specific food types showed no significant differences between dietary treatments (Supplementary Fig. 2).

### Discussion

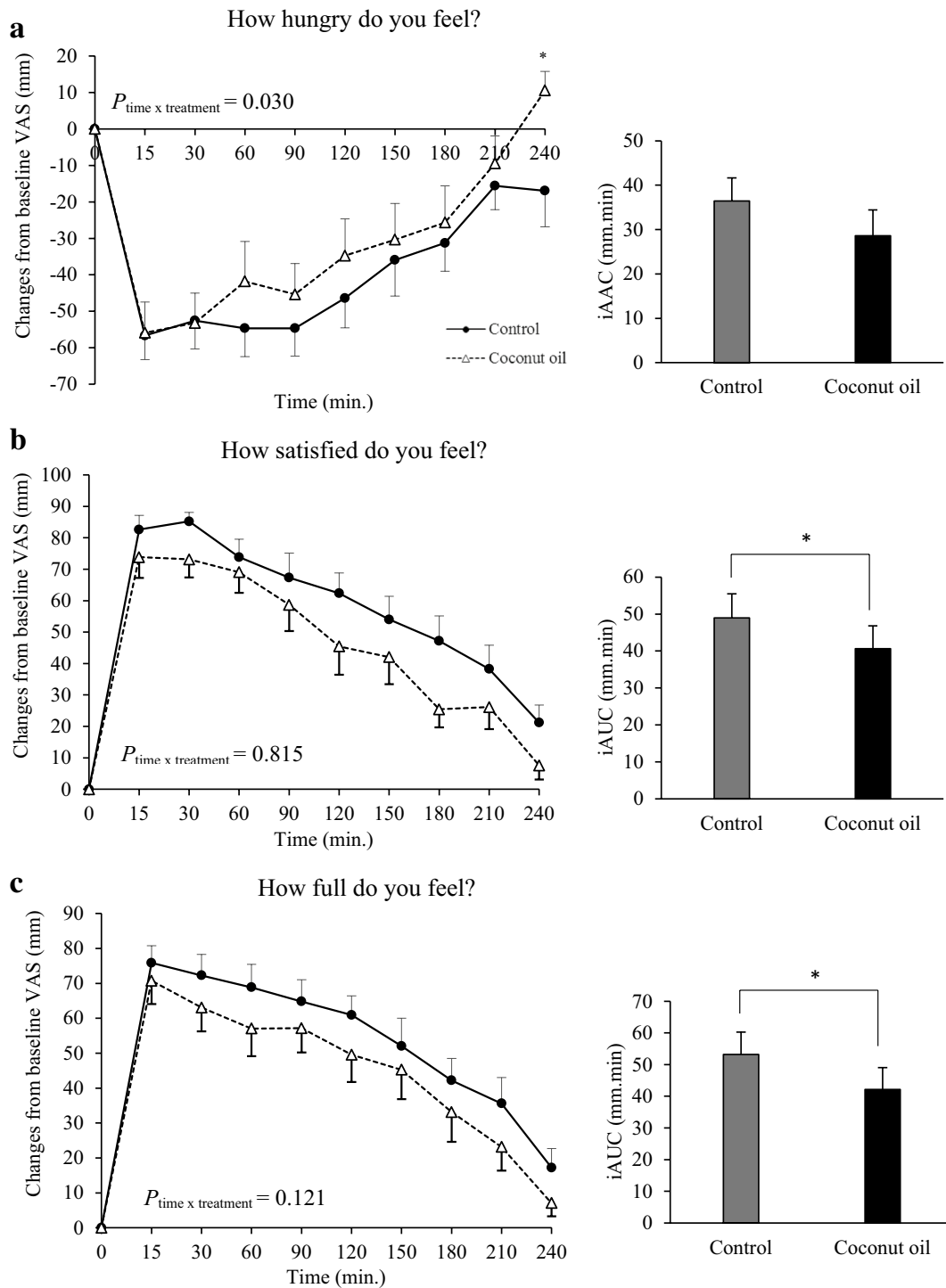
During decades, coconut oil consumption was discouraged due to its high saturated fat content and consequent potential to raise blood cholesterol and promote dyslipidemia [39]. Analyses of VCO fatty acid composition used

**Table 4** Postprandial changes in cardiometabolic risk markers of subjects consuming control or coconut oil (*n* = 15)

Meal induced changes	Coconut oil												P
	Control						Coconut oil						
	Postprandial time (min)			AUC*			Postprandial time (min)			AUC*			
	0	60	120	180	0	60	120	180	0	60	120	180	
Glucose (mmol/L)	4.51 ± 0.15	4.79 ± 0.20	4.53 ± 0.19	4.49 ± 0.09	812.04 ± 29.11	4.42 ± 0.07	4.94 ± 0.25	4.36 ± 0.16	4.43 ± 0.11	808.06 ± 32.91	Time effect	<b>0.010</b>	
Insulin (pmol/L)	84.18 ± 15.10	438.55 ± 53.70	254.83 ± 44.55	169.89 ± 51.96	47726.00 ± 6496.30	62.51 ± 7.26	363.50 ± 38.05	247.09 ± 35.75	175.01 ± 47.42	41958.00 ± 4698.60	Meal effect	0.056	
											Time × meal	0.335	
HOMA-IR	2.63 ± 0.65	13.26 ± 1.82	8.02 ± 1.67	5.18 ± 1.76	1453.79 ± 222.46	1.80 ± 0.23	12.16 ± 1.70	7.17 ± 1.23	5.31 ± 1.62	1326.60 ± 180.61	Time effect	<b>&lt;0.001</b>	
											Meal effect	<b>0.024</b>	
											Time × meal	0.640	
Uric acid (mmol/L)	0.22 ± 0.01	0.23 ± 0.01	0.22 ± 0.01	0.20 ± 0.01	37.26 ± 2.01	0.22 ± 0.01	0.23 ± 0.01	0.23 ± 0.01	0.21 ± 0.01	39.01 ± 1.88	Time effect	<b>&lt;0.001</b>	
											Meal effect	<b>0.047</b>	
											Time × meal	0.584	
Triglycerides (mmol/L)	1.33 ± 0.13	1.43 ± 0.14	1.60 ± 0.17	1.46 ± 0.18	257.97 ± 25.19	1.12 ± 0.11	1.34 ± 0.15	1.60 ± 0.19	1.63 ± 0.21	249.33 ± 30.70	Time effect	<b>0.002</b>	
											Meal effect	0.079	
											Time × meal	0.083	
Total cholesterol (mmol/L)	4.28 ± 0.16	4.52 ± 0.15	4.38 ± 0.17	4.31 ± 0.18	40.77 ± 5.85	4.00 ± 0.17	4.15 ± 0.14	4.17 ± 0.15	4.20 ± 0.15	31.03 ± 4.42	Time effect	<b>&lt;0.001</b>	
											Meal effect	0.851	
											Time × meal	0.954	
HDL-c (mmol/L)	1.26 ± 0.10	1.34 ± 0.09	1.27 ± 0.08	1.26 ± 0.06	232.24 ± 13.67	1.26 ± 0.06	1.29 ± 0.07	1.27 ± 0.07	1.26 ± 0.06	223.12 ± 11.61	Time effect	<b>0.015</b>	
											Meal effect	0.295	
											Time × meal	0.055	
LDL-c (mmol/L)	2.41 ± 0.13	2.53 ± 0.13	2.38 ± 0.15	2.23 ± 0.18	421.45 ± 23.16	2.23 ± 0.15	2.24 ± 0.15	2.16 ± 0.16	2.20 ± 0.16	391.23 ± 29.97	Time effect	0.306	
											Meal effect	0.266	
											Time × meal	0.247	
Triglycerides/HDL-c	1.62 ± 0.16	1.59 ± 0.15	1.62 ± 0.15	1.52 ± 0.08	277.67 ± 22.56	1.43 ± 0.08	1.46 ± 0.09	1.49 ± 0.09	1.51 ± 0.09	260.47 ± 17.80	Time effect	0.210	
											Meal effect	0.114	
											Time × meal	0.127	

Data were expressed as mean ± SEM. Bold letters indicated significant differences (RM-ANOVA in mixed model setting with time as within-subject factor and meal as between-subject factor, *P* < 0.05)

\* There were no significant changes (paired *t* test, *P* > 0.05)



**Fig. 1** Mean  $\pm$  SEM changes from baseline of self-reported hunger (a), fullness (b), and satiety (c) responses obtained from visual analog scales (VAS) in response to extra-virgin olive oil (control) or virgin coconut oil (test) intake ( $n = 15$ ). *iAUC* incremental area under the curve, *iAAC* incremental area above the curve. For the sake

of clarity, error bars are only given for the maximum and minimum values at each time point.  $P_{\text{time} \times \text{treatment}}$  values were obtained from RM-ANOVA in mixed model setting with time as within-subject factor and meal as between-subject factor. \*Significantly different from each other (Paired *t* test,  $P < 0.05$ )

in our study support this affirmation since it provided 94% of saturated fat. Nevertheless, we expected that high proportion of easily oxidized medium-chain fatty acid

(~62% of total saturated fat, 51.6% from lauric acid) could acutely increase postprandial fat oxidation, resulting in increased thermogenesis, less detrimental changes in blood

cardiometabolic risk markers and increased satiety. These effects could contribute to reduce cardiometabolic risks and promote long-term weight loss. In fact, it has long been accepted that MCFA are absorbed and metabolized as rapidly as glucose [5, 40] usually accessed for 120 min postprandially [41]. For this reason, 240 min would be enough to evaluate MCFA metabolism.

The hypothesis that an increase in fat oxidation and thermogenesis would occur after VCO consumption was based on studies showing that acute and long-term consumption of MCFA, but not whole VCO, resulted in increased fat oxidation [11, 23, 42, 43] and energy expenditure [23, 43, 44]. However, few prior studies compared acute effects of VCO on thermogenesis and substrate oxidation rates.

Differences in fatty acid structures, such as chain length, number and position of unsaturation, and stereoisomeric configuration affect fatty acid oxidation rate. Hierarchy seems to exist between saturated and unsaturated fatty acids when consumed individually. Saturated fatty acids oxidation rates decrease with increasing chain length [45, 46] while for unsaturated ones, oxidation decreases with an increase in the number of double bonds [46]. Comparing unsaturated to saturated fatty acids, the former seems to be oxidized more rapidly [47] apart from MCFA, which are oxidized faster than others [48].

In our study, there was no difference between VCO and EVOO fat oxidation rates when incorporated in usual mixed breakfasts. Lauric acid (C12:0) is the predominant MCFA in VCO and it is considered by some authors as a fatty acid with intermediary properties between MCFA and LCFA [49]. We believe that high content of lauric acid in this oil could give it metabolic characteristics more similar to long-chain fatty acid than MCFA present in synthetic oils (rich in C8:0 and C10:0) adopted in the majority of studies [11–13, 17–19, 21, 43, 50]. On the other hand, it was observed increase in carbohydrate oxidation and decrease in fat oxidation in the first hours after both breakfasts consumption. However, after 80 min of breakfast consumption, carbohydrate oxidation started to reduce, and increase in fat oxidation after VCO breakfast seemed to occur (Supplementary Fig. 3). Inclusion of carbohydrate source into breakfasts containing tested oils could change substrate oxidation profile, once carbohydrates are preferably oxidized in order to maintain carbohydrate balance. As consequence, fat oxidation is impaired [51]. Thus, despite the fact that both groups received equal amounts of carbohydrates, we believe that carbohydrate inclusion could have delayed fat oxidation response, masking treatment effects on fat metabolism. We are now testing the role of VCO added to low carbohydrate meal in energy metabolism.

Our study demonstrated no acutely negative effects of MCFA-source VCO in triglycerides, total cholesterol, LDL-c, and triglycerides/HDLc ratio when compared to EVOO

rich in cholesterol-neutral oleic acid. There were great differences in the methodology and results from published randomized clinical trials assessing coconut oil effects, and not synthetic MCFA, in lipid-related cardiometabolic risk factors. The majority of these published studies presented the results of chronic coconut oil consumption and there were divergent results even when coconut oil doses and control treatment were similar in different studies [14, 17]. In turn, there was only one published study comparing the effects of coconut oil consumption to cholesterol-neutral MUFA [52], but the lack of methodological criteria prevent us to discuss its results. Some studies published with synthetic MCFA used control fats with cholesterol increasing [53] or cholesterol decreasing [54], making their results difficult to interpret. MCFA cholesterol-increasing effects were observed only when high-doses of MCFA were consumed (e.g., 70 g/d [18] or 43E% [55]). Thus, MCFA cholesterol-increasing effect could be due to its excessive consumption and does not reflect the impact of reasonable doses of MCFA. In the study herein, we showed that acute consumption of 25 mL of VCO, a reasonable amount to be consumed without producing undesirable collateral effects of high-fat meals [14], was not able to cause detrimental changes on lipid profile. Long-term studies are now needed to confirm our results.

As well as postprandial dyslipidemia, increased level of glucose at postprandial state (also referred as postprandial dysmetabolism) is considered an independent predictor of future cardiovascular events, even in nondiabetic subjects [56]. Different fats addition to mixed meals could distinctly affect carbohydrate digestion and insulin secretion, both impacting postprandial glycaemia [57–59]. Long-chain saturated fat appears to be the worst type of fat to promote glycemic control [60]. Because MCFA are hydrolyzed and absorbed faster than other fats, promote more rapidly gastric emptying, and stimulate less secretion of gut hormones with insulinotropic properties than long-chain fatty acids [61, 62], their potential to reduce postprandial glycaemia have long been neglected. However, Clegg et al. [57] demonstrated that while addition of 22.4 g of long-chain saturated fat (butter) had quite similar glycemic response as oil-free control, addition of the same amount of MCFA significantly reduced almost 40% of total glycemic response, and this reduction was similar to the addition of MUFA (olive oil). Our results are in line with these findings since VCO, high in MCFA, presented the same impact as EVOO on postprandial glycaemia.

MCFA influence on satiety was assessed in few studies [12, 13, 20, 21, 63, 64] and the majority enrolled normal weight subjects [12, 13, 20]. Only one was conducted in normal weight to overweight subjects [21]. Reports from published studies failed to demonstrate MCFA effect on appetite suppression [12, 13, 20, 21, 63, 64], despite some



showed decreased food intake in subsequent meals [12, 13]. In the current study, we investigated VCO effects on subjective appetitive responses in overweight/obese women. Our study consistently showed that VCO addition to mixed breakfast promoted lesser satiating responses than control breakfast containing similar dose of EVOO rich in long-chain fatty acids. Results were characterized by reduced suppression of total hunger, fullness, and satiating responses, and were independent of palatability differences. These findings refute our previous hypothesis that VCO could increase satiety and, for this reason, reduce food intake contributing for weight loss.

Our study presented several strengths. First we properly used EVOO as control which is a natural source of the cholesterol-neutral oleic acid. Furthermore, we assessed the effects of a dietary source of MCFA, and not synthetic MCFA source, on energy metabolism, cardiometabolic risk markers, and satiety. This could improve the clinical implications of our findings, once VCO is the main commercial MCFA source which is largely available for population consumption worldwide. Also, the only difference between test and control breakfasts was the added oil. Finally, our study differed from most previous studies because it enrolled overweight/obese women and used reasonable dose of VCO.

Our study also has limitations. Despite of assessing metabolic rates for 240 min postprandially was considered adequate in several published studies, it is possible that this period of time was not enough to detect differences in fat oxidation between fat meals in presence of carbohydrate. Furthermore, it could not be neglected that 240 min may be not enough to detect changes in some serum lipid fractions in control group.

## Conclusion

Inclusion of reasonable amount (25 mL) of virgin coconut oil into mixed breakfast did not affect energy metabolism, fat oxidation rates, and cardiometabolic risk markers compared to similar control breakfast in generally healthy excess body fat women. Furthermore, this oil suppressed less the hunger, satiety, and fullness responses suggesting that VCO consumption is not effective in improving energy balance. Thus, we recommend caution in prescribing coconut oil as adjuvant in weight loss programs. Long-term studies assessing the role of VCO in obesity control are now necessary to confirm our results.

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## Compliance with ethical standards

**Ethical standards** The study protocol was approved by the Ethics Committee of Universidade Federal de Viçosa (protocol number: 541,836/2014), conducted in accordance with 1964 Declaration of Helsinki and its later amendments and registered at [http://www.ensaiosclinicos.gov.br/\(RBR-8NGPQ9\)](http://www.ensaiosclinicos.gov.br/(RBR-8NGPQ9)). All participants gave written consent after receiving verbal and written information.

**Conflict of interest** The authors declare that they have no conflict of interest.

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