FLÁVIA XAVIER VALENTE

EFEITOS DO CONSUMO DO ÓLEO DE COCO VIRGEM NO CONTROLE DA OBESIDADE E DE MARCADORES CARDIOMETABÓLICOS EM MULHERES

Tese apresentada à Universidade Federal de Viçosa, como parte das exigências do Programa de Pós-Graduação em Ciência da Nutrição, para obtenção do título de *Doctor Scientiae.*

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APROVADA: 23 de fevereiro de 2017.

Júlia Cristina Cardoso Carraro

Solange Silveira Pereira

Josefina Bressan
(Coorientadora)

Rita de Cássia Gonçalves Alfenas
(Coorientadora)

Maria do Carmo Gouveia Peluzio
(Orientadora)

Aos meus pais,

Meus irmãos e meu amor

Com muito carinho e gratidão

Dedico

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“A tarefa não é tanto ver aquilo que ninguém viu, mas pensar o que ninguém ainda pensou sobre aquilo que todo mundo vê.”

(Arthur Schopenhauer)

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LISTA DE ABREVIATURAS E SIGLAS

%E	Percentual de energia
AG	Ácidos graxos
AGCL / LCFA	Ácidos graxos de cadeia longa
AGCM / MCFA	Ácidos Graxos de Cadeia Média
ALT	Alanina aminotransferase
ANOVA	Análise de variância
AP	Fosfatase alcalina
AST	Aspartato aminotransferase
C10:0	Ácido caprílico
C12:0	Ácido láurico
C14:0	Ácido miristico
C16:0	Ácido palmítico
C18:0	Ácidos esteárico
C18:1	Ácido oléico
C18:2	Ácidos linoleico
C8:0	Ácido cáprico
CCK	Colecistoquinina
CEPE	Comitê de ética em pesquisa em seres humanos
CETP	Cholesterol ester transport protein
CHO	Carboidratos
CO_2	Gás carbônico
DEXA / DXA	Densitometria por dupla emissão de raios-X
EER	Estimated energy requirement
EM	Metabolismo energético
EP / SEM	Erro padrão
FAME	Fatty acid methyl esters
$FECO_2$	Fluxo expiratório de gás carbônico
FMI	Fat Mass Index;
g	Gramas
GC	Gas chromatography
GGT	Gama glutamiltransferase

GIP	Glucose-dependent insulinotropic polypeptide
GLP-1	Glucagon-like peptide-1
HDL-c	Lipoproteína de alta densidade de colesterol
HOMA-IR	Homeostatic model assessment of insulin resistence
iAAC	Área incremental acima de curva
iAUC	Área incremental abaixo da curva
IMC / BMI	Índice de massa corporal
IPAQ	Questionário internacional de atividade física
Kcal	Quilocalorias
L	Litros
LCAT	Lecithin cholesterol acyl transferase
LDL-c	Lipoproteína de baixa densidade de colesterol
LIP	Lipídios
min	Minutos
mL	Mililitros
MUFA	Monounsaturated fatty acids
NAF	Nível de Atividade Física
NU / UN	Nitrogênio urinário
O_2	Oxigênio
PTN	Proteínas
PUFA	Polyunsaturated fatty acids
QRNP / NPRQ	Quociente respiratório não proteico
REBEC	Registro brasileiro de ensaios clínicos
REE / TMR / REM	Taxa metabólica de repouso
SFA	Saturated fatty acids
TCL / LCT	Triglicerídeos de cadeia longa
TCM / MCT	Triglicerídeos de cadeia média
TG	Triglicerídeos
TID / DIT	Termogênese induzida pela dieta
TO_{CHO}	Taxa de oxidação de carboidratos
$TO_{CHOjejum}$	Taxa de oxidação de carboidratos de jejum
TO_{L}	Taxa de oxidação de lipídios
TO_{P}	Taxa de oxidação de proteínas

TOS	Taxa de oxidação de substratos
treat	Tratamento
Treat.*time	Interação tratamento e tempo
VAS	Escala visual analógica
VCO_2	Volume de gás carbônico
VLDL	Lipoproteína de muito baixa densidade
VO_2	Volume de oxigênio
X^2	Qui-quadrado

RESUMO

VALENTE, Flávia Xavier, D.Sc., Universidade Federal de Viçosa, fevereiro de 2017. **Efeitos do consumo do óleo de coco virgem no controle da obesidade e de marcadores cardiometabólicos em mulheres.** Orientadora: Maria do Carmo Gouveia Peluzio. Coorientadores: Josefina Bressan, Rita de Cássia Gonçalves Alfenas e Dennys Esper Corrêa Cintra.

O óleo de coco virgem (*Cocos nucifera L.*) tem sido promovido pela mídia comercial como um alimento capaz de auxiliar o tratamento da obesidade devido ao seu alto conteúdo de ácidos graxos de cadeia média (AGCM). Estes ácidos graxos são absorvidos e metabolizados mais rapidamente do que os ácidos graxos de cadeia longa, e, por este motivo são menos armazenados no tecido adiposo. Além disso, os possíveis mecanismos envolvidos na ação dos AGCM no controle da adiposidade corporal podem estar relacionados ao aumento do gasto energético e controle do apetite, favorecendo a perda de gordura corporal e manutenção de um perfil metabólico adequado. Porém, poucos estudos até o momento avaliaram os efeitos do óleo de coco virgem no manejo do peso corporal e nos mecanismos relacionados ao seu efeito. Os objetivos deste estudo foram avaliar o consumo do óleo de coco virgem no controle da obesidade e nos marcadores cardiometabólicos de mulheres obesas. Participaram deste ensaio clínico controlado, duplo-cego e randomizado mulheres obesas (IMC 26 - 35kg/m² e percentual de gordura corporal >30%) com idade entre 20 e 40 anos que foram aleatoriamente alocadas no grupo controle ou no grupo óleo de coco. Foi prescrita uma dieta de restrição calórica (-500 kcal/dia), que incluía 25mL de óleo de soja (controle) ou óleo de coco virgem no café da manhã. A intervenção dietética teve duração de nove semanas consecutivas. No primeiro e último dias da intervenção, foram realizadas medidas antropométricas e de composição corporal, além da avaliação das taxas de metabolismo energético, sensações subjetivas de apetite e dos marcadores de risco cardiometabólicos, em jejum e nas 4 horas pós-prandiais. O consumo alimentar foi avaliado ao final de cada dia de intervenção. Os resultados obtidos no estudo estão apresentados em três artigos, sendo o primeiro com dados da intervenção aguda e o segundo e terceiro com dados da intervenção crônica. **Artigo 1: Acute coconut oil consumption does not affect energy expenditure and cardiometabolic risk markers but positively affects subjective appetitive sensations in obese women** - Quarenta e duas mulheres com média de IMC 30.8 ± 0,5 kg/m² e de percentual de gordura corporal

46,9 ± 0,7% participaram do estudo. O consumo agudo do óleo de coco virgem não afetou o gasto energético e a oxidação de lipídios, mas reduziu a sensação subjetiva de fome, principalmente nas duas primeiras horas pós-prandiais, e a vontade prospectiva de se alimentar. Porém, não houve redução do consumo alimentar após a ingestão do óleo de coco. O óleo de coco virgem também não afetou as concentrações séricas dos marcadores de risco cardiometabólicos e o funcionamento hepático. Os resultados sugerem que o controle do apetite pode ser o mecanismo proeminente pelo qual o óleo de coco está relacionado ao controle da obesidade. Não houve influência do consumo deste óleo no metabolismo energético ou na melhora do perfil de risco cardiometabólico. **Artigo 2: Virgin coconut oil consumption does not improve weight loss and cardiometabolic risk profile of obese women following energy restricted diet** - Trinta e oito mulheres obesas (46,5 ± 0,6 % de gordura corporal) participaram deste estudo. Após nove semanas do consumo do óleo de coco virgem, as concentrações séricas de ácido láurico e mirístico aumentaram. Ainda, houve redução do peso corporal, IMC, das circunferências da cintura, do quadril, do pescoço, da coxa e do braço, do diâmetro abdominal sagital e da gordura corporal em ambos os grupos. Porém, somente no grupo controle foi observado redução do percentual de gordura androide. Houve diferença entre as mudanças das concentrações de HDL-c após o período de intervenção devido à redução das concentrações de HDL-c no grupo controle. Os resultados sugerem que o consumo crônico de óleo de coco virgem não melhora os benefícios causados pela dieta de restrição calórica em relação ao perfil antropométrico e à composição corporal. Além disso, este óleo não altera os marcadores de risco cardiometabólico após nove semanas de consumo. **Artigo 3: Virgin coconut oil chronic consumption does not improve energy metabolism, subjective appetitive sensations and food intake in obese women following energy restricted diet -** Participaram deste estudo trinta e oito mulheres obesas (46,5 ± 0,6 % de gordura corporal). Após o período de intervenção, a oxidação de lipídios aumentou e a oxidação de carboidratos diminuiu no grupo controle. O óleo de coco virgem não aumentou o gasto energético basal, pós-prandial e a termogênese induzida pela dieta. Porém, houve maior sensação subjetiva de fome após o consumo do óleo de coco virgem sem, no entanto, afetar o consumo subsequente de energia e macronutrientes. Estes resultados sugerem que o consumo diário de óleo de coco virgem não melhora o metabolismo energético e o consumo alimentar. Em contrapartida, aumenta a sensação de fome em

mulheres obesas que seguem uma dieta de restrição calórica. De forma geral, os resultados impõem-nos cautela quanto ao uso do óleo de coco virgem no tratamento da obesidade. No momento, nós desencorajamos a prescrição deste óleo como adjuvante no tratamento da obesidade.

ABSTRACT

VALENTE, Flávia Xavier, D.Sc., Universidade Federal de Viçosa, February, 2017. **Effects of virgin coconut oil consumption on obesity control and cardiometabolic risk markers in women.** Adviser: Maria do Carmo Gouveia Peluzio. Co-advisers: Josefina Bressan, Rita de Cássia Gonçalves Alfenas and Dennys Esper Corrêa Cintra.

Virgin coconut oil (*Cocos nucifera* L.) has been promoted by commercial media as an adjuvant in obesity treatment due to its high content of medium-chain fatty acids (MCFA). These fatty acids are absorbed and metabolized faster than long-chain fatty acids, and are therefore less stored in adipose tissue. In addition, the possible mechanisms involving MCFA in the control of body adiposity may be related to increased energy expenditure and appetite control, favoring body fat loss and the cardiometabolic profile. However, few studies have so far evaluated the effects of virgin coconut oil on body weight management and on mechanisms related to its effect. The objectives of this study were to evaluate virgin coconut oil consumption in obesity control and cardiometabolic risk markers of obese women. This is a double-blind, randomized, controlled clinical trial in which obese women (BMI 26 - 35kg/m² and body fat percentage > 30%) aged 20-40 years were randomly allocated in control group or in coconut oil group. Energy restricted diet (-500 kcal/day) was prescribed and included 25mL of either soybean oil (control) or virgin coconut oil at breakfast. Dietary intervention lasted nine consecutive weeks. In the first and last intervention days, anthropometric and body composition measurements were performed, as well as energy metabolism rates, subjective appetitive sensations, and cardiometabolic risk markers in fasted state and for 4 hours postprandially. Food consumption was assessed at the end of each intervention day. Results are presented in three articles: the first with cross-sectional data and the second and third with intervention data. **Article 1: Acute coconut oil consumption does not affect energy expenditure and cardiometabolic risk markers but positively affects subjective appetite sensations in obese women -** Forty-two obese women (BMI of 30.8 ± 0.5 kg / m² and body fat percentage 46, 9 ± 0.7%) participated in the study. The acute consumption of virgin coconut oil did not affect energy expenditure and lipid oxidation, but it reduced the subjective sensation of hunger, especially in the first two hours postprandially, and the prospective consumption sensation. However, there was no reduction in food consumption after virgin coconut oil consumption. Also, virgin coconut oil did not affect cardiometabolic

risk markers and liver function. The results suggest that appetitive control may be the prominent mechanism by which virgin coconut oil is related to weight management. There was no influence of this oil consumption on energy metabolism or on the improvement of cardiometabolic risk profile. **Article 2: Virgin coconut oil consumption does not improve weight loss and cardiometabolic risk profile of obese women following energy restricted diet** - Thirty-eight obese women (46.5 ± 0.6% body fat) participated in this study. After nine weeks of virgin coconut oil consumption, serum concentrations of lauric and myristic acid increased. In addition, there was reduction in body weight, BMI, waist circumference, hip, neck, thigh and arm circumference, sagittal abdominal diameter and body fat in both groups. However, only control group reduced the percentage of android fat. There was a difference between changes in HDL-c concentrations after the intervention period due to the reduction of HDL-c concentrations in the control group. The results suggest that the chronic consumption of virgin coconut oil does not improve the benefits caused by the caloric restriction diet in anthropometric profile and body composition. In addition, this oil does not alter cardiometabolic risk markers after nine weeks of consumption. **Article 3: Virgin coconut oil chronic consumption does not improve energy metabolism, subjective appetitive sensations and food intake in obese women following energy restricted diet -** Thirty-eight obese women (46.5 ± 0.6% of body fat) participated in this study. After the intervention period, lipid oxidation increased and carbohydrate oxidation decreased in the control group. Virgin coconut oil did not increase basal, postprandial energy expenditure and diet-induced thermogenesis. However, there was a greater subjective sensation of hunger after virgin coconut oil consumption without, however, affecting the subsequent energy and macronutrients consumption. These results suggest that daily consumption of virgin coconut oil does not improve energy metabolism and food consumption. On the other hand, it increases hunger sensation of obese women following an energy restricted diet. In general, the results impose caution on virgin coconut oil consumption in obesity treatment. At present, we discourage the prescription of this oil as adjuvant in the weight management.

1 INTRODUÇÃO GERAL

A obesidade é caracterizada como uma das doenças crônicas não transmissíveis mais prevalentes em todo o mundo e atualmente é uma grande preocupação para a saúde pública[1]. Parte dessa preocupação se deve à sua associação com a hipertensão arterial[2], diabetes *mellitus* tipo 2[3], doenças cardiovasculares[4] e alguns tipos de câncer[5].

Apesar da patogênese da obesidade ser multifatorial, o balanço energético positivo parece ser ainda o principal fator associado ao seu desenvolvimento. Como o aumento do consumo de lipídios constitui um fator associado ao desenvolvimento desta doença[6], o estudo do comportamento metabólico dos lipídios da dieta é de grande importância para o entendimento da mesma.

Os lipídios, devido a sua alta densidade energética, são considerados um importante fator causal responsável pelo balanço energético positivo[7,8]. Constituem cerca de 40% da ingestão de energia na dieta humana ocidental e quantitativamente, os triglicerídeos (TG) são os componentes lipídicos de maior importância no fornecimento de energia[9]. Neste contexto, buscam-se novas estratégias alimentares que auxiliem ou previnam o acúmulo de gordura corporal.

Os triglicerídeos de cadeia média (TCM) têm chamado atenção como parte de uma dieta saudável, pelo fato de apresentarem um comportamento metabólico diferenciado em relação aos triglicerídeos de cadeia longa (TCL), podendo resultar em menor acúmulo no tecido adiposo[10]. Porém, os mecanismos relacionados a esta característica ainda não são bem determinados.

O óleo de coco virgem (*Cocos nucifera L.*) tem sido promovido pela mídia comercial como um alimento capaz de promover perda de peso e de gordura corporal devido ao seu alto teor de triglicerídeos de cadeia média (TCM)[11,12]. Porém, estudos que avaliaram a ação dos TCM no peso corporal utilizaram um óleo sintético, que contém em sua composição exclusivamente os ácidos graxos caprílico (C8:0) e cáprico (C10:0)[13–15]. Como o ácido graxo de cadeia média (AGCM) predominante no óleo de coco é o láurico (C12:0; ~50-60%)[16], os efeitos metabólicos observados pela ingestão deste óleo podem ser diferentes dos observados pelo consumo dos TCM sintéticos. Este fato, associado à escassez de estudos com o óleo de coco, sugere que as alegações referentes ao consumo deste óleo sejam extrapolações dos resultados com outros tipos de TCM.

Assim, considerando a rápida aceitação deste tipo de produto na dieta, principalmente pelo público feminino, e a falta de evidências em relação aos efeitos fisiológicos e metabólicos da ingestão do óleo de coco, torna-se de extrema importância verificar a veracidade dos efeitos benéficos resultantes da utilização deste produto, bem como elucidar os mecanismos pelos quais ele agiria na prevenção e reversão da obesidade.

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2 OBJETIVOS

2.1 Objetivo geral

Avaliar o efeito do consumo do óleo de coco virgem no controle da obesidade e de marcadores cardiometabólicos de mulheres obesas.

2.2 Objetivos específicos

✓ Avaliar os efeitos do consumo agudo e crônico de óleo de coco virgem no metabolismo energético, no controle do apetite e na ingestão alimentar de mulheres obesas;

✓ Avaliar os efeitos do consumo agudo e crônico de óleo de coco virgem nos marcadores de risco cardiometabólicos e de função hepática em mulheres obesas;

✓ Avaliar os efeitos do consumo crônico de óleo de coco virgem na antropometria e na composição corporal de mulheres obesas.

3. ARTIGOS CIENTÍFICOS

3.1 Artigo 1: Original research

Acute coconut oil consumption does not affect energy expenditure and cardiometabolic risk markers but positively affects subjective appetitive sensations in obese women

(Artigo apresentado no exame de qualificação, em 09 de dezembro de 2016).

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Abstract

Background: Coconut oil is being considered as healthy oil, which includes its use on obesity treatment. However, the mechanisms attributed to coconut oil on body weight management has been extrapolated from the results obtained in studies conducted with oils that have a medium-chain fatty acids profile very different than the verified in coconut oil. Thus, the aim of this study was to evaluate acute effects of coconut oil intake on energy metabolism, appetite sensations, and cardiometabolic risk markers in obese women.

Methods: Forty-two obese women (26.8 ± 0,9 years; BMI 30.8 ± 0,5 kg/m² and body fat 46.9 ± 0.7%) participated in this one-day intervention, randomized, double-blind, controlled trial. Subjects were randomly allocated into two groups and each received a test drink containing 25mL of soybean oil (control) or virgin coconut oil (VCO). Energy expenditure, substrate oxidation, diet-induced thermogenesis, subjective appetite sensations, cardiometabolic risk markers and liver enzymes were measured at fasting and up to 4 hours postprandially.

Results: Acute coconut oil intake did not increase energy expenditure and fat oxidation but significantly decreased hunger sensation (iAAC: -3,029.7 ± 1,047.8 vs -1,120.8 ± 901.1 mm/4h, *P*=0.027) mainly in the first 2 hours, and prospective food consumption sensation (iAAC: -4,515.2 ± 876.3 vs -2,541.1 ± 879.4 mm/4h; P=0.048). However, there was no influence on subsequent food intake. Coconut oil also did not affect cardiometabolic risk markers and liver function.

Conclusion: The results suggest that postprandial changes in energy metabolism may not be the prominent mechanism by which coconut oil leads to body weight loss. This effect might be related to appetitive control.

Introduction

Obesity is caused by a combination of factors that together result in imbalance in energy balance caused by increased energy intake and/or decreased energy expenditure that lead to adipose tissue[1]enlargement . Thus, new strategies for obesity management has been used including the use of dietary supplements for weight loss[2].

In this context, interest in coconut oil as a weight loss agent has increased. Coconut oil is extracted from *Cocos nucifera L.* under many different processes[3]. Virgin coconut oil (VCO) is extracted from fresh coconut meat by mechanical or natural process with no refining process following extraction[4]. It contains mainly saturated fatty acids (SFA) (~ 93%), and of these around 60% are medium-chain triglycerides (MCT)[5]. The most prevalent fatty acid present is lauric acid (C12:0) (~50–55%), but it also has other medium-chain fatty acids (MCFA) such as caprylic (C8:0) and capric acid (C10:0) in small proportion (~5%). An important amount of long-chain saturated fatty acids (~ 25%) is also present, especially myristic (C14:0; ~20%) and palmitic acid (C16:0; ~5%)[6].

It has been demonstrated that the consumption of synthetic MCT oils has positive effect in reducing body weight and promote health. Daily intake of synthetic MCT oil reduced both body weight[7] and body fat[8–10], increased fat oxidation, energy expenditure, diet-induced thermogenesis (DIT)[11–13], satiety and delaying meal requests, thus reducing food intake[14,15] in humans. However, its role on cardiometabolic markers is still controversial[16–18]and apparently it is beneficial only in the hypertriglyceridemic Chinese population[19,20].

Although coconut oil has been classified as a MCFA source, its fatty acids composition is very different from the synthetic MCT oils[21,22] adopted in the above mentioned studies [7–20]. Synthetic MCT oils contain only caprylic (C8:0) and capric (C10:0) fatty acids, varying from 65-75% of C8:0, 25-35% of C10:0 and do not have lauric acid[23]. Once lauric acid (C12:0) demonstrate intermediate properties between MCFA and long-chain fatty acid (LCFA)[24]. Since coconut oil claims are extrapolations of the results of studies conducted with synthetic MCT oil [25], we questioned if coconut oil would have the same effects as synthetic MCT oils.

Up to now, there are almost no studies evaluating the role of virgin coconut oil as weight loss agent in humans. To the best of our knowledge, there is only one study in wich the impacts of thist oil on body weight management[26], energy metabolism[27] and appetite[28]were assessed. Likewise, the studies that evaluated cardiometabolic markers have important limitations that warrant caution when interpreting the obtained results[29].

Therefore, the aim of this study was to evaluate the acute effects of VCO intake on energy metabolism, appetite sensations, cardiometabolic markers in obese women. Additionally, we evaluated liver function due to its important role in fatty acid metabolism.

Methodology

Subjects

Obese women were recruited by public advertisement to participate in this trial. Eligibility criteria were the following: age ≥18 years, excess of body fat (> 30% of body weight), stable body weight (changes < 5%) and physical activity level during previous 3 months of study beginning, and regular menstrual history. Exclusion criteria were diagnosis of any acute or chronic diseases other than obesity; regular use of prescribed medication other than contraceptives; pregnancy, breastfeeding, smokers or alcohol consumption over 15 g/day.

Prior to participation, all subjects were tested for suitability through a pretest questionnaire. Thus, from seven hundred and fifty two potential volunteers screened, seventy three attended all inclusion criteria (Figure 1). Reasons for not receiving allocated intervention in both groups were: unavailability to follow study protocol (n= 7); acute pathological events (n= 5), withdraw (n= 5), started energy restricted-diet after screening (n= 3); moved to another town (n= 1). The final sample included forty two participants aged 26.8 ± 0,9 years (19 – 41 years); BMI 30.8 ± 0,5 kg/m² and 46.9 ± 0.7% body fat.

Study protocol was approved by the Human Ethics Committee of Federal University of Viçosa (reference: 892.467/2014) (clinical trial number: RBR-7z358j) and conducted in accordance with 1964 Declaration of Helsinki and its later amendments. Written informed consent was obtained from all subjects.

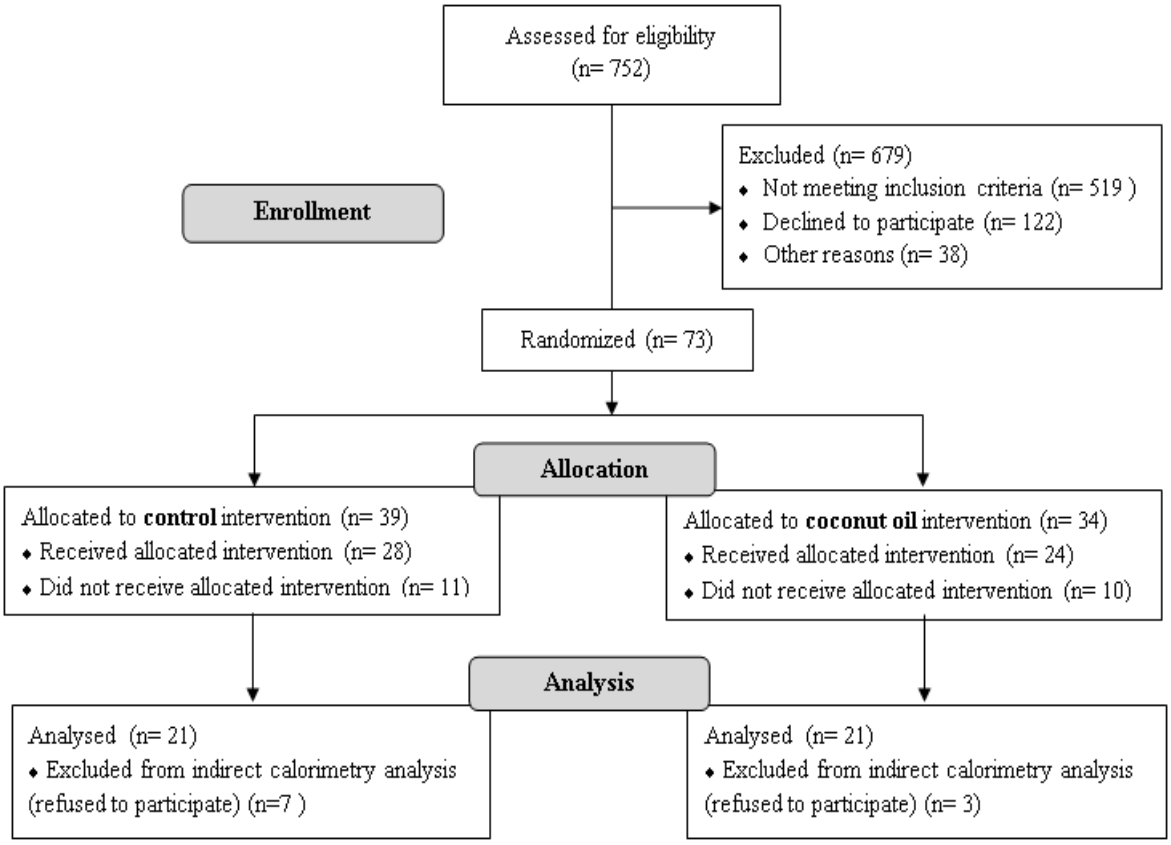

Figure 1 - CONSORT diagram showing the flow of participants through each stage of the trial. CONSORT: Consolidated Standards of Reporting Trials

Study Design

This trial was a single-day intervention which had a randomized, double-blind, controlled, parallel group design, examining the effects of VCO versus soybean oil (control) consumption. Women interested in participating in the trial were submitted to nutritional evaluation to assess inclusion criteria. Those selected were randomly assigned to one of two groups control (n=21) or coconut oil (n=21) using the block randomization technique[30].

Subjects were asked to refrain from alcohol and caffeine containing drinks, and heavy physical activities 48 hours prior to test day. A list of foods and beverages containing caffeine was given to volunteers. Also, they could not be on menstrual period on test day. Carbohydrate-rich standard dinner was provided to be consumed on the evening before assessments (see Test meal section). Subjects were instructed to eat dinner at 9 p.m. after emptying their bladder and to collect all urine after dinner until test meal consumption. This was considered 12 hours urine sample.

Participants were asked to stay at Energy Expenditure and Body Composition Laboratory from 7 a.m. to 1 p.m. They arrived at the laboratory after 10 hours fasting, and with the minimal physical effort as possible. After anthropometric and body composition were assessed, participants rested for 15 minutes before energy expenditure measurement. Visual analogic scale (VAS) for subjective appetitive evaluation was applied and test meal was then offered according to designated group. Immediately after test meal consumption VAS was again applied and thirty minutes later energy expenditure and appetitive sensations were assessed in regular intervals over the next 4 hours (Figure 2). Water (200ml) at room temperature was offered at each interval and the urine of the entire postprandial period was collected.

After completing the study protocol, a standard lunch composed of sandwich and fruit juice was offered (325kcal; carbohydrate: 61.8%E, protein: 22.4%E, fat: 28%E) and subjects were instructed to record all foods and beverages consumed for rest of the day in a food diary.

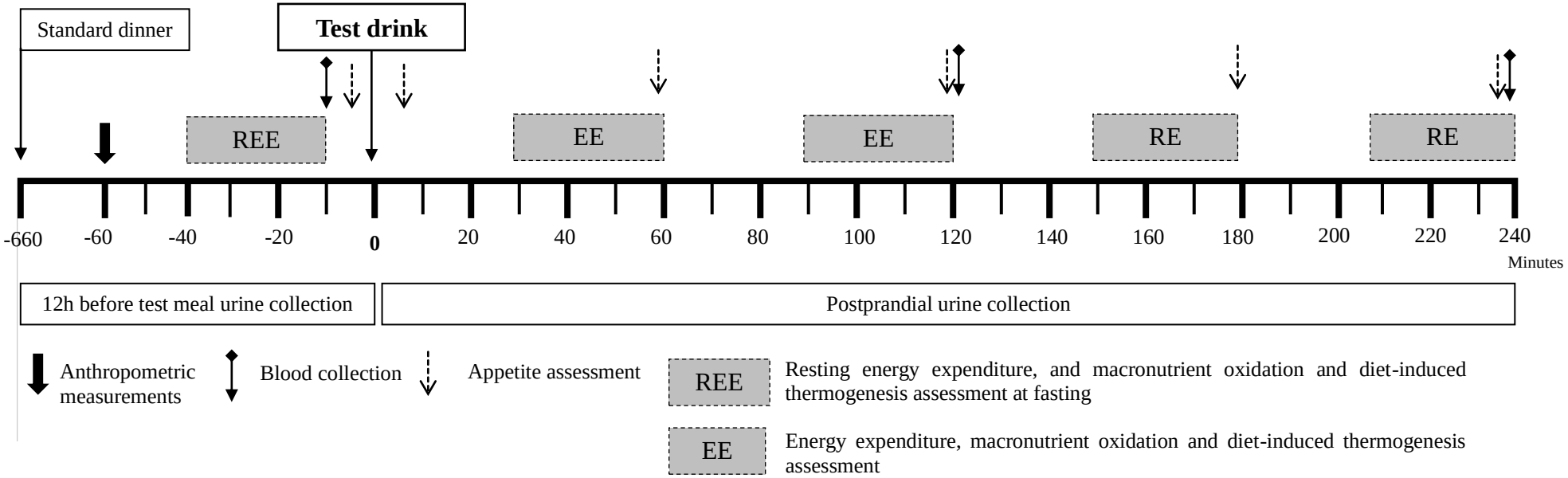

Figure 2 - Study protocol

Meals

Standard carbohydrate-rich dinner consisting of 109g instant plain noodles (Nissin®), 10g parmesan cheese, and 200mL of orange juice (600 kcal, carbohydrate: 62E%, protein: 8.5E%, fat: 29.4E%) was consumed the night before de test and to reduce the influence of differing nutrient consumed the night before fasting measurements.

Test drinks were given for breakfast and consisted of high-fat isocaloric milk shakes which contained the same amount and type of ingredients, except for the type of oil (Table 1). It contained 20g of powdered skimmed milk, 5g of grape flavoring powder, 15 cyclamate/saccharin-sweetener drops and 25 mL of either soybean or VCO, and water in sufficient amount to result in 300 mL. Soybean oil (Corcovado, Archer Daniels Midland Company, Brazil) and VCO (Copra, Copra Indústria Alimentícia Ltda, Brazil) were purchased from local market. Each oil was the only source of fat of the beverages and fatty acid composition was the only difference between them. Beverages were prepared immediately prior to consumption and were consumed within 10 minutes. The amount of oil added was based on previous studies, considered safe and sufficient enough to see the possible effect in case they existed[26,31].

The fatty acids composition of test oils was determined by gas chromatography (GC) after methylation by Hartam and Lago[32] methodology. CG was performed using CG-17A Shimadzu/Class model®, with capillary column DB-5 (30 m x 0.25 µm id, 0.25 mm film thickness, J&W Scientific, USA) and a flame ionization detector. The programming of the analysis presented an initial temperature of 100°C, being isothermic for 5 minutes, and a posterior heating of 4°C per minute up to 220°C, maintaining this temperature for 30 minutes. The temperature of the vaporizer was 200°C and the temperature of the detector was 240°C. The carrier gas used was nitrogen at 43.2 cm/second. The split of the sample in the injector was 1/50 and 1 µL of the sample was injected. 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). Percentage of individual FAME was made in relation to total area of the chromatogram (Table 1).

Table 1 - Test drinks energy and nutrient content.

	Control	Coconut Oil
Energy content (kcal)	298.6	298.6
Carbohydrate (g / %E)	10.0 / 13.4	10.0 / 13.4
Protein (g / %E)	6.9 /9.2	6.9 /9.2
Total fat (g / %E)	25.0 / 75.2	25.0 / 75.2
Fatty acids (%)		
C8:0		2.5 ± 0.4
C10:0		5.5 ± 0.2
C12:0	0.3 ± 0.1	55.0 ± 0.9
C14:0	0.2 ± 0.1	19.5 ± 0.1
C16:0	10.8 ± 0.1	8.5 ± 0.2
C18:0	3.7 ± 0.1	3.1 ± 0.1
C18:1 ω9	31.7 ± 0.3	-
C18:2 ω6	52.3 ± 0.4	5.3 ± 0.2
Total SFA	15.0 ± 0.2	94.1 ± 0.2
Total PUFA	53.0 ± 0.4	5.9 ± 0.2
Total MUFA	32.0 ± 0.3	-

C8:0: caprilic acid; C10:0: capric acid; C12:0: lauric acid; C14:0: myristic acid; C16:0: palmitic acid; C18:0: stearic acid; C18:1ω9: oleic acid; C18:2 ω6: linoleic acid; SFA: saturated fatty acids; PUFA: polyunsaturated fatty acids; MUFA: monounsaturated fatty acids

Anthropometry and body composition assessment

Body weight were measured on a digital platform scale with a resolution of 0.5 kg (Toledo®, Model 2096PP/2, São Paulo, Brazil), while subjects were barefoot and wearing lightweight clothing. Height was measured to the nearest 0.1 cm using a wall-mounted stadiometer (Wiso, Chapecó, SC, Brazil). BMI was calculated dividing body (kg) by height (m) squared. Waist circumference was assessed in the midpoint between the last rib and iliac crest. Anthropometric measurements were assessed by a single investigator.

Body composition (lean mass and total body fat) was evaluated by Dual energy X-ray absorptiometry scan (DXA) (model Prodigy Advance, GE Healthcare Inc., Waukesha, WI) according to manufacturer's instructions. Fat mass index were calculated by the ratio between the weight of fat (kg) and square of

height (m), and was used as indicative of body fat adequacy. Results above 9 kg/m^2 were considered excess of body fat[33].

Metabolic rates measurement and calculations

Fasting (REE) and postprandial energy expenditure (EE), carbohydrate and lipid oxidation rates were measured by indirect calorimetry using an open-circuit ventilated canopy measurement system (Carefusion Vmax ® Series, California, EUA). The flow meter and flow sensor calibration of the bidirectional digital turbine flowmeter were performed daily using a 3 L syringe and analyzers were calibrated prior to every run with gases of known concentration as recommended by manufacturer (gas #1: 26% O_2 nitrogen balance; gas #2: 4% CO_2 and 16% O_2 nitrogen balance; gas #3 ambient air).

Subjects laid supine, with their head elevated 30 degrees, for a mandatory 20 minutes rest period before REE test. They were in a quiet room with stable temperature (22-24ºC) and were not allowed to sleep during measurements. A transparent ventilated hood was positioned over the subject's head and expired gases were continuously collected. REE was measured after 11 hours fast, at 8 a.m. to 9 a.m. Postprandial measurements were made every 30 minutes for each hour during 4h after test meal[34]. Flow rate were regularly adjusted to maintain a constant $FECO_2$ through all the time. During protocol intervals, subjects remained awake but inactive, allowed only to perform quiet activities.

Oxygen and carbon dioxide volumes (VO_2 and VCO_2, respectively) readings were recorded every minute. The first 10 minutes (adaptation phase) and individual outlier values of those volumes were excluded. Means of VO_2 (L/min) and VCO_2 (L/min) from remaining data were used for calculations[35].

Twelve hours before test drink intake and postprandial (4 hours) urine were collected for estimation of total urinary nitrogen excretion as a marker of protein oxidation. Total volumes were noted and an aliquot was acidified with Timerozal® to prevent microorganism growth. Urea content was analyzed in the urine by colorimetric enzymatic kit (Bioclin®, Minas Gerais, Brazil) in automatic biochemical analyzer BS-200 (Mindray Medical International Ldt., Shenzen, China). Urinary nitrogen (UN) was than calculated[36]. The result was divided by hours of urine collection and expressed as g/min.

REE, EE (kcal/min)[37] and substrate oxidation rates (carbohydrate, protein and fat oxidation rates)[36] were calculated using VO_2, VCO_2 and UN of each period of time. Values of Non-Protein Respiratory Quotient (NPRQ) were also calculated[38]. Diet-induced thermogenesis (DIT) was assessed[34] and expressed as percentage of test meal energy content. Changes between fasting and fed states of carbohydrate and lipid oxidation were calculated by subtracting the total postprandial value over 4 h from fasting value multiplied by the same postprandial time[35].

Subjective appetite sensations

Visual analogue scale (VAS) of 100 mm were used to assess subjective appetite sensations (Flint et al. 2000) before, immediately after, and hourly for 4 h following standardized test meal (Figure 2).

These scales included words anchored at the left and right ends with opposing statements, expressing the most negative and positive ratings for each question. Subjects were instructed to rate appetite dimensions by indicating on the scale how they felt at the moment they completed the questions: Hunger ("How hungry do you feel?"), fullness ("How full do you feel?"), satiety ("How satisfied do you feel?"), and prospective consumption (How much do you think you can eat?). They could not refer to their previous ratings when answering questions.

Results were expressed as changes from the baseline value over postprandial period. Also incremental area under the curve (iAUC) was determined for fullness and satiety subjective sensations, and the incremental area above the curve (iAAC) was determined for hunger and prospective food consumption by trapezoidal method[39]. VAS were also used to rate the palatability of high-fat meals by following questions: visual appeal, smell, taste, aftertaste, and palatability[40].

In order to evaluate if coconut oil was able to influence subsequent food consumption like synthetic MCT oil[29] subjects were instructed to fill out a food diary from the moment they left laboratory until the next morning. Food record was reviewed individually by a dietitian with subjects to check for errors or omissions. Analysis of energy and macronutrients consumption was assessed using Dietpro software (Dietpro 5.2i, Agromidia Software Sistemas, Brazil) based on national and international composition food tables[41,42].

Cardiometabolic markers and liver enzymes

Antecubital blood samples were collected in the fasting state (12 h) and at 1 and 2 hours postprandially. Serum samples were separated from whole blood by centrifugation (3,500 rpm, 4°C, 15 min) and immediately frozen at -80°C until analyses. Glucose, uric acid, total cholesterol, high-density lipoprotein cholesterol (HDL-c), low-density lipoprotein cholesterol (LDL-c), triglycerides (TG), γ-glutamyltransferase (GGT), aspartate amino transferase (AST), and alanine amino transferase (ALT), alkaline phosphatase (AP) were quantified by standard colorimetric kits (Bioclin®, Minas Gerais, Brazil) by automatic biochemical analyzer BS-200 (Mindray Medical International Ldt., Shenzen, China).

Very low-density lipoprotein (VLDL) was estimated by Friedewald et al[43]. Insulin was assessed by chemiluminescence method (Elecsys-Modular E-170, Roche Diagnostics Systems). Insulin resistance was estimated by the Index of Homeostasis Model Assessment of Insulin Resistance (HOMA-IR) using Matthews et al[44] equation. Insulin resistence was considered when HOMA-IR index were ≥ 2.71[45]. Incremental area above the curve (iAAC) was calculated to evaluate postprandial glucose response. Incremental area under the curves (iAUC) were calculated for the others cardiometabolic marker and liver enzymes[39].

Complete biochemical data at 4 hours postprandially is not available due to technical difficulties during the collection procedure in one subject of control group.

Statistical Analysis

Statistical analyses were carried out with SPSS 20 for Windows (SPSS, Inc., Chicago, IL, USA). Descriptive statistics are presented as mean ± standard error unless otherwise indicated. Data normality and homogeneity of variance were assessed by Shapiro-Wilk and Levene tests, respectively. Treatment effects on energy metabolism and appetite sensation variables were tested by repeated-measures ANOVA in a mixed model setting, with time as within-subject factor and treatment as between-subject factor. Student *t* test or Mann-Whitney U signed-rank test were used to assess differences between groups on total postprandial response of cardiometabolic risk markers and dietary intake. Student *t* test or Mann-Whitney U signed-rank test were also used along with Bonferroni´s correction to determine differences in REE, carbohydrate and fat oxidation, changes in appetite scores, and cardio-metabolic and liver enzymes at each time-point. For this multiple post hoc comparisons, Bonferroni

correction was applied to control for type I error. Statistical significance criterion adopted was $P < 0.050$.

Power analysis was performed according to It indicated that a sample of 21 subjects per group provided 80% power to detect a significant difference between hunger response to different treatments[46].

Results

Subjects' characteristics at baseline (fasting condition) did not differ between groups. Test drinks were well tolerated by participants. Despite the fact that both test drinks presented high palatability scores, control drink was considered more tasty than VCO drink (P=0.024) (Table 2).

Table 2 - Baseline characteristics of study subjects.

Characteristic	Control (n=21)	Coconut Oil (n=21)
Age (years)	26.9 ± 1.4	26.9 ± 1.3
BMI (kg/m^2)	30.9 ± 0.7	30.9 ± 0.7
Waist circumference (cm)	98.9 ± 1.5	97.1 ± 1.8
Waist to hip ratio	0.86 ± 0.1	0.86 ± 0.01
Fat mass (kg)	39.3 ± 1.6	37.0 ± 1.7
Body fat percentage (%)	47.4 ± 0.8	46.3 ± 1.0
FMI (kg/m^2)	14.7 ± 0.5	14.3 ± 0.5
Fat-free mass (kg)	43.2 ± 1.1	42.5 ± 1.4
REE (kcal/day)	1356.6 ±25.9	1374.8 ± 37.3
REE / fat-free mass (kcal/day)	31.7 ± 0.6	32.6 ± 0.6
Visual appeal (mm)	93 ± 4	87 ± 4
Smell (mm)	94 ± 2	85 ± 4
Taste (mm)	84 ± 2	77 ± 5*
Aftertaste (mm)	54 ± 2	41 ± 6
Palatability (mm)	82 ± 4	74 ± 5

BMI: Body Mass Index; FMI: Fat Mass Index; REE: Resting Energy Expenditure. Data are presented as mean ± SEM. * P<0.05 Student t test or U-Mann Whitney test as appropriate.

Metabolic rates

Mean REE were not different between groups (0.95 ± 0.02 kcal/min; P= 0.689) and energy expenditure values at 4 hours returned to baseline. There was no interaction effect of treatment*time for all metabolic rates assessed in this study (Figure 3). Energy expenditure increased significantly after consumption of both test drinks (P_{time}<0.001), nevertheless without difference between groups ($P_{treat.}$= 0.801) (Figure 3A). Mean NPRQ at baseline was not different between control and coconut oil group (0.89 ± 0.01 vs. 0.88 ± 0.001, respectively). NPRQ decreased along 4 hours (P_{time} <0.001), with no differences between groups ($P_{treat.}$= 0,515 and $P_{treat.*time}$ = 0.827). Both groups reached values of 0.79 ± 0.01 at 4 hours.

Carbohydrate oxidation was reduced after test drink consumption (P_{time} < 0,01) and fat oxidation increased (P_{time} < 0,01), mainly at 150 minutes in both groups (Figures 3C and 3E). Absence of differences between groups was maintained when DIT and changes on total postprandial responses of carbohydrate and lipid oxidation were assessed (Figures 3B, 3D and 3F, respectively).

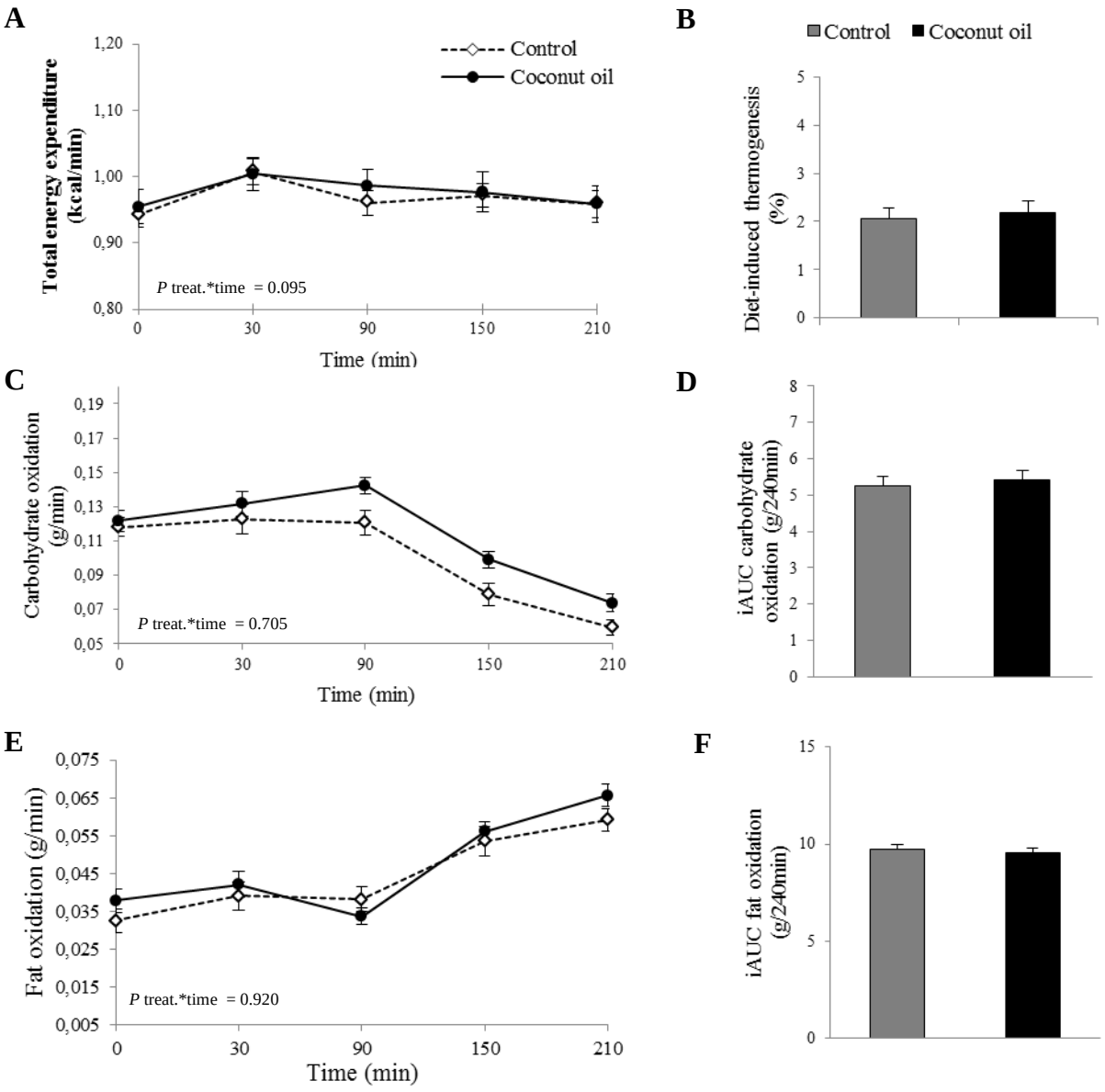

Figure 3 - Means ± SEM changes in total energy expenditure (A), diet induced thermogenesis expressed as percentage of test meal energy content (B), carbohydrate oxidation (C), iAUC carbohydrate oxidation (D), fat oxidation (E), and iAUC fat oxidation (F).

iAUC: incremental area under the curve. Changes between fasting and postprandial state of carbohydrate and lipid oxidation were calculated by subtracting postprandial values over 4 h - (fasting value per h x 4) and iAUC were calculated by trapezoidal method. For (A), (B) and (C) variables, repeated-measures ANOVA in a mixed model with time as within-subject factor and treatment as between-subject factor were performed. For (B), (D) and (F) variables, *t* test or U Mann-Whitney test were conducted. There was only effect of time for all variables analyzed. Test drinks contained 25mL of virgin coconut oil (coconut oil) or soybean oil (control).

Subjective appetite sensations and subsequent intake

All subjects consumed the test drinks within 5 minutes. There was no interaction of treatment*time for any of the appetite dimensions analyzed (Figure 4). As expected, all subjective sensations changed over time (P_{time}<0.001) – hungry and prospective food consumption decreased (Figure 4A and 4G, respectively), while fullness and satiety increased over 4 hours (Figure 4E and 4F, respectively). There was difference in hunger sensation between groups (P_{treat} = 0.041). There was a delay in hunger sensation in response to VCO drink compared to control drink, and subjects felt less hungry in the first 2 hours after its intake (Figure 4A).

Concerning the overall response for appetitive sensations, hunger and prospective food consumption VCO iAAC had greater decrease than control (Figure 4B and 4H, respectively), indicating that subjects that consumed this test drink felt less motivation to eat in the 4 hours postprandial interval than control. Nonetheless, there was no difference for fullness and satiety sensations (P_{iAUC} >0.05).

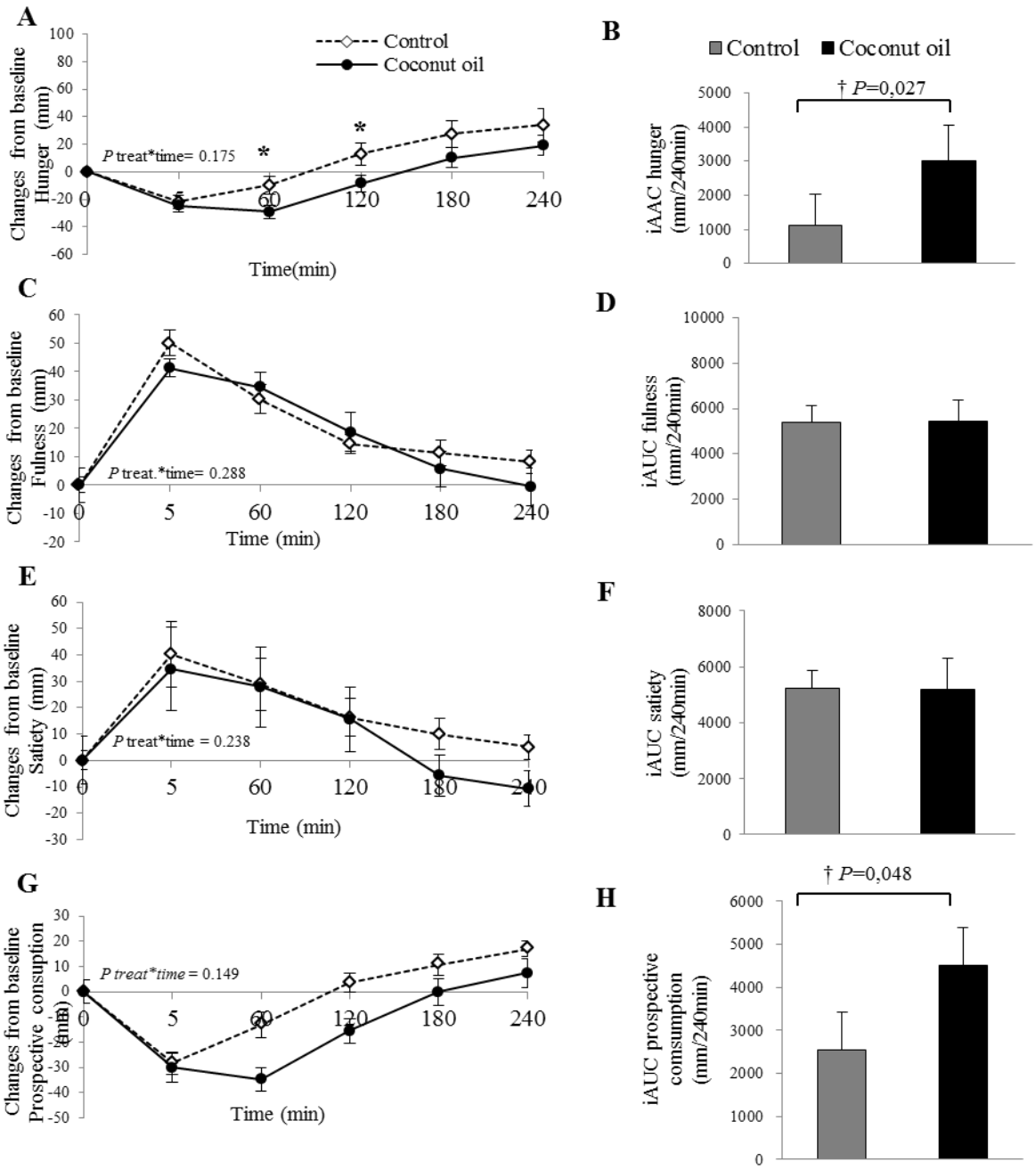

Figure 4 – Mean ± SEM of incremental hunger (A), fullness (C), satiety (E) and prospective food consumption (G) subjective sensations scores and iAAC of hunger (B) and prospective consumption (H) and iAUC of fullness (D), and satiety sensations (F) after teste meal consumption. iAUC: incremental area under the curve and iAAC incremental area above the curve. For (A), (C), (E) and (G) parameters, repeated-measures ANOVA in a mixed model with time as within-subject factor and treatment as between-subject factor were performed. There was no treatment*group interaction for all variables analyzed ($P>0.050$). *t* test or *U*-Mann Whitney test as appropriate with Bonferroni´s correction was performed to verify differences between groups in each point of time (*$P<0.010$). For (B), (D), (F) and (H) parameters, *t* test or *U*-Mann Whitney test as appropriate was conducted (†$P<0.050$). Test drinks contained 25mL of virgin coconut oil (coconut oil) or soybean oil (control).

In accordance with these results, assessment of food diaries from subsequent meals showed no differences between groups on energy and macronutrient intake (Table 3)

Table 3 - Mean ± SEM of energy and macronutrient intake after test drink consumption.

	Control (n = 17)	Coconut oil (n = 14)	*P**
Energy (kcal)	764.9 ± 124.0	738.6 ± 93.1	0.984
Carbohydrate (g)	99.3 ± 14.6	98.2 ± 12.4	0.957
%E	53.4 ± 15.8	55.2 ± 13.2	0.681
Protein (g)	19.8 ± 4.7	23.8 ± 3.0	0.981
%E	14.8 ± 0.7	15.5 ± 1.2	0.957
Lipids (g)	24.6 ± 7.9	21.3 ± 4.4	0.570
%E	20.1 ± 3.6	15.4 ± 1.3	0.486

%E: percentages of total energy intake. * Mann-Whitney U signed-rank test as appropriate. Bold type P values. Test drinks contained 25mL of virgin coconut oil (coconut oil) or soybean oil (control).

Cardiometabolic markers and liver enzymes

Concentrations of all cardiometabolic markers and hepatic enzymes at each time-point are presented on Table 4.

Two women from control group and three from coconut oil group presented insulin resistance (HOMA-IR >2.71). There was no difference between groups on insulin, glucose concentration and HOMA-IR at baseline (P>0.05).

There was significant treatment*time interaction for uric acid, which means that in different time-points acid uric concentration differed between coconut oil and control group. In fact, uric acid concentration only increased after 4 hours, remaining constant until 2 hours. On the other hand uric acid concentration increased in all time-points for control group. There was no interaction effect for any other cardiometabolic risk marker. No differences between groups were observed in postprandial response of any cardiometabolic marker analyzed (Table 4).

Table 4 - Fasting and postprandial concentrations of cardiometabolic markers and liver enzymes.

	Control (*n* = 21)				Coconut oil (*n* = 21)				*P**	
	Time			Total	Time			Total		
	0	2 hours	4 hours**	postprandial response	0	2 hours	4 hours	postprandial response		
Glucose (mg/dL)	83.4 ± 1.9	83.4 ± 1.9	81.7 ± 1.0	444.4 ± 105.7	86.0 ± 2.2	83.1 ± 2.7	80.9 ± 1.9	655.4 ± 110.2	Time effect Treat. effect Time x treat	**0.005** 0.784 0.161
Insulin (µU/L)	10.0 ± 1.5	11.7 ± 1.1	8.1 ± 0.8	319.7 ± 79.2	9.9 ± 2.2	9.7 ± 1.4	7.0 ± 1.3	263.7 ± 103.6	Time effect Treat. effect Time x treat	**<0.001** 0.631 0.912
Uric acid (mg/dL)	3.8 ± 0.3	3.9 ± 0.2	4.1 ± 0.3	40.5 ± 6.9	3.6 ± 0.3	3.6 ± 0.3	3.7 ± 0.3	22.3 ± 5.7	Time effect Treat. effect Time x treat	**0.002** 0.213 **0.036**
Total cholesterol (mg/dL)	162 ± 8.3	168.2 ± 7.6	172.6 ± 7.7	1,276.3 ± 220.5	165.1 ± 7.3	179.3 ± 7.2	176.1 ± 7.0	1,334.0 ± 170.1	Time effect Treat effect Time x treat	**<0.001** 0.700 0.993
HDL-c (mg/dL)	46.5 ± 2.5	46.7 ± 2.7	44.9 ± 2.0	211.4 ± 67.2	45.1 ± 2.4	4 6.3 ± 2.6	46.9 ± 2.6	255.0 ± 52.8	Time effect Treat. effect Time x treat	0.095 0.645 0.108
LDL-c (mg/dL)	95.5 ± 6.7	97.6 ± 6.4	99.8 ± 6.7	479.4 ± 81.1	99.7 ± 6.1	102.7 ± 6.1	105.9 ± 5.9	718.9 ± 105.1	Time effect Treat. effect Time x treat	**<0.001** 0.509 0.536

Data expressed as mean ± SEM. n = number of subjects. HOMA-IR: Homeostatic Model Assessment; HDL-c: High density lipoprotein cholesterol. ** n =20. Total postprandial response was calculate as incremental area above the curve (iAAC) for glucose and as incremental area under the curve (iAUC) for all the remaining cardiometabolic markers. There were no differences between groups for total postprandial response (Student *t* test or Mann-Whitney U signed-rank test, $P > 0.050$). *RM- ANOVA in a mixed model setting, with time as within-subject factor and treatment as between-subject factor. Bold type P values indicate significantly differences ($P < 0.050$). Test drinks contained 25mL of virgin coconut oil (coconut oil) or soybean oil (control).

Table 4 – Fasting and postprandial concentrations of cardio-metabolic markers and liver enzymes (Continued)

	Control (n = 21)				Coconut oil (n = 21)				P*	
	Time			Total postprandial response	Time			Total postprandial response		
	0	2 hours	4 hours**		0	2 hours	4 hours			
VLDL (mg/dL)	18.0 ± 1.7	23.7 ± 2.0	24.5 ± 2.3	900.0 ± 113.9	18.9 ± 1.4	22.8 ± 1.6	23.0 ± 1.7	718.2 ± 105.1	Time effect	**<0.001**
									Treat. effect	0.791
									Time x treat	0.174
TG (mg/dL)	89.9 ± 9.8	118.7 ± 11.4	122.5 ± 13.1	5,080.1 ± 734.1	94.6 ± 9.1	113.8 ± 10.1	115.0 ± 10.9	650.2 ± 90.6	Time effect	**<0.001**
									Treat. effect	0.791
									Time x treat	0.174
GGT (U/L)	24.8 ± 4.0	24.4 ± 3.9	25.7 ± 4.2	94.5 ± 32.3	24.6 ± 2.8	24.4 ± 3.0	25.0 ± 2.9	3,456.3 ± 426.0	Time effect	0.207
									Treat. effect	0.885
									Time x treat	0.818
AST (U/L)	34.1 ± 2.0	34.4 ± 1.6	35.7 ± 1.3	670.1 ± 140.3	34.6 ± 2.5	34.1 ± 2.4	33.9 ± 2.5	363.5 ± 120.3	Time effect	0.287
									Treat. effect	0.962
									Time x treat	0.620
ALT (U/L)	20.6 ± 2.7	21.9 ± 2.6	21.9 ± 2.7	267.9 ± 72.7	18.6 ± 2.4	19.6 ± 2.5	19.4 ± 2.7	309.9 ± 57.5	Time effect	**0.026**
									Treat. effect	0.413
									Time x treat	0.888
AP (U/L)	65.8 ± 4.6	68.1 ± 4.2	69.3 ± 4.6	601.6± 104.4	61.7 ± 5.8	66.2 ± 6.0	65.2 ± 6.6	716.8 ± 137.8	Time effect	**0.016**
									Treat. effect	0.657
									Time x treat	0.439

LDL-C: Low density lipoprotein cholesterol; VLDL: very low density lipoprotein; TG: Triglycerides; GGT: gamma-glutamyltransferase; AST: Aspartate aminotransferase; ALT: alanine aminotransferase; AP: Alkaline phosphatase. ** n =20. Total postprandial response were calculate as incremental area above the curve (iAAC) for glucose and as incremental area under the curve (iAUC) for all the remaining cardiometabolic markers. There were no differences between groups for total postprandial response (Student *t* test or Mann-Whitney U signed-rank test, $P > 0.050$). *RM- ANOVA in a mixed model setting, with time as within-subject factor and treatment as between-subject factor. Bold type P values indicate significantly differences ($P < 0.050$). Test drinks contained 25mL of virgin coconut oil (coconut oil) or soybean oil (control).

Discussion

In this study, we investigated coconut oil acute effects on obese women metabolism. Results demonstrate VCO did not increase energy expenditure and fat oxidation, but significantly delayed hunger and prospective consumption sensations. Also, there were no improvements in cardiometabolic risk markers or impairment of liver enzymes after coconut oil consumption.

It has been highly demonstrated that different types of triglycerides are metabolized differently by human body due to its fatty acid composition[47,48] and within fatty acid classes, different members may have different actions and effects[49]. Consequently the shorter chain length of MCFA presents distinct metabolism from LCFA as they are absorbed faster and transported directly to liver and are not transported by chylomicron[50].

In short term studies MCFA consumption increased fat oxidation[11–13], energy expenditure[11,12] and diet-induced thermogenesis (DIT)[11,51] compared to LCT. Indeed, the first characteristic of MCT synthetic oil described by all authors is that it is rapidly oxidized[47,52].

The majority of studies[11–13,53] that observed significant effects of MCFA on energy expenditure and fat oxidation used the synthetic MCT oil containing only caprilic (C8:0) and capric (C10:0) fatty acids in its composition (47 – 74% C8:0 and ~30% C10:0). On the other hand, coconut oil has small amounts of C8:0 and C10:0 (~5%) and high amounts of C12:0 (~ 55%) and myristic acid (C14:0; ~20%)[21].

Despite the fact that lauric acid (C12:0) has been classified as MCFA by some[24,52,54,55], but not all authors[21,22,56,57], this fatty acid shows intermediate properties between MCFA and long-chain fatty acid (LCFA)[24]. It was demonstrated that ~ 50% of lauric acid is absorbed by chylomicron[58,59], which could lead to different metabolic fate compared to other MCFA (e.g. caprilic and capric acids), like less fat oxidation and more fat deposit in adipose tissue. This is a possible and reasonable explanation by which there were no differences in energy metabolism and metabolic rates in coconut oil versus the LCT source soybean oil in our study.

Flatt et al[60] tested the intake of 42%E as MCT margarines compared to LCT margarines. Only one study testing coconut effects on metabolic rates was identified. White et al[27] evaluated the energy expenditure response to a mixture of coconut oil and butter as MCFA sources (17.7% C12:0; 13.3% C14:0 and 25.4% C16:0) in nonobese

women for seven and fourteen days. They did not observe differences in total energy expenditure, DIT, fat and carbohydrate oxidation. However, after seven and 14 days consuming coconut oil-butter mixture REE increased. It suggests that some effects may be seen after longer period of intake rather than the consumption of a single dose of coconut oil also assessed in our study. Its thermogenic effect should be further explored in long-term studies.

The fact that the results obtained by us and other authors showed no increase in energy expenditure, DIT or fat oxidation rate after coconut oil intake reinforce the theory that this oil does not behave like MCT oil. Also, to the best of our knowledge, this was the first study to assess coconut oil effects on obese women. The majority of MCT oil studies involved nonobese men[11,56,60] or women[13,27], which in general have higher body fat free mass resulting in higher substrates oxidation and energy expenditure[61]. The lack of studies with obese subjects consuming coconut oil suggests that caution is necessary for recommendation of its consumption for obesity treatment.

There was a difference in taste scores between dietary treatments. Although both drinks were rated with high scores for taste, coconut oil was considered less pleasant. VCO does not go through processing of deodorization as refined coconut oil does[3] and thus, a residual taste may persist although we tried to mask it with grape flavor. Torres-González et al[62] reported improvement of coconut oil sensorial features after deodorization. Before that process, subjects in their study characterized coconut oil taste as “rancid”, “coconut”, “bitter” or “soap”. As this oil is not commonly consumed in our region, we believe that subjects were surprised and rated the unknown taste as not too good.

Results from few clinical studies to date regarding satiety effects of MCFA remain inconclusive. Poppit et al[28] compared subjective appetite sensations and subsequent food intake of lean men after intake of high fat muffins containing 10g of coconut oil or 3g of short chain fatty acid with LCT-rich muffins. In that study, there was no difference between the 3 fatty acids in any of the outcomes, possibly because the two test breakfast also had high amounts of LCT (~ 48%) as control and low content of test oil. In accordance, Van Wymelbeke et al[15], Coleman et al[29], Rizzo et al[63], and Roll et al[64] also failed to observe any differences in subjective appetite sensations after synthetic MCT oil intake compared to LCT.

Thus, satiety sensations regarding MCFA intake has been demonstrated from studies that evaluated energy and macronutrient intake in subsequent meals after MCFA and not from subjective appetitive sensations. Results from these studies showed reduced food intake in ad libitum lunch[15,64] or for total consumption during subsequent meals (snack or dinner)[14,29] following a MCFA rich breakfast, although Poppit et al[28], Rizzo et al[63] and we could not observe these effects.

The exact mechanism underlying a reduction in food intake after MCFA is not fully understood[29,65]. High ketone body production after synthetic MCT oil intake was demonstrated[15]. Thus, it is believed that a rapid oxidation of MCFA leads to ketone bodies production increase, mainly β-hidroxybutyrate, that inhibits food intake[66]. Nevertheless, it is unlikely that this mechanism occurred in our population since fat oxidation results do not support this hypothesis.

Both types of fats tested in this study increased fullness and satiety sensation equally. Increase in cholecystokinin (CCK) plasma levels due to fat intake is a potent endogenous satiety factor[67]. In short term, it has been suggested that MCFA stimulate CCK less than LCFA[21]. However, lauric acid seems to differently stimulate CCK secretion than others MCFA. Duodenal infusion of MCT emulsions containing only C8:0 and C10:0 did not stimulated CCK release just like infusion of saline[68]. In contrast, McLaughlin et al[69] and Feltrin et al[70] demonstrated that duodenal infusion of lauric acid increased in CCK secretion likewise LCT. This could be an underlying mechanism in the increase of fullness and satiety sensations.

On the other hand, coconut oil was able to delay subjective sensation of hunger and prospective food consumption in obese women. These are beneficial effects with regard to body weight regulation as delaying onset and desire for food consumption which could lead to less energy intake at next meal and / or throughout the day. Despite these findings in our study, there were no differences in daily energy and macronutrient intake.

A limitation of this study concerned the potential impact of standard lunch given after assessments on test day in the satiety assessment. We are aware that this method does not allow reproduction of participants' normal eating pattern such as meal sizes, but the assessment of energy and macronutrient intake in the rest of the day by food diaries allows accurate interpretation of the effects of test oils in long term satiety. Nevertheless, this strategy has the advantage of standardizing the eating pattern after

long period without food, avoiding overconsumption due to high hunger sensation showed by VAS analyses.

Although our study did not observed any positive effect of coconut oil on cardiometabolic risk markers, several studies have demonstrated that coconut oil consumption increases HDL-c concentrations[26,71–74]. Consistently, coconut oil also increased Apo A-I concentrations[75]. These results were also observed when palm kernel oil, another oil rich in lauric acid, was tested[76]. Recent systematic review of coconut oil effects on cardiovascular markers concluded that despite this increase in HDL-c concentrations, the overall lipid profile after its intake was not able to cause substantial reductions in cardiovascular risk[25]. Some studies indeed observed increase in total cholesterol and LDL-c[71,73,77] by coconut oil, but this effect could not be seen in a short period of time.

Any of the above mentioned studies suggested a possible mechanism for HDL-c increase after coconut oil intake. It has been suggested that lauric acid intake could be able to increase total HDL-c when compared to MCT oil through increase in mean rates of cholesterol esterification and transfer by Lecithin Cholesterol Acyl Transferase (LCAT) and Cholesterol Ester Transport Protein (CETP) enzymes, respectively[78]. However, further investigation on this mechanism is necessary.

Lower increase in uric acid concentration was seen after VCO intake. This was an unexpected effect once there are no studies that associated different fatty acid with uric acid. Moreover, uric acid effects were not the same between increase in all time points with soybean oil and only at 4 hours with VCO. Increases in uric acid have been associated with metabolic syndrome[79] and cardiovascular disease[80], both strongly related to obesity. Thus, an improvement on this parameter could be beneficial for obese subjects. However, further investigations of the effects of fatty acids on uric acid concentrations must be performed.

Conclusion

This study demonstrated that acute consumption of virgin coconut oil does not increase energy expenditure or fat oxidation but delays the motivation to eat compared to soybean oil. It also did not improve cardiometabolic markers in obese women. The results suggest that postprandial changes in energy metabolism are not the prominent mechanism by which coconut oil leads to weigh loss. Thus, long-term studies with

obese subjects should be conducted before the widespread use of coconut to prevent or treat obesity

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3.2 Artigo 2 - Original research

Virgin coconut oil consumption does not improve weight loss and cardiometabolic risk profile of obese women following energy restricted diet

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Abstract

Background: Virgin coconut oil (VCO) is the main natural source of medium-chain fatty acid with possible benefits on obesity treatment. Despite the attributed benefits which support its prescription worldwide, the role of VCO on obesity treatment remains unknown.

Objective: To investigate if VCO consumption associated with energy restricted diet could chronically improve body weight/fat loss, cardiometabolic risk markers, and liver function in obese women.

Methods: Thirty-eight obese women (46.5 ± 0.6 % of body fat) aged between 20-40 participated in this randomized, double-blind, placebo-controlled clinical trial assessing the effects of daily consumption of soybean oil (control) or virgin coconut oil (VCO) associated with energy restricted diet (-500 kcal/d) for 9 weeks. Anthropometric (hip, arm, neck, thigh and waist circumferences, and sagittal abdominal diameter), body composition (lean mass, total, truncal, android, and gynoid fat mass by dual-energy X ray absorptiometry), and blood pressure were measured in the first and last day of intervention. Blood was collected in fasting state and after 2 and 4 hours postprandially.

Results: Body weight, body mass index, all circumferences and sagittal abdominal diameters, systolic blood pressure, total and truncal body fat decrease in both groups after intervention period. However, only control group had the percentage of android fat mass reduced after 9 weeks. There was difference between changes in HDL-c concentration between groups due to reduction in HDL-c values in control group.

Conclusion: Virgin coconut oil did not improve anthropometric and body composition benefits caused by energy restricted diet. Also, it did not improve cardiometabolic risk profile of obese women.

Introduction

Obesity is mainly a result of long-term energy overconsumption. To prevent excessive adiposity and its metabolic consequences, such as cardiovascular disease, efforts to investigate dietary factors that could lead to body fat accumulation and obesity-related disorders have been of great interest[1]. Negative energy balance through energy restriction is a mandatory nutritional strategy for weight loss[2]. Despite high energy density of dietary fats, the role of fatty acid in weight management has been extensity revised[3,4].

Medium-chain fatty acids (MCFA) are metabolically active agents which have been associated with weight control[5]. The results of human studies have been demonstrated that the consumption of synthetic MCT oil, exclusively composed by caprilyc (C8:0) and capric (C10:0) acids, results in weight and fat loss[6–9]. On the other hand, the results of studies evaluating MCFA effects on cardiometabolic risk markers have been inconclusive[10–12].

Virgin coconut oil (VCO) is the main natural source of MCFA. For this reason, it is being promoted as healthy oil with benefits including its use for obesity treatment[13]. However, its real effect on weight management still unknown. The main MCFA in VCO is lauric acid (C12:0). Caprily and capric acids correspond to less than 10% of its total fatty acid content[14]. Thus, the difference in fatty acids composition between VCO and synthetic medium-chain triglyceride (MCT) oil raise questions if it is appropriate to extrapolate health claims attributed to synthetic MCT oil to its natural source. Randomized clinical trials assessing VCO effects on weight loss are very scarce and have methodological issues[15,16] and those evaluating lipid profile are very contradictory[17].

Therefore, we investigated if chronic VCO consumption associated with energy restricted diet could chronically improve body weight/ fat loss, and cardiometabolic risk markers. Additionally, because saturated fat could impair liver metabolism, we evaluated the role of this high-saturated oil in liver function.

Methods

Subjects

Obese women aged 20-40 years, with high body fat content (>30% of body weight), non-smokers, and ethanol consumption lower than 15g/day were recruited by

written advertisements and social network. Seven hundred and fifty two women were assessed for eligibility. Exclusion criteria were changes in body weight (>5%) and physical activity level over the previous 3 months, practice of >10 h of exercise/week, following weight loss diet, use of any drugs other than contraceptives, presence of acute or chronic diseases other than obesity, pregnancy and breastfeeding. Fifty two young adult obese women were included in the study. Fourteen were excluded during intervention period due to pregnancy (n = 1), pathological events not related with intervention (n=4), lack of adhesion to study protocol (n = 2) and withdrawal (n = 7) (Figure 1).

The study was approved by the Ethical Committee in Human Research from Federal University of Viçosa, Brazil (protocol number 892,467/2014), was conducted in accordance with 1964 Declaration of Helsinki and its later amendments and was registered at http://www.ensaiosclinicos.gov.br/ (protocol number: 7558514.5.0000.5153). All subjects signed a written informed consent.

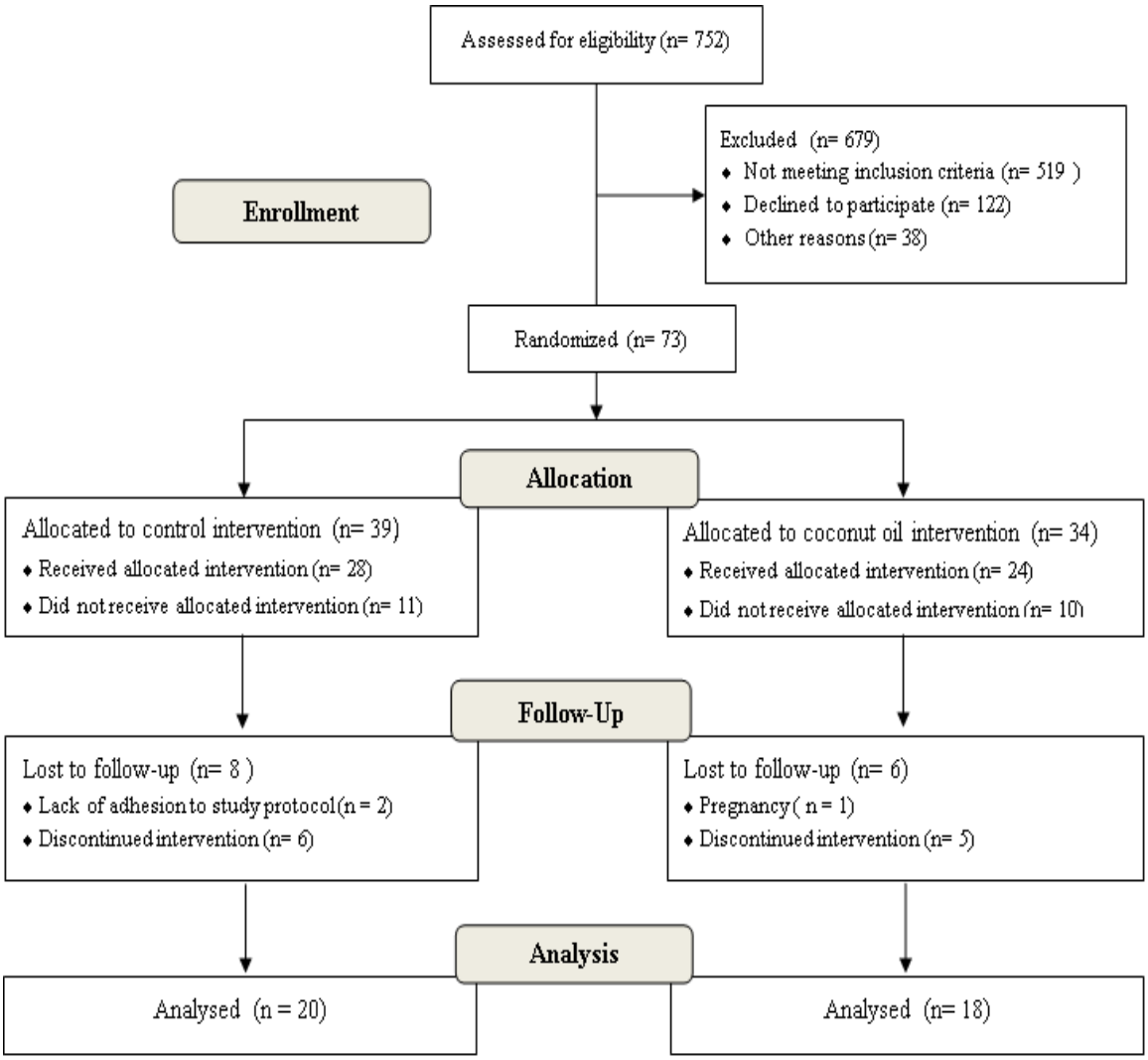

Figure 1 - CONSORT diagram showing participants flow through each stage of the trial. CONSORT: Consolidated Standards of Reporting Trials

Experimental design

This study was a 9-week, double-blind, randomized, and controlled trial with parallel group design in free-living subjects. Selected volunteers were randomly assigned to one of the two experimental groups - control (soybean oil high in the major dietary PUFA, linoleic acid, 18:2 n-6) or virgin coconut oil (VCO high in the SFA, lauric acid, 12:0) by the block randomization technique[18], with allocation ratio 1:1. All evaluations were performed in the first and last day of the study and the same protocol was followed in both days.

One week before trial beginning women were instructed not to consume alcohol and caffeine-contained beverages and to maintain their usual dietary and lifestyle habits. A standard dinner was given to volunteers to be consumed the night before test day. This meal consisted of instant plain noodles (Nissin®), parmesan cheese (Santa Amália Alimentos®), and 200 ml of orange juice (Sucos Tial®) (600kcal, carbohydrate: 62%E, protein: 8.5% and fat: 29.4%E). Women reported to laboratory in fasting state for anthropometric, body composition, and blood pressure assessments. Then, subjects underwent blood collection and consumed one of the two test drinks containing 25 mL of soybean oil (Corcovado, Archer Daniels Midland, Uberlândia, Brazil) or VCO (Copra, Copra Indústria Alimentícia Ltda, Brazil) for breakfast. Each drink was consumed within 10 minutes and participants remained in the laboratory for 4 hours for further blood collections. After completing all study protocol, a standard lunch composed of sandwich and fruit juice was offered (325 kcal; carbohydrate: 61,8%E, protein: 18.2% and fat: 28.0%E). Three-day food records were filled one week before the first evaluation day and at the last week of intervention period to asses for diet compliance. At the end of first test day, the individualized diet was prescribed.

During intervention period, subjects attended the laboratory daily on week days to have breakfast containing the tested oils according to allocated group. On weekends, identical breakfasts containing the test oils were provided to be consumed at home. Daily breakfasts consisted of 300 mL of isocaloric milk-based drinks matched for all ingredients other than oil content and two low-fat cookies (Table 1). A rotating menu of six breakfasts flavors with very similar nutritional composition were prepared to avoid monotony and improve compliance to study protocol. In test days (baseline and final), it was offered only a high-fat grape-artificially flavored milk drink (test drink) containing test oils for breakfast in order to avoid interference from other food components on analyses (Table 1). Except for fat quality, the drinks were identical with regard to energy, fat, carbohydrate, and protein, as well as taste and structure (Table 1).

Table 1 - Test drink and daily breakfasts nutritional composition and fatty acid profile of tested oil.

	Control	Coconut oil
Test drink*		
Energy (kcal)	298.6	298.6
Carbohydrate (g)	10.0	10.0
Protein (g)	6.9	6.9
Total fat (g)	25.0	25.0
Daily Breakfasts meals§		
Energy (kcal)	374.8 ± 4.4	374.8 ± 4.4
Carbohydrate (g)	26.2 ± 0.8	26.2 ± 0.8
Protein (g)	9.7 ± 0.2	9.7 ± 0.2
Total fat (g)	25.7 ± 0.1	25.7 ± 0.1
Fatty acid profile (%)		
C8:0	-	2.5 ± 0.4
C10:0	-	5.5 ± 0.2
C12:0	0.3 ± 0.1	55.0 ± 0.9
C14:0	0.2 ± 0.1	19.5 ± 0.1
C16:0	10.8 ± 0.1	8.5 ± 0.2
C18:0	3.7 ± 0.1	3.1 ± 0.1
C18:1 ω9	31.7 ± 0.3	-
C18:2 ω6	52.3 ± 0.4	5.3 ± 0.2
Total MCFA	0.3 ± 0.1	63.1 ± 0.6
Total LCFA	99.7 ± 0.1	36.9 ± 0.6
Total SFA	15.0 ± 0.2	94.1 ± 0.2
Total MUFA	32.0 ± 0.3	-
Total PUFA	53.0 ± 0.4	5.9 ± 0.2

C8:0 = caprylic acid; C10:0 = capric acid; C12:0 = lauryc acid; C14:0 = myristic acid; C16:0 = palmitic acid; C18:0 = stearic acid; C18:1 ω9 = oleic acid; C18:2 ω6 = linoleic acid; MCFA: medium-chain fatty acids; LCFA: long-chain fatty acids; SFA: saturated fatty acids; MUFA: monounsaturated fatty acids; PUFA: polyunsaturated fatty acids. *Offered only test days (baseline and final) in order to avoid interference from other food components on measurements. §Mean ± SE of the six rotating menu offered along intervention period. Fatty acids profile was obtained after esterification[19] by gas chromatography.

Diet prescription

Energy restricted nutritionally balanced diets were individually prescribed by a single dietitian. General pattern of the diets such as macronutrient distribution were maintained during the intervention to reduce influence of prescribed diets on outcomes. Individuality was attained by adapting portions size in relation to caloric prescription of each individual (Table 2). Energy requirements were estimated according to total energy expenditure using specific formula for overweight/obese women[20] and the energy restriction (-500 kcal/d) were applied to calculated values. Physical activity levels[21] were based on physical activity coefficients (1.00 for low-active or 1.16 for irregularly-active individuals)[22].

Table 2 - Mean ± SEM prescribed diets nutritional composition according to experimental groups.

	Control (n = 20)	Coconut oil (n = 18)
Energy (kcal)	1,884.5 ± 51.3	1,865.8 ± 51.3
Carbohydrate (g)	232.8 ± 7.8	229. 4± 7.8
(%E)	49.1 ± 0.5	49.1 ± 0.6
Protein (g)	89.4 ± 2.6	92.3 ± 2.9
(%E)	19.0 ± 0.4	19.9 ± 0.6
Fat (g)	66.5 ± 1.6	64.1 ± 2.1
(%E)	31.8 ± 0.5	31.0 ± 0.6

%E: Percentages of total energy prescribed. Nutritional information was obtained from manufacturer's products information and from Brazilian Food Composition Table[23]. Diet prescriptions were conducted by a single dietitian using DietPro software (version 5.2i, Agromídia, Viçosa, Brazil). There were no significant differences between groups ($P > 0.050$, Student's t-test).

Dietary Intake Assessment

Dietary intake was assessed by three non-consecutive days (two week days and one weekend day) 24-h food records. Subjetcs were asked to fill food records one week before the beginning of study (baseline) and on the last week of intervention period (final) to asses for diet compliance. Macro- and micronutrient intakes were analyzed by a single dietitian using DietPro software (version 5.2i, Agromídia, Viçosa, Brazil) based on reliable composition tables[23,24].

Fatty acid profile

Fatty acids composition of soybean and VCO was assessed in laboratory after esterification[19] and serum fatty acids profile was assessed after transesterification[25] by gas chromatography (GC). Chromatographic analysis was carried out using a Shimadzu GC Solution instrument (Shimadzu Seisakusho Co., Kyoto, Japan) equipped with a flame ionization detector (FID) and a DB5 column (30 m x 0,25 mm). The temperature program started with an initial temperature of 100 º C with a 5 min hold, which was increased with 4 ºC/min to 220 ºC, followed by 40 min isothermal period. The temperature of the FID and the injection port was 200ºC and 240ºC, respectively. Nitrogen gas flow was 43.2cm/s and FAME was identified by direct comparison with a FAME standard mix (Supelco 37 Component FAME Mix; Sigma-Aldrich). Each individual peak was integrated and the percentage of individual FAME was made in relation to total area.

Anthropometric, body composition, and blood pressure measurements

Anthropometric measurements were assessed by a single investigator. Body weight were measured on a digital platform scale with a resolution of 0.5 kg (Toledo®, Model 2096PP/2, São Paulo, Brazil), while subjects were barefoot and wearing lightweight clothing. Height was measured to the nearest 0.1 cm using a wall-mounted stadiometer (Wiso, Chapecó, SC, Brazil). BMI was calculated by dividing body (kg) by height (m) squared. Waist, hip, neck, and thigh circumferences, as well as sagittal abdominal diameter were measured in triplicate as described by Vasques et al[26]. Waist circumference and sagittal abdominal diameter were measured in four distinct regions: midpoint between the last rib and iliac crest; narrowest waist; umbilical level and immediately above the iliac crests.

Blood pressure was measured by an automatic Omron HEM-7200 device (Omron Inc., Dalian, China) in both arms in a sitting position after 5 minutes of rest. That measurement was repeated two more times with 5 minutes of interval in the arm that presented the higher values. The average of the two nearest measurements was recorded.

Dual energy X-ray absorptiometry scan (DXA) (model Prodigy Advance, GE Healthcare Inc., Waukesha, WI) was performed to assess changes in body composition

according to manufacturer's instructions. Values of lean mass, total body fat, and fat distribution (truncal, gynoid, and android regions) were obtained.

Cardiometabolic markers and liver enzymes

Antecubital blood samples were collected in the fasting state (12 h) and at 120 min and 240 minutes postprandially in the first and last day of intervention period. Serum samples were separated from whole blood by centrifugation (3,500 rpm, 4°C, 15 min) and immediately frozen at -80°C until analyses. Glucose, uric acid, total cholesterol, high-density lipoprotein cholesterol (HDL-c), low-density lipoprotein cholesterol (LDL-c), triglycerides (TG), γ-glutamyltransferase (GGT), aspartate amino transferase (AST), and alanine amino transferase (ALT), alkaline phosphatase (AP) were quantified by standard colorimetric kits (Bioclin®, Minas Gerais, Brazil) by automatic biochemical analyzer BS-200 (Mindray Medical International Ldt., Shenzen, China).

Very low-density lipoprotein (VLDL) was estimated by Friedewald et al[27]. Insulin was assessed by chemiluminescence method (Elecsys-Modular E-170, Roche Diagnostics Systems). Insulin resistance was estimated by the Index of Homeostasis Model Assessment of Insulin Resistance (HOMA-IR) using Matthews et al[28] equation. Insulin resistance was considered when HOMA-IR index were ≥ 2.71[29]. Atherogenic Index (TG/HDL ratio)[30], total cholesterol / HDL ratio and incremental area under (iAUC) or above (iAAC) the curves of each cardiometabolic risk marker were also calculated[31].

Statistical analysis

Data were typed by two independent investigators to ensure data reliability. Sample size was calculated[32] considering 10% difference in body weight, and a statistical power of 90%. Statistical analyses were carried out on SPSS 20 for Windows (SPSS, Inc., Chicago, IL, USA). Data are expressed as means ± (standard errors of the mean (SEM). Individual outlier values were excluded before analyses. Data normality and homoscedasticity were assessed by Shapiro-Wilk and Levene tests, respectively. Student's t-test or Mann-Whitney U signed-rank test were used to assess differences in changes (Δ) values between two interventions days (P_{inter}). To assess intra-individual differences within each group over time, paired t test or Wilcoxon test were performed.

Chi-square test were used to evaluate association between categorical variables. A P-value of < 0.05 was considered significant (P_{intra}).

Results

Breakfasts were well tolerated and no side effects were reported by the subjects of both groups. There were no differences in subjects' characteristics at baseline (Table 3). The majority of women were low-active (79%; n = 30) and there were no association between group and physical activity level (P_X^2= 0,154).

Table 3 - Subjects' baseline characteristics.

Characteristic	Control (n=20)	Coconut Oil (n=18)
Age (years)	27.2 ± 1.4	27.2 ± 1.5
BMI (kg/m^2)	29.8 ± 0.7	30.9 ± 0.8
Waist circumference (cm)	96.2 ± 1.5	96.3 ± 2.0
Fat mass (kg)	37.0 ± 1.5	36.5 ± 1.8
Body fat percentage (%)	46.6 ± 0.7	46.3 ± 1.1
Systolic/diastolic blood pressure (mmHg)	109.1 ± 2.2 / 68.3 ± 1.8	111.4 ± 2.2 / 67.5 ± 1.6

BMI: Body Mass Index; Waist circumference was assessed in the midpoint between the last rib and iliac crest. There were no differences in baseline characteristics between groups (Student's t-test, $P > 0.050$).

There were no differences in energy, carbohydrate, protein and total fat consumption between groups during intervention period. As expected, coconut oil group had the highest saturated fat and lauric acid intake and the lowest monounsaturated and polyunsaturated fat, such as oleic and linoleic fatty acids respectively (Figure 2).

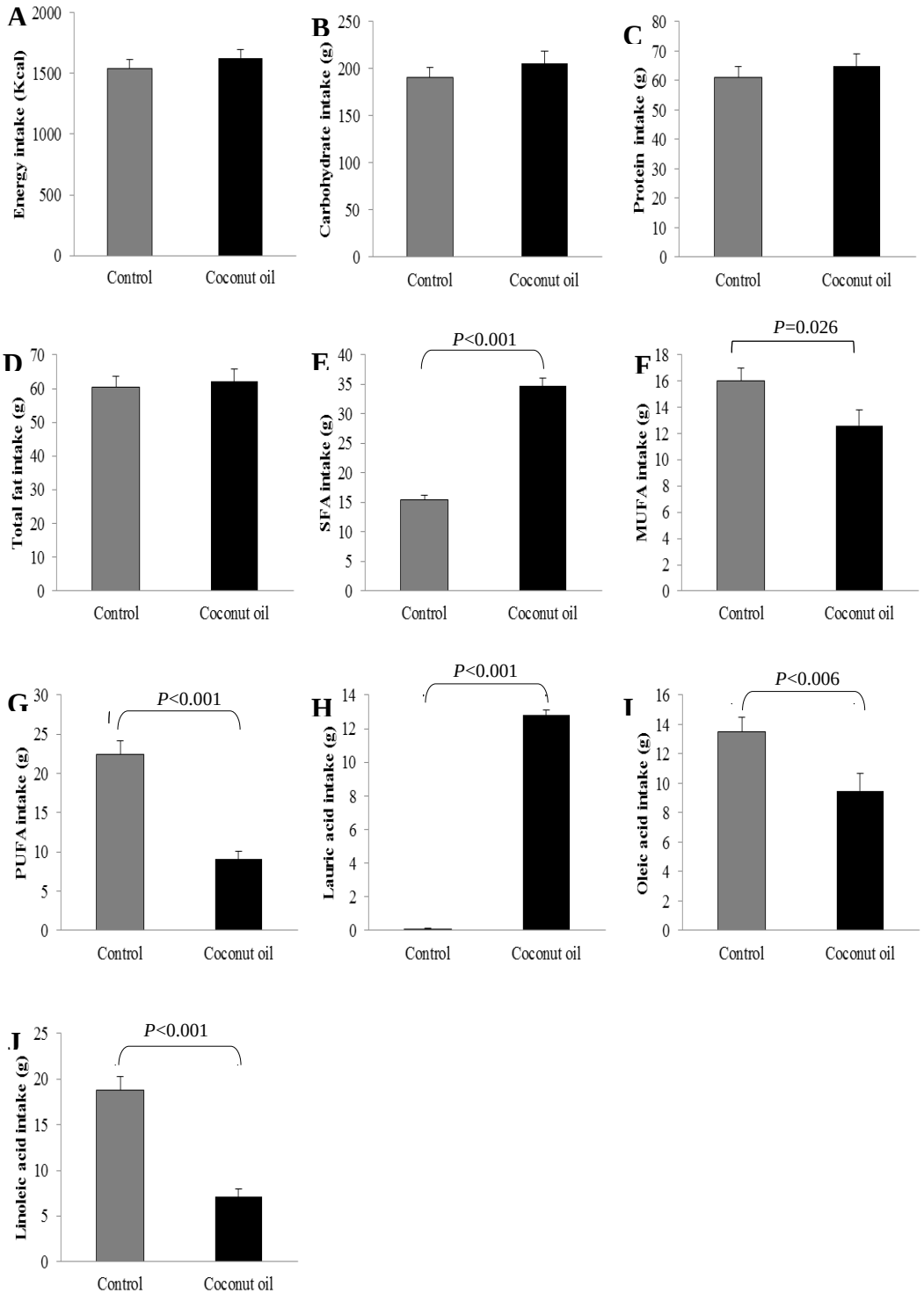

Figure 2 - Mean ± SEM of energy (A), carbohydrate (B), protein (C), total fat (D), saturated fat (E), monounsaturated fat (F), polyunsaturated fat (G), lauric acid (G), oleic acid (H) and linolenic acid (G) consumption during intervention period according to experimental groups.

*$P < 0.050$ Student's t test or Mann-Whitney U signed-rank test as appropriate. Control is soybean oil.

Although in the first intervention day serum fatty acid profile was not affected, lauric and myristic acid concentrations increased postprandially after 9 weeks of VCO consumption. There were no differences in mono and polyunsaturated fatty acid analyzed both on the first day and after 9 weeks (Figure 3).

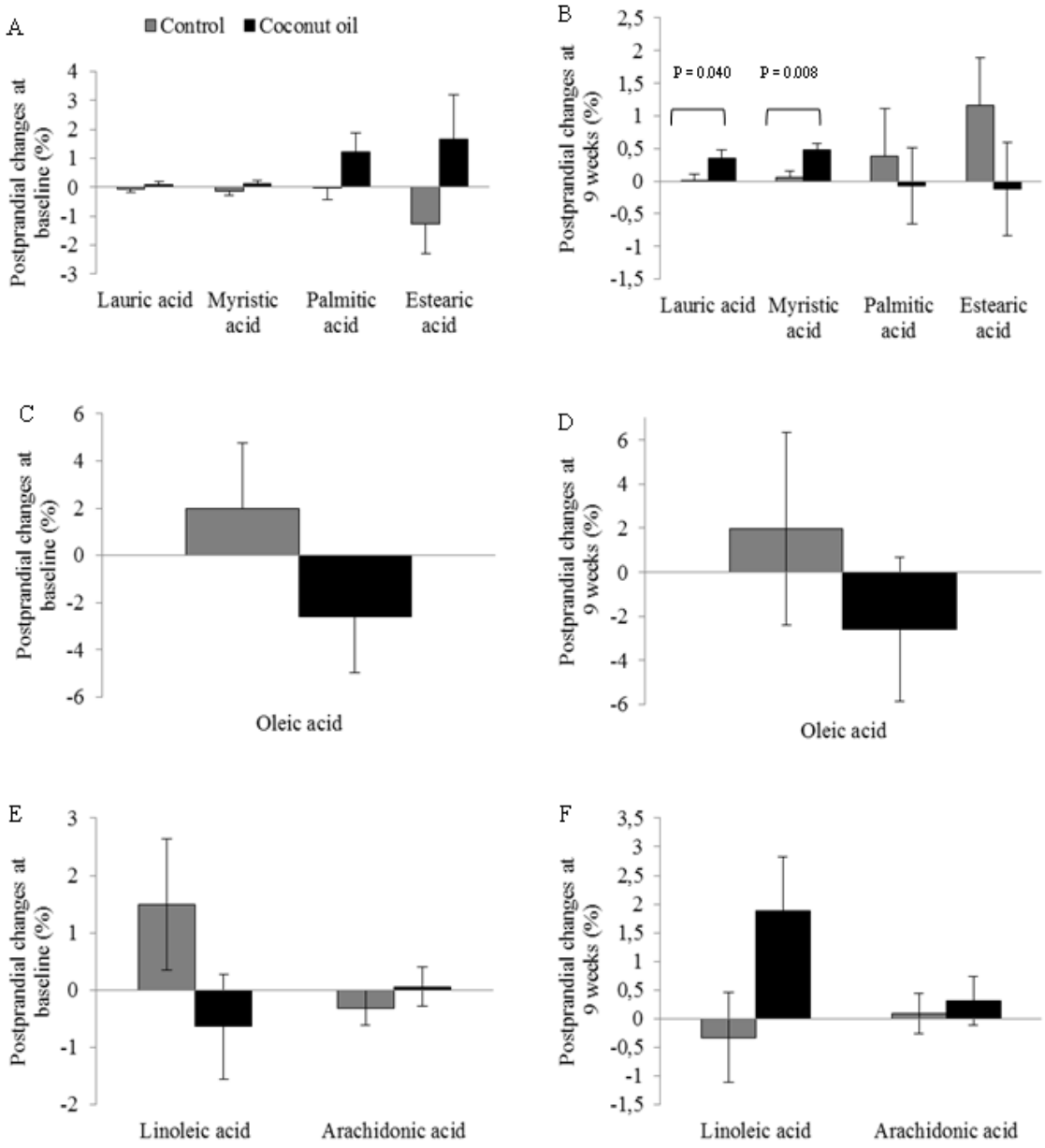

Figure 3 - Mean ± SEM of changes in saturated (A and B), monounsaturated (C and D) and polyunsaturated (E and F) serum fatty acids before (baseline) and after (9 weeks) the dietary intervention with 25mL of soybean oil (control) or virgin coconut oil (coconut oil). Changes values were calculated by subtracting postprandial values (2 hours after test drink consumption) from fasting values. Student's t-test, $P<0.050$.

Except for thigh circumference in coconut oil group, all anthropometric variables assessed reduced in both groups. However, between groups analyses did not indicate differences in changes from the baseline values for all variables evaluated (Table 4).

Regarding body composition measurements, lean mass and gynoid fat were not affected by any treatment. Total body fat and truncal fat decreased in both groups. However, only control group had the percentage of android fat mass reduced after the intervention (Table 4).

Systolic blood pressure was reduced after both treatments (-3.9 ± 1.9 mmHg for control group and -3.6 ± 1.6 mmHg for coconut oil group) without differences between groups (P_{inter} = 0.864). Diastolic blood pressure did not change along time in both groups (P_{intra} = 0.591 for control and P_{intra} = 0.660 for coconut oil) and changes between groups also not differed (P_{inter} = 0.421).

Table 4 - Anthropometric and body composition measurements assessed before (baseline) and after (9 weeks) dietary intervention according to experimental groups

	Control			Coconut oil			P_{Inter}
	Baseline	9 weeks	P_{Intra}	Baseline	9 weeks	P_{Intra}	
Anthropometric measurements							
Body weight (kg)	79.2 ± 2.3	77.5 ± 2.3	**0.003**	78.5 ± 3.1	75.3 ± 2.6	**0.010**	0.582
BMI (kg/m^2)	29.8 ± 0.6	29.2 ± 0.6	**0.003**	30.8 ± 0.8	30.3 ± 0.7	**0.014**	0.767
Waist circumference (cm)							
Narrowest waist	87.3 ± 1.6	84.6 ± 1.3	**<0.001**	87.4 ± 1.9	85.1 ± 1.9	**<0.001**	0.580
Midpoint between last rib and iliac crest	96.2 ± 1.4	93.0 ± 1.6	**<0.001**	96.3 ± 2.0	93.8 ± 2.0	**<0.001**	0.397
Umbilical level	99.2 ± 1.4	95.9 ± 1.6	**<0.001**	99.1 ± 2.3	96.2 ± 2.3	**<0.001**	0.135
Immediately above iliac crests	102.0 ± 1.4	99.1 ± 1.5	**<0.001**	101.9 ± 2.3	99.4 ± 2.1	**<0.001**	0.289
Sagittal abdominal diameter (cm)							
Narrowest waist	19.6 ± 0.4	18.5 ± 0.4	**<0.001**	19.3 ± 0.5	18.5 ± 0.5	**0.004**	0.522
Midpoint between last rib and iliac crest	19.6 ± 0.4	18.5 ± 0.4	**<0.001**	19.4 ± 0.5	18.3 ± 0.5	**<0.001**	0.883
Umbilical level	19.7 ± 0.5	18.8 ± 0.4	**0.001**	19.7 ± 0.5	18.8 ± 0.4	**<0.001**	0.697
Immediately above the iliac crests	20.9 ± 0.5	20.1 ± 0.5	**0.004**	20.9 ± 0.5	19.9 ± 0.5	**<0.001**	0.343

Values are means ± SEM P_{Intra}: within group variation to assess time effect (paired Student's t-test or Wilcoxon signed-rank test). P_{Inter}: between group variations to assess group effect over change (Δ) values (baseline values – 9 week values, Student's t-test or Mann-Whitney's test). Bold type values indicated significantly differences ($P < 0.050$). Test drinks contained 25mL of virgin coconut oil (coconut oil) or soybean oil (control).

Table 4 - Anthropometric and body composition measurements assessed before (baseline) and after (9 weeks) dietary intervention according to experimental groups (Continued)

	Control			Coconut oil			P_{Inter}
	Baseline	9 weeks	P_{Intra}	Baseline	9 weeks	P_{Intra}	
Hip circumference (cm	113.8 ± 1.5	111.5 ± 1.2	**0.029**	113.3 ± 2.0	111.8 ± 2.1	**0.001**	0.076
Thigh circumference (cm)	59.8 ± 1.0	58.53 ± 0.78	**0.001**	60.4 ± 1.1	59.74 ± 1.27	**0.077**	0.104
Neck circumference (cm)	35.4 ± 0.5	34.6 ± 0.5	**<0.001**	35.1 ± 0.5	34.4 ± 0.5	**<0.001**	0.633
Arm circumference (cm)	33.1 ± 0.4	32.3 ± 0.4	**<0.001**	34.8 ± 0.8	34.2 ± 0.8	**<0.001**	0.416
Body composition measurements							
Lean mass (kg)	39.0 ± 1.1	38.7 ± 1.1	0.320	39.0 ± 1.7	38.8 ± 1.7	0.553	0.813
Total body fat (kg)	37.0 ± 1.4	35.7 ± 1.4	**0.006**	36.5 ± 1.8	35.4 ± 1.9	**0.013**	0.685
Truncal fat (kg)	19.2 ± 0.8	18.5 ± 0.8	**0.027**	18.9 ± 0.9	18.2 ± 1.0	**0.030**	0.943
Gynoid fat (%)	19.6 ± 0.4	19.7 ± 0.4	0.844	19.4 ± 0.3	19.6 ± 0.4	0.390	0.288
Android fat (%)	8.5 ± 0.2	8.2 ± 0.2	**0.027**	8.2 ± 0.2	8.1 ± 0.2	0.421	0.320

Values are means ± SEM P_{Intra}: within group variation to assess time effect (paired Student's t-test or Wilcoxon signed-rank test). P_{Inter}: between group variations to assess group effect over change (Δ) values (baseline values – 9 week values, Student's t-test or Mann-Whitney's test). Bold type values indicated significantly differences ($P < 0.050$). Gynoid and Android fat are percentage of total body fat. Test drinks contained 25mL of virgin coconut oil (coconut oil) or soybean oil (control).

There was difference between changes in HDL-c concentration between groups due to reduction in HDL-c values in control group. Moreover, fasting glucose was reduced only after 9 weeks of control consumption (Table 5).

Table 5 - Fasting and total postprandial response of metabolic biomarkers assessed before (baseline) and after (9 weeks) dietary intervention according to experimental groups

	Control (n = 20)			Coconut oil (n = 18)			P_{Inter}
	Baseline	**9 weeks**	P_{Intra}	**Baseline**	**9 weeks**	P_{Intra}	
Glucose (mg/dL)	85.7 ± 1.7	83.5± 1.5	**0.025**	86.8 ± 2.0	84.9 ± 2.0	0.053	0.857
Total postprandial response (iAAC)[1]	727.5 ± 156.0	687.0 ± 168.1	0.222	712.3 ± 134.3	802.7 ± 162.9	0.677	0.359
Insulin (µIU/mL)	8.3 ± 0.5	9.5 ± 1.0	0.120	7.6 ±0.8	7.1 ± 0.6	0.953	0.250
Total postprandial response (iAUC)	128.4 ± 112.4	201.5 ± 185.8	0.176	242.8 ± 112.6	138.2 ± 80.6	0.672	0.068
Uric acid (mg/dL)	3.5 ± 0.1	3.7 ± 0.2	0.355	3.6 ± 0.2	3.4 ± 0.2	0.126	0.168
Total postprandial response (iAUC)	25.6 ± 7.5	24.5 ± 8.9	0.510	-1.8 ± 7.1	4.3 ± 6.6	0.353	0.081
Total cholesterol (mg/dL)	164.5 ± 7.4	156.5 ± 6.0	0.052	167.7 ± 6.4	168.8 ± 6.4	0.685	0.234
Total postprandial response (iAUC)	1,188.3 ± 226.4	1,352.6 ± 396.9	0.993	966.0 ± 200.9	1,157.5 ± 312.5	0.640	0.984

Values are means ± SEM. P_{Intra}: within group variation to assess time effect (paired Student's t-test or Wilcoxon signed-rank test). P_{Inter}: between group variation to assess group effect over changes (Δ) values (baseline values – 9 week values, Student's t-test or Mann-Whitney's test). Bold type values indicated significantly differences ($P < 0.050$). [1]Postprandial response of each cardiometabolic and liver enzyme were calculated as incremental area above the curve (iAAC) for glucose and incremental area under the curve (iAUC) over 4 hours for the other markers. Test drinks contained 25mL of virgin coconut oil (coconut oil) or soybean oil (control).

Table 5 - Fasting metabolic biomarkers and total postprandial response (iAUC) assessed before (baseline) and after (9 weeks) dietary intervention according to experimental groups (Continued)

	Control			Coconut oil			P_{Inter}
	Baseline	9 weeks	P_{Intra}	Baseline	9 weeks	P_{Intra}	
HDL-c (mg/dL)	49.2 ± 3.1	42.5 ± 2.7	**0.042**	45.4 ± 2.1	47.2 ± 2.8	0.161	**0.017**
Total postprandial response (iAUC)	67.3 ± 80.5	268.1 ± 85.5	0.071	165.0 ± 44.7	256.5 ± 111.1	0.282	0.476
LDL-c (mg/dL)	93.5 ± 6.0	91.0 ± 5.3	0.281	102.6 ± 5.3	102.1 ± 5.3	0.507	0.844
Total postprandial response (iAUC)	412.6 ± 84.8	407.5 ± 184.7	0.801	645.1 ± 143.8	481.6 ± 189.6	0.466	0.906
Triglycerides (mg/dL)	86.9 ± 8.0	77.1 ± 6.5	0.128	88.9 ± 4.6	83.5 ± 6.8	0.425	0.661
Total postprandial response (iAUC)	5,037.5 ± 346.7	4,601.3 ± 483.9	0.586	2,894.9 ± 488.7	3,370.5 ± 504.8	0,499	0.640
VLDL-c (mg/dL)	18.5 ± 2.2	15.4 ± 1.3	0.132	17.8 ± 0.9	16.7 ± 1.4	0.425	0.661
Total postprandial response (iAUC)	1,007.5 ± 173.8	920.3 ± 96.8	0.652	578.3 ± 97.9	674.1 ± 101.0	0.496	0.642
Total cholesterol/HDL-c	3.5 ± 0.2	3.6 ± 0.2	0.347	3.8 ± 0.2	3.6 ± 0.2	0.405	0.081
Total postprandial response (iAUC)	20.9 ± 6.2	20.9 ± 3.6	0.301	7.46 ± 3.2	11.6 ± 4.8	0.375	0.690
Triglycerides/ HDL-c	2.1 ± 0.3	1.7 ± 0.1	0.224	2.1 ± 0.2	1.9 ± 0.2	0.147	0.659
Total postprandial response (iAUC)	118.2 ± 25.6	109.3 ± 18.1	0.959	61.9 ± 11.8	72.2 ± 13.0	0.492	0.957

Values are means ± SEM. HDL-c: high-density lipoprotein cholesterol; LDL-c: low-density lipoprotein cholesterol; VLDL-c: very-low-density-lipoprotein cholesterol. P_{Intra}: within group variation to assess time effect (paired Student's t-test or Wilcoxon signed-rank test). P_{Inter}: between group variations to assess group effect over changes (Δ) values (baseline values – 9 week values, Student's t-test or Mann-Whitney's test). Bold type values indicated significantly differences ($P < 0.050$). [1]Postprandial response of each cardiometabolic and liver enzyme were calculated as incremental area above the curve (iAAC) for glucose and incremental area under the curve (iAUC) over 4 hours for the other markers. Test drinks contained 25mL of virgin coconut oil (coconut oil) or soybean oil (control).

Table 5 - Fasting metabolic biomarkers and total postprandial response (iAUC) assessed before (baseline) and after (9 weeks) dietary intervention according to experimental groups (Continued)

	Control			Coconut oil			P_{Inter}
	Baseline	9 weeks	P_{Intra}	Baseline	9 weeks	P_{Intra}	
AST (IU/L)	34.0 ± 1.6	31.3 ± 1.8	0.271	35.1 ± 2.1	34.6 ± 2.0	0.787	0.424
Total postprandial response (iAUC)	202.9 ± 129.5	399.2 ± 633.2	0.306	200.2 ± 127.4	174.2 ± 105.1	0.914	0.981
ALT (IU/L)	17.8 ± 1.6	16.1 ± 1.5	0.100	16.5 ± 1.3	15.8 ± 1.2	0.253	0.408
Total postprandial response (iAUC)	80.7 ± 68.8	214.4 ± 59.7	0.144	108.0 ± 64.4	186.8 ± 75.0	0.435	0.981
Gamma GT (IU/L)	21.9 ± 0.6	21.1 ± 1.5	0.627	21.6 ± 1.3	20.3 ± 1.0	0.392	0.660
Total postprandial response (iAUC)	-68.6 ± 46.6	28.7 ± 62.0	0.125	23.0 ± 32.6	74.4 ± 62.0	0.480	0.532
Alkaline phosphatase (IU/L)	61.1 ± 3.5	59.0 ± 3.7	0.312	56.8 ± 3.5	60.6 ± 4.0	0.323	0.701
Total postprandial response (iAUC)	549.4 ± 113.1	1,020.0 ± 169.0	0.051	430.1 ± 104.3	695.2 ± 153.3	0.398	0.346

Values are means ± SEM. Gamma GT: γ-glutamyltransferase; AST: aspartate amino transferase; ALT: alanine amino transferase. P_{Intra}: within group variation to assess time effect (paired Student's t-test or Wilcoxon signed-rank test). P_{Inter}: between group variation to assess group effect over changes (Δ) values (baseline values – 9 week values, Student's t-test or Mann-Whitney's test). Bold type values indicated significantly differences ($P < 0.050$). Control is soybean oil. [1]Postprandial response of each cardiometabolic and liver enzyme were calculares as incremental area above (iAAC) or under (iAUC) the curve over 4 hours.

Discussion

We investigated the effect of chronic VCO consumption associated with energy restricted diet as an adjuvant in obesity treatment. It has been argued that MCFA are easily and rapidly oxidized[33] they are less efficiently stored and are unlikely to promote obesity via direct storage in adipocytes[34]. Thus, we hypothesized that the MCFA-rich coconut oil could increase weight and fat loss promoted by energy restricton without impairing cardiometabolic risk markers and liver function. Unexpectedly, despite anthropometric and body composition benefic changes observed in both groups, VCO was not able to enhance these benefits compared to control treatment. Furthermore, VCO did not affect cardiometabolic profile. The postprandial increase in serum lauric and myristic fatty acids only after 9 weeks of coconut oil consumption indicate that the treatment was effective in changing the fatty acid profile in bloodstream.

Studies that evaluated MCFA effects on anthropometric and body composition measurements were conducted with synthetic oil composed mostly by caprylic (C8:0) and capric (C10:0) acids, which are greatly different from VCO which contains mainly lauric acid (C12:0). However, coconut oil is the natural source of MCFA and has been consumed for obesity control[13]. To our knowledge, there is only one study that evaluated coconut oil effects on anthropometry. In that study[15] women presenting abdominal obesity consumed 30mL of coconut oil or soybean oil for 12 weeks. A small increase in body weight (100g) and reduction in waist circumference (2 cm) was observed after coconut oil consumption. Contradictorily, we observed reduction in android body fat percentage in control group but not in the VCO group. In the previously mentioned study, the nutritional intervention was associated with physical activity protocol and several changes in dietary habits without monitoring oils consumption and diet prescription. Thus, their results should not be attributed exclusively to coconut oil and this fact could be responsible for the absence of corroboration with our results.

Similarity in anthropometric and body composition results obtained for control and VCO could be explained by the high content of lauric acid in VCO. Despite lauric acid (C12:0) has been classified as MCFA by some[35–39] this fatty acid shows intermediate properties between MCFA and long-chain fatty acid (LCFA)[36], which could lead to different metabolic fate compared to other MCFA (e.g. caprilic C8:0 and C10:0), like more fat deposit in adipose tissue.

AST and ALT are two of the most reliable markers of hepatocellular injury and their concentrations can be elevated in a variety of hepatic disorders[40]. Dietary factor such as SFA can modulate liver function [41,42] and promote hepatic steatosis in isocaloric and hypercaloric conditions[43,44]. Besides having high lauric acid content, which can behave like LCFA as mentioned above, VCO also is rich in saturated LCFA such as myristic and palmitic acids (~ 25%).

Changes in HDL-C concentrations after the intervention period were different between groups, but contrarily of studies that showed that coconut oil increased HDL-c concentrations[15,45–49] we demonstrate that these changes were due to a decrease in HDL-c in the control group. It is well documented that PUFA ingestion reduced HDL-c serum concentration [50–53]. VCO in our study behaved like a cholesterol-neutral oil in accordance to the observed by Cox et al[54], McKenney et al[55], Schwab et al[56], and Vijayakumar et al[57]

Our study has several strengths. First it addresses a topic of great debate in nutrition practice which is still poorly explored in scientific literature. Also, it has relevant clinical appeal once VCO is the main commercial MCFA source, which is largely available for population consumption worldwide. Furthermore, changes in body composition are strengthened by consistent findings using well-established DXA protocol. Personal influences over subjective measurements were avoided by our double-blind design, which rarely is feasible in dietary interventions that include foods rather than supplements or capsules. Because we evaluated VCO effects only in obese women, extrapolation for other population groups should not be appropriate.

Our study has potential limitations. Despite the fact that we selected women with very high body fat content (~ 46% at baseline) they were metabolic health and young and it is possible that the influence of dietary treatment in some metabolic biomarkers could not be assessed. Although the absence of effects observed, some authors suggest that obesity-related changes in gene expression could precede body changes[58–60]. Thus, we encourage the conduction of further studies to evaluate the role of VCO on adipose tissue gene expression capable to confirm the lack of VCO effects on obesity management.

Conclusion

Our results suggested that virgin coconut oil was unable to improve anthropometric and body composition benefits caused by energy restricted diet. Also, it

did not alter cardiometabolic risk profile. Given the observed results, we discourage the use of coconut oil as an strategy for obesity treatment.

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3.3 Artigo 3: Short communication

Virgin coconut oil chronic consumption does not improve energy metabolism, subjective appetitive sensations and food intake in obese women following energy restricted diet

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ABSTRACT

Background: Virgin coconut oil (VCO) has been receiving attention of scientific community due to its possible benefits over obesity treatment. However, the underlying mechanisms regarding VCO role on weight management needs to be clarified.

Objective: To investigate the chronic effects of coconut oil consumption associated with energy restricted diet on metabolic rates, subjective appetitive sensations, and food consumption in obese women.

Methods: This is a double-blind, randomized, placebo-controlled clinical trial which included 38 obese (46.5 ± 0.6 % of total body fat) adult (20-40 years) women. Women were assigned to receive 25mL/d of soybean oil (control group; n=20) or VCO (VCO group; n=18) associated with energy restricted diet (-500kcal/d) for 9 weeks. Metabolic rates, anthropometric, and body composition measurements were assessed at baseline and in the last day of the intervention period.

Results: Total fat oxidation increased, total carbohydrate oxidation and fasting fat oxidation decreased only in the control group. VCO did not increase resting or postprandial energy expenditure and diet-induced thermogenesis after the intervention period. However, VCO intake increased hunger sensation on week 9 compared to control group after 9 weeks without however changing subsequent energy or macronutrients intake.

Conclusion: Daily consumption of VCO not only improved metabolic rates and food intake, but also impaired hunger sensation in obese women following energy restricted diet. Thus, due to our results we do not recommend the use of VCO for obesity treatment.

Introduction

Virgin coconut oil (VCO) has been widely spread in the media as a health food ingredient due mainly to its high content of medium-chain fatty acids (MCFA)[1,2]. These fatty acids have 6 to 12 carbon atoms in its backbone and are directly absorbed into the portal vein and transported rapidly to the liver for β-oxidation[3]. For this reason, it has been suggested that MCFA are less accumulated as body fat, favoring weight loss[4].

Randomized clinical trials have shown that substitution of long-chain fatty acids (LCFA) for synthetic medium-chain triglycerides (MCT) oils could increase fat oxidation[5,6], energy expenditure[7,8], and diet-induced thermogenesis[9], besides promoting satiety and reducing food intake[10,11]. For these reasons, it has been widely reported that coconut oil consumption could also be helpful during obesity treatment due to postabsortive effects capable of causing weight loss similar to those showed by synthetic MCT oils[4,12,13.] However, these attributed claims lack scientific confirmation because the aforementioned studies[5–11] did not test coconut oil, which has different fatty acid composition to the one present in synthetic MCT oil, and were conducted in non-obese subjects. Thus, whether coconut oil consumption will promote the same benefits in obese individuals remains unknown.

Evidences that support coconut oil thermogenesis and appetite suppression benefits are scarce, inconsistent and/or present methodological limitations. Papamandjaris et al[14] failed to observe any effect of coconut oil associated with butter on total energy expenditure in non-obese women. On the other hand, White et al[15] observed increased basal metabolic rates after seven but not after fourteen days of coconut oil associated with butter consumption also in non-obese women. Likewise, Poppit et al[16] and Rizzo et al[17] did not find any changes in subjective appetitive sensations or food intake after coconut oil intake, possibly because the amount of LCFA on treatment diet was as high as in control.

Finally, energy restriction diets are the main nutritional strategy for weight loss[18] but very few studies evaluated the role of coconut oil and/or MCFA associated with energy restriction[19,20]. Therefore, considering the need to clarify the underlying mechanisms related to coconut oil on weight management, and the lack of studies in which coconut oil was consumed in association with energy restricted diet, we are now investigating the chronic effects of coconut oil consumption associated with energy

restricted diet on metabolic rates, subjective appetite sensations, and food consumption in obese women.

Methodology

Subjects

Written advertisements and social network were used for recruitment. Seven hundred and fifty two women were screened. Inclusion criteria were: women aged between 20-40 years old, Body Mass Index (BMI) between 26-35 kg/m^2, high body fat content (>30% of body weight), non-smoker, and ethanol consumption lower than 15g/day. Body weight (>5%) and physical activity level changes over previous 3 months, following weight loss diet, the use of any drugs other than contraceptives, presence of acute or chronic diseases, pregnancy and breastfeeding were the exclusion criteria. Fifty two young adult obese women were included in the study. Fourteen were excluded during intervention period due to pregnancy (n = 1), pathological events not related with intervention (n=4), lack of adhesion to study protocol (n = 2) and withdrawal (n = 7). From the 38 remaining volunteers, 8 refused to be submitted to final indirect calorimetry assessment and 1 scored VAS wrongly and was excluded from this analysis. Final sample included 29 subjects analyzed for energy metabolism and 37 for appetite assessments. Details of the screening process and study population have been previously described (article 2).

The study was approved by the Ethical Committee in Human Research from Federal University of Viçosa, Brazil (protocol number 892,467/2014) and was conducted in accordance with 1964 Declaration of Helsinki and its later amendments. All subjects signed a written informed consent.

Study design

This was a randomized, controlled, double-blind clinical trial of 9-weeks (± 5 days) duration. Subjects were randomly assigned to one of the two experimental groups: control (soybean oil) or virgin coconut oil (VCO). All evaluations were performed in the first and last day of the study and the same protocol was followed in both days. A standardized meal was given to volunteers to be consumed in the night before evaluation day (600kcal, carbohydrate: 62%E, protein: 8.5% and fat: 29.4%E). Subjects were also instructed not to consume alcohol or caffeine containing drinks, to refrain

from heavy physical activity and to maintain a regular sleep-wake schedule (8 hours/night) on the day before evaluation days.

After 10 hours of overnight fast, subjects attended the laboratory where they remained for 6 hours for all evaluations. For more study protocol details see article 1. After completing all study protocol, a standard lunch composed of sandwich and fruit juice was offered (325 kcal; carbohydrate: 61,8%E, protein: 18.2% and fat: 28.0%E) and subjects were instructed to record all foods and beverages consumed for the rest of the day to evaluate subsequent food intake. Also, three-day food records were filled one week before the first evaluation day and at the last week of intervention period to asses for diet compliance. At the end of first evaluation day, the individualized diet was prescribed.

On week days, subjects went to laboratory daily to have breakfasts containing 25 mL of soybean oil (Corcovado, Archer Daniels Midland Company, Brazil) or VCO (Copra, Copra Indústria de Alimentícia Ltda, Brazil). On weekends, identical breakfasts containing the test oils were provided to be consumed at home. Daily breakfasts consisted of 300 mL of isocaloric milk-based drinks matched for all ingredients other than oil content and two low-fat cookies (~374.8 kcal; carbohydrate: 27,9%E, protein: 10.3% and fat: 61.6%E) (article 2). A rotating menu of six breakfast flavors with the same nutritional composition were prepared to avoid monotony and improve compliance to study protocol. In evaluation days, it was offered only a high-fat grape-artificially flavored milk drink (test drink) containing test oils for breakfast in order to avoid interference from other food components on analyses (298.6 kcal; carbohydrate: 13.4%E, protein: 9.2% and fat: 75.3%E).

Dietary intervention

Energy restricted diets (-500 kcal/day) were individually prescribed by a single dietitian, considering total energy requirements and physical level activity of each subject. Energy requirements were estimated according to total energy expenditure for overweight/obese women[21] and the physical activity factor was determined using the International Physical activity Questionnaire (IPAQ)[22] and calculations were based on physical activity coefficients (1.00 for sedentary or 1.16 for low-active individuals)[23]. Then, energy restriction was applied.

The prescribed diets contained 1870 kcal ± 51 kcal; 49% carbohydrate, 19% protein and 31% fat and for both groups. The total caloric prescription was distributed in 5-6 meals a day. Dietary intervention was implemented in a free-living condition. Thus, all subjects received nutritional advice and a one-day plan menu containing foods according to their preferences. They also received and were instructed on how to use a substitution food list. This list contained food items assigned into categories according to their macronutrient composition, allowing subjects to plan their own menus and choose foods for their meals based on their one-day dietary prescription. The caloric value of the breakfasts containing the test oil was considered in the prescribed diet. Subjects were requested to follow strictly the prescribed diet and to maintain their habitual level of physical activity during intervention period. The monitoring of diet compliance and side effects of test oils was assessed daily by subjects' attendance to have breakfasts and individual conversations whenever necessary.

Anthropometry and body composition

Body weight, height, waist circumference, and body composition were assessed in fasting state. Body weight were measured on a digital platform scale (Toledo®, Model 2096PP/2, São Paulo, Brazil) while subjects were barefoot and wearing lightweight clothing. Height was measured to the nearest 0.1 cm using a wall-mounted stadiometer (Wiso, Chapecó, SC, Brazil). BMI was calculated dividing body (kg) by height (m) squared. Waist circumference was assessed in the midpoint between the last rib and iliac crest. Anthropometric measurements were assessed by a single investigator. Body composition (lean mass and total body fat) was evaluated by Dual energy X-ray absorptiometry scan (DEXA) (model Prodigy Advance, GE Healthcare Inc., Waukesha, WI) according to manufacturer's instructions.

Metabolic rates measurement and calculations

Fasting (REE) and postprandial energy expenditure, carbohydrate and lipid oxidation rates were measured by indirect calorimetry using an open-circuit ventilated canopy measurement system (Carefusion Vmax ® Series, California, EUA). The flow meter and flow sensor calibration of the bidirectional digital turbine flowmeter were performed daily using a 3 L syringe and analyzers were calibrated prior to every run with gases of known concentration as recommended by manufacturer (gas

#1: 26% O_2 nitrogen balance; gas #2: 4% CO_2 and 16% O_2 nitrogen balance; gas #3 ambient air).

Subjects laid supine, with their head elevated 30 degrees, for a mandatory 20 minutes rest period before REE test. They were in a quiet room with stable temperature (22ºC) and humidity (55%) and were not allowed to sleep during measurements. A transparent ventilated hood was positioned over the subject's head and expired gases were continuously collected. REE was measured after 11 hours fast, at 0800–0900. Postprandial measurements were made every 30 minutes for each hour during 4h after test meal[24]. Flow rate were regularly adjusted to maintain a constant $FECO_2$ through all the time. During protocol intervals, subjects remained awake but inactive, allowed only to perform quiet activities.

Oxygen and carbon dioxide volumes (VO_2 and VCO_2, respectively) readings were recorded every minute. The first 10 minutes (adaptation phase) and individual outlier values of those volumes were excluded. Means of VO_2 (L/min) and VCO_2 (L/min) from remaining data were used for calculations[25].

Total urinary nitrogen excretion was estimated from 12 hours urine collection before test drink consumption and 4 hour urine collection postprandially. Total volumes were noted and an aliquot was acidified with Timerozal® to prevent microorganism growth. Urine was analyzed for urea by colorimetric enzymatic kit (Bioclin®, Minas Gerais, Brazil) in automatic biochemical analyzer BS-200 (Mindray Medical International Ldt., Shenzen, China). Urinary nitrogen (N_2) was than calculated[26]. The result was divided by hours of urine collection and expressed as g/min.

REE, postprandial energy expenditure (kcal/min)[27] and substrate oxidation rates (carbohydrate, protein and fat oxidation rates)[26,28] were calculated using VO_2, VCO_2 and N_2 of each period of time. Values of non-protein respiratory quotient (NPRQ) were also calculated[28]. Diet-induced thermogenesis (DIT) was assessed[24] and expressed as percentage of test meal energy content. Incremental area under the curve (iAUC) of energy expenditure, fat and carbohydrate oxidation was calculated by trapezoidal method[29] to evaluate total response over time in both test days.

Subjective appetitive sensations

Visual analogue scale (VAS) of 100 mm were used to assess subjective appetite sensations[30] before, immediately after, and hourly for 4 h following standardized test meal (60, 120, 180 and 240 minutes).

These scales included words anchored at the left and right ends with opposing statements, expressing the most negative and positive ratings for each question. Subjects were instructed to rate appetite dimensions by indicating on the scale how they felt at the moment they completed the questions: Hunger ("How hungry do you feel?"), fullness ("How full do you feel?"), satiety ("How satisfied do you feel?"), and prospective consumption (How much do you think you can eat?). They could not refer to their previous ratings when answering questions.

Results were expressed as changes from the baseline value over postprandial period. Also incremental area under the curve (iAUC) was determined for fullness and satiety, and the incremental area above the curve (iAAC) was determined for hunger and prospective food consumption by trapezoidal method[29]. VAS were also used to rate the palatability of high-fat meals by the following questions: visual appeal, smell, taste, aftertaste, and palatability[30].

In order to evaluate if coconut oil was able to influence subsequent food consumption subjects were instructed to fill out a food diary in the intervention day from the moment they left laboratory until the next morning. Food record was reviewed individually by dietitian along with subjects to check for errors or omissions. Analysis of nutrient content was made using Dietpro software (Dietpro 5.2i, Agromidia Software Sistemas, Brazil) based on Brazilian Food Composition Table[31] and USDA National Nutrient Database for Standard Reference[32]. The amount of energy and macronutrient after standard lunch were evaluated as food intake later in the test day.

Statistical analysis

Statistical analyses were carried out on SPSS 20 for Windows (SPSS, Inc., Chicago, IL, USA). Data are expressed as mean ± standard error of the mean (SEM). Individual outlier values were excluded before analyses. Data normality and homoscedasticity were assessed by Shapiro-Wilk and Levene tests, respectively. Student's t-test or Mann-Whitney U signed-rank test were used to assess differences between baseline, 9 weeks and changes (Δ) values between two interventions days.

Changes values were calculated as values at 9 week – baseline values to verify differences between treatments due to dietary intervention. To assess intra-individual differences within each group over time, paired t test or Wilcoxon test were performed. A P-value of < 0.05 was considered significant.

Based on Mera et al[33] formula, published values of fasting fat oxidation and estimated change of 3.6g over postprandial state[25], a sample size of twelve subjects was needed for this study. To account for dropouts, twenty subjects were enrolled.

Results

Breakfasts were well tolerated by all study subjects and there were no report side effects. There were no differences between test drinks' palatability questions (P>0.050) and subjects' characteristics at baseline (Table 1). All subjects followed the prescribed diet and there were no differences in energy, carbohydrate, protein and total fat consumption between groups during the intervention period (article 2).

Table 1 - Subjects' baseline characteristics.

Characteristic	Control (n=20)	Coconut Oil (n=18)
Age (years)	27.2 ± 1.4	27.2 ± 1.5
BMI (kg/m^2)	29.8 ± 0.7	30.9 ± 0.8
Waist circumference (cm)	96.2 ± 1.5	96.3 ± 2.0
Fat mass (kg)	37.0 ± 1.5	36.5 ± 1.8
Body fat percentage (%)	46.6 ± 0.7	46.3 ± 1.1
Fat-free mass (kg)	42.6 ± 5.4	42.0 ± 1.7
REE (kcal/day)*	1,336 ± 31.9	1,369 ± 42.7
REE / fat-free mass (kcal/day)*	31.4 ± 0.7	32.2 ± 0.8

BMI: Body Mass Index; REE: Resting Energy Expenditure. Data are presented as mean ± SEM. There was no differences between groups (Student t test, P <0.050). *Data from 29 volunteers (15 allocated in control group and 14 allocated in coconut oil group).

Interestingly, after 9 weeks, fasting NPRQ and total fat oxidation increased and total carbohydrate oxidation and fasting fat oxidation decreased in control group. VCO did not increase resting or postprandial energy expenditure after intervention period. Also, there was no diet-induced thermogenesis increase after VCO consumption (Table 2). There was no difference between groups in changes (Δ) of any metabolic rates.

Table 2 - Fasting and postprandial energy expenditure and substrate oxidation rates of subjects consuming control (n = 15) or coconut oil (n = 14)

Meal induced change	**Control**				**Coconut oil**				
	Baseline	9 weeks	P_{intra}	**Δ**	**Baseline**	**9 weeks**	P_{intra}	**Δ**	P_{Δ}
EE (kcal/min)									
Fasting state	0.93 ± 0.02	0.93 ± 0.01	0.505	0.02 ± 0.01	0.95 ± 0.03	0.93 ± 0.02	0.106	-0.01 ± 0.01	0.100
Postprandial state	0.96 ± 0.02	0.94 ± 0.02	0.791	-0.01 ± 0.01	0.98 ± 0.03	0.95 ± 0.03	0.258	-0.02 ± 0.02	0.398
Total (iAUC)	3.93 ± 0.82	6.03 ± 1.17	0.433	-0.73 ± 0.90	6.24 ± 0.63	6.99 ± 1.40	0.509	-0.57 ± 1.83	0.939
NPRQ									
Fasting state	0.86 ± 0.01	0.90 ± 0.01	**0.024**	0.03 ± 0.01	0.88 ± 0.01	0.88 ± 0.01	0.924	0.01 ± 0.02	0.210
Postprandial state	0.82 ± 0.01	0.82 ± 0.01	0.951	- 0.01 ± 0.01	0.83 ± 0.01	0.82 ± 0.01	0.184	-0.01 ± 0.01	0.370
Carbohydrate oxidation (g/min)									
Fasting state	0.11± 0.01	0.12 ± 0.01	0.093	0.02 ± 0.01	0.11 ± 0.01	0.11 ± 0.01	0.441	0.01 ± 0.01	0.336
Postprandial state	0.09 ± 0.01	0.09 ± 0.01	0.992	0.01 ± 0.01	0.09 ± 0.01	0.08 ± 0.01	0.052	-0.01 ± 0.01	0.147
Total (iAAC)	-3.84 ± 0.69	-6.93 ± 0.92	**0.004**	-3.10 ± 0.90	-3.79 ± 0.06	-6.66 ± 1.44	0.167	- 1.64 ± 2.00	0.515
Fat oxidation (g/min)									
Fasting state	0.04 ± 0.01	0.03 ± 0.01	**0.020**	-0.01 ± 0.01	0.03 ± 0.01	0.03 ± 0.01	0.499	-0.01 ± 0.01	0.230
Postprandial state	0.05 ± 0.01	0.05 ± 0.01	0.974	0.01 ± 0.01	0.05 ± 0.01	0.05 ± 0.01	0.672	0.01 ± 0.01	0.915
Total (iAUC)	2.78 ± 0.18	3.84 ± 0.46	**0.041**	1.39 ± 0.46	2.78 ± 0.48	2.47 ± 0.48	0.604	0.03 ± 0.46	0.103
Protein oxidation (g/min)									
Fasting state	0.03 ± 0.01	0.03 ± 0.01	0.982	-0.01 ± 0.01	0.03 ± 0.01	0.03 ± 0.01	0.226	-0.01 ± 0.01	0.202
Postprandial state	0.04 ± 0.01	0.03 ± 0.01	0.093	-0.01 ± 0.01	0.04 ± 0.01	0.03 ± 0.01	0.613	-0.01 ± 0.01	0.914
Diet Induced Thermogenesis (% energy intake)	2.03 ± 0.23	1.77 ± 0.34	0.431	-0.22 ± 0.26	1.83 ± 0.18	2.02 ± 0.45	0.509	-0.17 ± 0.54	0.934

EE: energy expenditure; NPRQ: non-protein respiratory quotient. iAUC: incremental area under the curve; iAAC: incremental area above the curve. Data were expressed as mean ± SEM. P intra was analyzed by paired *t*-test between values at baseline and 9 weeks for each group. Changes (**Δ)** were calculated as values at 9 weeks – values at baseline and P_{Δ} was calculates by Student's t test. Bold type P values indicate significantly differences (P<0.050). Test drinks contained 25mL of virgin coconut oil (coconut oil) or soybean oil (control).

There was a reduction in prospective consumption sensation at baseline in response to VCO consumption (P=0.001), but this effect was not observed after 9 weeks of consumption. Comparing the baseline values with those at the end of intervention period in each group individually, prospective consumption sensation reduced in the control group (P_{intra} = 0.021). However, VCO intake resulted in increased total hunger sensation after 9 weeks (Figure 1). Despite subjective appetite sensation results, subsequent energy and macronutrients intake were not different between groups (Table 3).

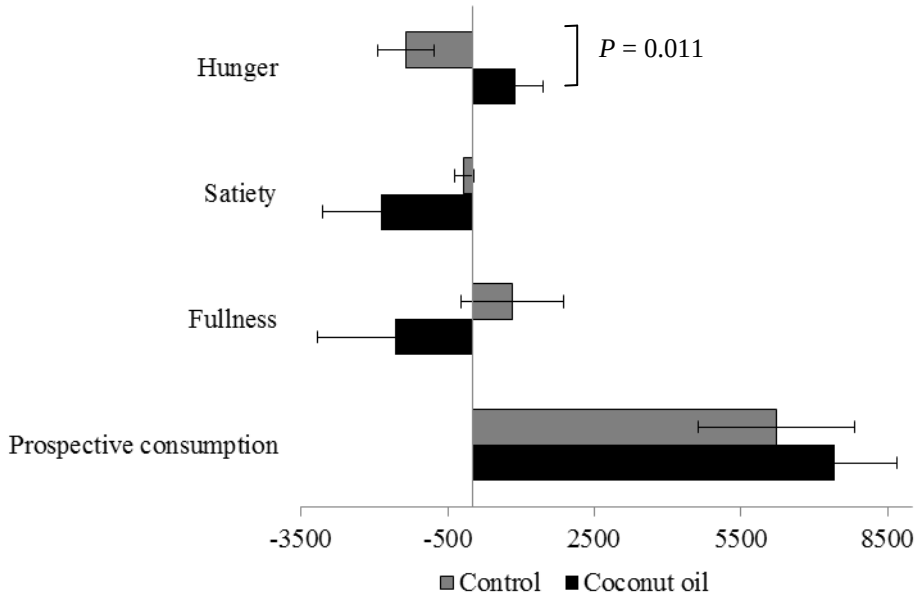

Figure 1 - Means ± SEM changes (Δ) in total subjective appetitive sensations after nine weeks of 25 mL of soybean oil (control) or virgin coconut oil (coconut oil) consumption.

Changes were calculated as 9 weeks values – baseline values. Total responses values for hunger and prospective consumption feeling were calculated as incremental area above the curve (iAAC). Total responses for satiety and fullness feelings were calculated as incremental area under the curve (iAUC). Δ = final values – initial values. * Student's *t* test, $P < 0.050$.

Table 3 - Changes from baseline in energy and macronutrient intake on subsequent meals after control or virgin coconut oil consumption after nine weeks.

	Control (n =10)	Coconut oil (n =10)	*P*
Δ Energy (kcal)	-27.0 ± 158.9	49.0 ± 129.4	0.714
Δ Carbohydrate (g)	4.8 ± 26.4	45.2 ± 9.6	0.210
(%E)	1.9 ± 3.2	3. 6 ± 5.1	0.780
Δ Protein (g)	9.7 ± 4.7	3.0 ± 11.1	0.586
(%E)	1.2 ± 2.9	1.0 ± 3.8	0.961
Δ Lipids (g)	-3.3 ± 4.9	-1.5 ± 4.8	0.797
(%E)	-9.5 ± 4.4	-0.94 ± 2.7	0.118

%E: Percentages of total energy intake. Changes (Δ) were calculated as values at 9 weeks – values at baseline. Values did not differ statistically by Student's t test ($P>0.050$). Test drinks contained 25mL of virgin coconut oil (coconut oil) or soybean oil (control).

Discussion

VCO has gained popularity due to its attributed health claims related to weight management[1] and has been widely considered as an adjuvant in weight loss treatment[34]. Despite containing more than 90% of saturated fatty acids, VCO is composed mainly by the MCFA lauric acid (C12:0), which differs coconut oil from others vegetables oils and were in part responsible for its attributed claims[2]. MCFA are known to be absorbed directly into the portal vein, transported rapidly to the liver where they are easily and rapidly oxidized[13]. For this reason, MCFA are less efficiently stored than other fatty acids and are unlikely to promote obesity via direct storage in adipocytes[35]. Hence, it has been proposed that VCO should be consumed by people who are attempting to control their weight.

The mechanisms by which coconut oil may affect energy balance are related to increased energy expenditure and/or to appetite control[4,12,19,36]. Several studies have demonstrated that synthetic MCFA oil (containing only caprylic – C8:0 and capric – C10:0 acids) but not VCO as a whole food, were able to increase energy expenditure, fat oxidation, and diet-induced thermogenesis[5–7,37,38], besides delaying and reducing subsequent food intake[11,16,38–40]. The propensity of these MCFA to be oxidized results in increased concentrations of acetyl-coenzyme A molecules into mitochondria and cytosol. Once oxidative capacity of Krebs cycle is exceeded, the glut of acetil-coA tends

to trigger protective uncoupling mechanisms enhancing thermogenesis[35]. Also, it stimulates the production of ketone bodies[13], which may be involved in appetite control after MCFA consumption[41].

On the other hand, our study demonstrated that daily consumption of reasonable amounts (25 mL) of VCO did not increase fasting nor postprandial energy and fat oxidation rates. This result is similar to the ones observed by the authors of few studies in which chronic coconut oil consumption failed to increase thermogenesis[15,42]. Surprisingly, fat oxidation increased after 9 weeks only in the control group receiving high PUFA-soybean oil. Hierarchy seems to exist between saturated and unsaturated fatty acids when consumed individually. Saturated fatty acids oxidation rates decrease with increasing chain length[43,44], while for unsaturated ones, oxidation decreases with increasing number of double bonds[44]. Comparing unsaturated to long-chain saturated fatty acids (>16 carbons), the former seems to be oxidized more rapidly[45] except for MCFA, which are oxidized faster than others[42]. For this reason and based in our results, it is clear that VCO behaved metabolically more as a long-chain fatty acid than MCFA.

Since VCO is predominantly composed of lauric acid (~50%), it is believed that the biological effects of this oil could be consequence of the presence of this fatty acid[46]. Animal studies[47,48] showed that lauric acid has a higher propensity to be absorbed by chylomicrons and consequently, its access to the liver is delayed, resulting in less prominent rise in ketone bodies[35]. Also, by following lymphatics pathway, lauric acid could be incorporated in adipose tissue[49], as no increase in fat oxidation was observed. For this reason, it has been suggested that the MCFA lauric acid potentially behave more like a LCFA explaining the absence of effects observed in the present and other studies[14,15].

Unexpectedly, our results showed increased hunger sensation after 9 weeks of VCO consumption. However, no alteration in subsequent food consumption was observed in obese women following energy restricted diet. Evaluation of subjective appetitive sensations failed to be different between MCFA and LCFA[11,17,39,50]. Indeed, increase in satiety due to MCFA intake has been indirectly demonstrated from studies that evaluated energy and macronutrient intake in subsequent meals after MCFA consumption. Results from these studies showed reduced food intake in ad libitum lunch[11,39] or for the rest of the day[10,16] following a rich-MCFA breakfast, although studies testing coconut oil intake[17,50] could not observe this effects. Our previews acute

interventional study showed less suppression of hunger sensation after 4 hours of VCO intake when compared to consumption of the monounsaturated fatty acid source extra-virgin olive oil[51]. Contradictorily, when compared to soybean oil at the same period of time, hunger and prospective food consumption were lower after coconut oil consumption (see article 2). This discrepancy in results depending on control oil must be further investigated to help explaining the increase in total hunger sensation observed after chronic intake of coconut oil.

To our knowledge, this is the first study to evaluate the effect of chronic consumption of VCO associated with energy restricted diet on metabolic rates and appetite sensations in obese women. The majority of the research on this topic has being conducted in non-obese men[7,37,52] or women[5,14,15] for periods less than 2 weeks. The lack of studies envolving obese subjects for longer periods of time and with coconut oil as a whole food suggest that coconut oil should be recommended with caution for obesity treatment.

Conclusion

Daily consumption of VCO for 9-weeks did not affect energy metabolism, fat oxidation rates, and food consumption but increased hunger sensation in obese women following energy restricted diet. Thus, changes in thermogenesis and appetite control do not seem to be the mechanisms responsible for weight loss associated with VCO consumption. So, we conclude that coconut oil does not have the potential to be an adjuvant in weight loss therapies and its use in clinical practice should not be related to weight management practices.

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4 CONCLUSÕES GERAIS

- O consumo do óleo de coco virgem (OCV) não foi melhorou a antropometria e composição corporal de mulheres obesas submetidas à restrição calórica.
- Apesar da leve alteração aguda da uricemia provocada pelo OCV, não encontramos diferenças, nos demais marcadores bioquímicos de risco cardiometabólico após nove semanas de intervenção.
- A diferença observada nas mudanças da concentração de HDL-c ao longo de nove semanas foi decorrente da redução destas concentrações no grupo controle, e não pelo aumento desta lipoproteína no grupo OCV;
- Ao contrário do sugerido pela hipótese inicial, o consumo de OCV não aumentou o gasto energético total, a oxidação de lipídios e nem a termogênese induzida pela dieta, avaliados no primeiro dia e após nove semanas de intervenção. Desta forma, os resultados sugerem a inexistência de efeitos termogênicos agudos e crônicos promovidos pelo OCV.
- Os efeitos do consumo do OCV nas sensações subjetivas de apetite sugerem que, seu consumo agudo reduz a fome e suprime a sensação subjetiva de consumo prospectivo. Porém, o consumo em longo prazo mostrou efeito inverso na sensação de fome, devendo, portanto este resultado ser avaliado com cautela.
- Na avaliação do consumo alimentar, não identificamos diferença no consumo de calorias e macronutrientes nas refeições subsequentes ao consumo da bebida teste nos dias de avaliação.
- Diante dos resultados obtidos, acreditamos que mais estudos sejam necessários para elucidar a questões não respondidas. Porém, no momento, desencorajamos a prescrição do óleo de coco como adjuvante no tratamento da obesidade.

5 PERSPECTIVAS FUTURAS

- Diante da ausência de efeitos do OCV na taxa metabólica, recomendamos o desenvolvimento de estudos que avaliem se há a incorporação dos ácidos graxos de cadeia média, principalmente o ácido láurico, no tecido adiposo das participantes e como este fato poderia influenciar a expressão de genes relacionados à obesidade.
- Devido a influência dos corpos cetônicos na saciedade associada ao consumo de OCV, a avaliação das concentrações plasmáticas destes compostos poderia auxiliar no entendimento dos resultados conflitantes nas sensações subjetivas de saciedade em curto e em longo prazo.
- Estudos experimentais contrapondo os efeitos do ácido láurico isolado com aqueles observados pelo consumo de óleos sintéticos formados somente pelos ácidos graxos de cadeia média caprílico e cáprico podem ser uteis para desmistificar o efeito atribuído ao óleo de coco na obesidade.

6. APÊNDICES

6.1 Apêndice 1 – Questionário de triagem pelo telefone

MINI-QUESTIONÁRIO DE TRIAGEM

Nome: _____

Data: ___/___/____

Idade (anos):_____Peso (kg): _____ Altura (m): _____ IMC: _____

(20 a 40 anos) (27 - 34,9 kg/m²)

Fumante: ( ) Sim ( ) Não Gravidez: ( ) Sim ( ) Não Lactação: ( ) Sim ( ) Não

Possui diagnóstico de alguma doença: ( ) Sim ( ) Não

(doenças cardiovasculares, diabetes mellitus 1 e 2, hipertensão arterial, distúrbios da tireoide, doenças hepáticas e gastrointestinais)

Uso de medicamentos? ( ) Sim ( ) Não

Quais?_____

Agendada para próxima etapa: ( ) Sim ( ) Não Dia: ___/___/____

Telefones de contato:_____

Responsável: _____

6.2 Apêndice 2 – Questionário de Triagem

Data: ____/____/____

Iniciais:	**ID da voluntária:**
Responsável:	

DADOS PESSOAIS:

Data de nascimento: ___/__/___ Sexo: ( ) Masculino ( ) Feminino

Telefones de contato: _____

E-mail: _____

Escolaridade (anos de estudo):_____

Estado civil: Número de filhos:

Você tem disponibilidade para vir todos os dias ao laboratório para o café da manhã? **( ) Sim** ( ) Não

HISTÓRIA CLÍNICA:

Você tem ou já teve alguma das doenças indicadas a seguir?
( ) Tireoide (hipo ou hipertireoidismo, câncer)
( ) Síndrome do ovário policístico
( ) Problema nos rins
( ) Doença do fígado
( ) Doença ou na vesícula ou retirada
( ) Doenças intestinais (Doença Celíaca, Diverticulite, Doença de Crhon, Síndrome de intestino irritável ou outra)
( ) Transtornos alimentares (anorexia, bulimia, compulsão alimentar)
( ) Doença psiquiátrica diagnosticada (depressão, distúrbio de ansiedade generalizado, distúrbio do pânico, transtorno bipolar, esquizofrenia)
( ) Colesterol alto
( ) Triglicerídeos alto
( ) Diabetes
( ) Pressão alta
( ) Câncer
( ) Outras Quais _____

Você toma algum medicamento? (anticoncepcional, remédio para cólica)
( ) Sim ( ) Não Quais e em que dose?_____
_____

Você já realizou algum tipo de cirurgia com anestesia? (dental, lipoaspiração)
( ) Sim ( ) Não Quais?_____

Você já teve ou sabe se tem reação a algum tipo de anestésico?
( ) Sim ( ) Não Quais?_____

Você se importaria em fazer um pequeno procedimento para retirar um pouquinho de gordura da barriga com agulha? ( ) Sim ( ) Não

Sua menstruação veio regularmente, sem alterações de fluxo e data, nos últimos 3 meses?
( ) Sim ( ) Não DUM: ____/____/_____
Você usa suplementos ou vitaminas? (cápsulas de óleos, vitamina C, polivitaríminicos - centrum)

( ) Sim ( ) Não Quais?_____

Você toma algum medicamento ou chá para emagrecer?
( ) Sim ( ) Não Quais e em que doses?_____

Você toma algum medicamento para reposição de hormônios?
Dosagens?_____

Você considera que seu intestino funciona bem ou é preguiçoso?_____
Com qual frequência vai ao banheiro evacuar?_____

Você pratica algum tipo de atividade física regularmente? ( ) Sim ( ) Não
Qual e frequência? _____

Você aumentou ou diminuiu seu nível de atividade física nos últimos meses?
( ) Sim ( ) Não ( ) Aumentou ( ) Diminuiu
Mudança: _____

Há quanto tempo você tem excesso de peso? _____

Você já está fazendo alguma dieta para perder peso? ( ) Sim ( ) Não
Qual? _____ Duração: _____

Você é vegetariano? ( ) Sim ( ) Não

Nos últimos 3 meses você :
Ganhou peso: ( ) Sim ( ) Não
Perdeu peso: ( ) Sim ( ) Não Quantos quilos?_____

HABITOS ALIMENTARES:

Você tem alergia alimentar? (principalmente leite e derivados, corantes, frutos do mar, castanhas, amendoim, soja)
( ) Sim ( ) Não A qual alimento?_____

Você tem aversão alimentar? (Principalmente a coco, castanha, soja, amendoim, frutos do mar, azeite de oliva ou alguma fruta, legume ou vegetal)
( ) Sim ( ) Não A qual alimento?_____

Você sente dores de cabeças, náuseas, tem diarreia ou gases quando come algum alimento específico? (Principalmente frituras, queijos amarelos e massa com molho branco)
( ) Sim ( ) Não A qual alimento?_____

Você não gosta do sabor de alguma dessas bebidas a seguir: (vitamina)
( ) Shake de maracujá ( ) Shake de morango ( ) Shake de goiaba ( ) Shake de manga
( ) Chocolate quente ( ) Capuccino ( ) Shake de uva

Você consome bebida alcoólica? ( ) Sim ( ) Não
Se sim: qual tipo: _____ com que frequência?-_____ Quantidade?_____
Você consome café, refrigerante tipo cola, chás? ( ) Sim ( ) Não
Se sim, com que frequência?_____ Quantidade?_____

Você consome algum tipo de adoçante? ( ) Sim ( ) Não
Quais preparações e quantidade:_____
Qual frequencia?_____
Qual adoçante você usa? _____
Qual óleo você utiliza para o preparo de suas refeições? _____

7. ANEXOS

7.1 Anexo 1: Questionário de 3 fatores

1- Quando eu sinto o cheiro de um bife fritando, ou vejo um pedaço suculento de carne, eu encontro muita dificuldade para não comê-lo, se eu tiver acabado de fazer uma refeição.	V	F
2- Eu geralmente como muito em ocasiões sociais, gosto de festas e picnics.	V	F
3- Eu geralmente estou faminto por isso como mais de três vezes por dia.	V	F
4- Quando eu como minha cota de calorias, eu normalmente me sinto bem em não comer mais nada	V	F
5- Fazer dieta é muito difícil para mim porque sinto muita fome.	V	F
6- Eu intencionalmente como pequenas refeições para ajudar no controle do meu peso	V	F
7- Às vezes, alguns alimentos têm sabor tão bom que consigo comer mesmo quando não estou com fome	V	F
8- Visto que estou sempre com fome, às vezes desejo que enquanto estou comendo, um especialista me diga se comi o suficiente ou se poderia comer mais alguma coisa.	V	F
9- Quando estou ansioso (a), costumo comer mais do que normalmente como.	V	F
10- A vida é muito curta para perdê-la fazendo dieta.	V	F
11- Quando meu peso aumenta ou diminui, faço dieta	V	F
12- Sempre que sinto muita fome tenho que comer alguma coisa.	V	F
13- Quando estou com alguém que come muito, eu também como muito.	V	F
14- Eu tenho uma boa noção de quantas calorias têm os alimentos mais comuns.	V	F
15- Às vezes, quando eu começo a comer, não consigo parar.	V	F
16- Não é difícil para eu deixar resto no prato.	V	F
17- Em determinados horários do dia, eu fico com fome porque tenho o hábito de comer nesses horários	V	F
18- Quando estou fazendo dieta, se eu como algo que não é permitido, eu intencionalmente como menos por um período de tempo para compensar.	V	F
19- Quando estou com alguém que está comendo, as vezes sinto fome suficiente para comer também	V	F
20- Quando me sinto deprimido, eu sempre como muito	V	F
21- Eu divirto comendo muito e fico deprimido contando calorias ou vigiando meu peso.	V	F
22- Quando eu vejo uma guloseima, eu freqüentemente fico com fome e tenho que comer imediatamente.	V	F
23- Eu freqüentemente paro de comer antes de estar completamente cheio, como forma consciente de limitar a quantidade de comida ingerida.	V	F
24- Eu sinto tanta fome que meu estômago, freqüentemente, parece um buraco sem fundo.	V	F
25- Meu peso mudou pouco durante os últimos 10 anos.	V	F
26- Eu estou sempre faminta, por isso é difícil para eu parar de comer antes de acabar a comida no meu prato.	V	F
27- Quando eu me sinto sozinha, eu me consolo comendo.	V	F
28- Eu conscientemente vomito uma refeição com objetivo de não ganhar peso.	V	F
29-Eu, algumas vezes, tenho muita fome pela tarde ou à noite.	V	F
30- Eu como qualquer coisa que quero, quando eu quero.	V	F
31- Sem pensar em comida, eu agüento ficar muito tempo sem comer.	V	F
32- Eu conto calorias como meio consciente de controlar meu peso.	V	F
33- Eu não como alguns alimentos porque eles podem me engordar.	V	F
34- Eu estou sempre com fome o suficiente para comer por muito tempo.	V	F
35- Eu presto muita atenção às mudanças no meu corpo.	V	F
36- Enquanto estou fazendo dieta, se eu como um alimento que não é permitido, eu, muitas vezes, como outros alimentos com elevado teor calórico.	V	F

PARTE 1

PARTE 2 - Por favor responda as seguintes questões marcando um "x" na resposta apropriada para você.

37- Com que freqüência você faz dieta com intenção de controlar seu peso?

1 - raramente 2 - algumas vezes 3 - freqüentemente 4 - sempre

38- Poderia a flutuação de peso de 2.kg afetar a maneira como você vive sua vida?

1 - não totalmente 2 - pouco 3 - moderadamente 4 - muito

39- Qual a freqüência que você sente fome?

1 - somente na hora das refeições | 2 - algumas vezes entre as refeições | 3 - freqüentemente entre as refeições | 4 - quase sempre

40- Sua sensação de culpa por comer muito ajuda você a controlar sua ingestão de alimentos?

1 - nunca | 2 - raramente | 3 - freqüentemente | 4 - sempre

41- Quão difícil seria para você parar de comer a meio caminho de terminar o jantar e ficar sem comer nas próximas quatro horas?

1 - fácil | 2 - pouco difícil | 3 - moderadamente difícil | 4 - muito difícil

42- Você tem consciência sobre o que você está comendo?

1 - não totalmente | 2 - pouco | 3 - moderadamente | 4 - extremamente

43- Qual a freqüência que você tem resistido a alimentos tentadores?

1 - quase nunca | 2 - raramente | 3 - freqüentemente | 4 - quase sempre

44- Qual a probabilidade de você comprar alimentos de baixa caloria?

1 - improvável | 2 - pouco provável | 3 - moderadamente provável | 4 - muito provável

45- Você come moderadamente diante de outros e sozinho come grande quantidade de alimentos?

1 - nunca | 2 - raramente | 3 - freqüentemente | 4 - sempre

46- Qual a probabilidade de você, conscientemente, comer lentamente com objetivo de reduzir o quanto você come?

1 - improvável | 2 - pouco provável | 3 - moderadamente provável | 4 - muito provável

47- Com qual freqüência você dispensa uma sobremesa porque você já está satisfeita?

1 - quase nunca | 2 - raramente | 3 - no mínimo uma vez por semana | 4 - quase todo dia

48- Qual a probabilidade de você comer conscientemente menos do que você quer?

1 - improvável | 2 - pouco provável | 3 - moderadamente provável | 4 - muito provável

49- Você costuma comer mesmo sem estar com fome?

1 - nunca | 2 - raramente | 3 - Algumas vezes | 4 - ao menos uma vez por semana

50- Na escala de 0 a 5, onde 0 quer dizer sem restrição alimentar (comer tudo que você quer, sempre que você quer) e 5 significa restrição total (limita constantemente a ingestão de alimentos e nunca cede) qual o número você poderia dar para você mesmo?

0 - Come tudo que você quer, quando que você quer
1 - Freqüentemente come tudo que você quer, quando você quer
2 - Muitas vezes come tudo que você quer, Quando você quer
3 - Muitas vezes limita ingestão de alimentos, mas freqüentemente cede
4 - Freqüentemente limita ingestão de alimentos, mas raramente cede
5 - Constantemente limita ingestão de alimentos, nunca cede

51- Até que ponto esta declaração descreve seu comportamento alimentar? Eu começo fazer dieta pela manhã, mas devido algum número de coisas que acontecem durante o dia, pela tarde eu me rendo e como o que eu quero e prometo a mim mesma (o) começar, novamente, a dieta amanhã.

1 - não parece comigo | 2 - parece um pouco comigo | 3 - me descreve muito bem | 4 - me descreve perfeitamente

7.2 Anexo 2: QUESTIONÁRIO INTERNACIONAL DE ATIVIDADE FÍSICA (IPAQ)

Nome:_____Data: ___/ ___ / ___ Idade : ____
Sexo: F ( ) M ( ).
Quantas horas você trabalha por dia: ____ Quantos anos completos você estudou: _____

Nós estamos interessados em saber que tipos de atividade física as pessoas fazem como parte do seu dia a dia. Este projeto faz parte de um grande estudo que está sendo feito em diferentes países ao redor do mundo. Suas respostas nos ajudarão a entender que tão ativos nós somos em relação à pessoas de outros países. As perguntas estão relacionadas ao tempo que você gasta fazendo atividade física em uma semana **ULTIMA SEMANA**. As perguntas incluem as atividades que você faz no trabalho, para ir de um lugar a outro, por lazer, por esporte, por exercício ou como parte das suas atividades em casa ou no jardim. Suas respostas são MUITO importantes. Por favor, responda cada questão mesmo que considere que não seja ativo. Obrigado pela sua participação!

Para responder as questões lembre que:

- Atividades físicas VIGOROSAS são aquelas que precisam de um grande esforço físico e que fazem respirar MUITO mais forte que o normal
- Atividades físicas MODERADAS são aquelas que precisam de algum esforço físico e que fazem respirar UM POUCO mais forte que o normal

SEÇÃO 1-
ATIVIDADE FÍSICA NO TRABALHO

Esta seção inclui as atividades que você faz no seu serviço, que incluem trabalho remunerado ou voluntário, as atividades na escola ou faculdade e outro tipo de trabalho não remunerado fora da sua casa. **NÃO** incluir trabalho não remunerado que você faz na sua casa como tarefas domésticas, cuidar do jardim e da casa ou tomar conta da sua família. Estas serão incluídas na seção 3.

1a. Atualmente você trabalha ou faz trabalho voluntário fora de sua casa?
( ) Sim ( ) Não – Caso você responda não **Vá para seção 2: Transporte**
As próximas questões são em relação a toda a atividade física que você fez na **ultima semana** como parte do seu trabalho remunerado ou não remunerado. **NÃO** inclua o transporte para o trabalho.P ense unicamente nas atividades que você faz por **pelo menos 10 minutos contínuos**:

1b. Em quantos dias de uma semana normal você **anda,** durante **pelo menos 10 minutos contínuos**, **como parte do seu trabalho**? Por favor, **NÃO** inclua o andar como forma de transporte para ir ou voltar do trabalho.
_____dias por **SEMANA**
( ) nenhum - **Vá para a seção 2 - Transporte**.

1c. Quanto tempo no total você usualmente gasta **POR DIA** caminhando **como parte do seu trabalho** ?
____ horas _____ minutos

1d. Em quantos dias de uma semana normal você faz atividades **moderadas,** por **pelo menos 10 minutos contínuos,** como carregar pesos leves **como parte do seu trabalho**?
_____dias por **SEMANA**
( ) nenhum - **Vá para a questão 1f**

1e. Quanto tempo no total você usualmente gasta **POR DIA** fazendo atividades moderadas **como parte do seu trabalho**?
_____ horas _____ minutos

1f. Em quantos dias de uma semana normal você gasta fazendo atividades **vigorosas,** por **pelo menos 10 minutos contínuos,** como trabalho de construção pesada, carregar grandespesos, trabalhar com enxada, escavar ou subir escadas **como parte do seu trabalho:**
_____dias por **SEMANA** ( ) nenhum - **Vá para a questão 2a.**

1g. Quanto tempo no total você usualmente gasta **POR DIA** fazendo atividades físicas vigorosas **como parte do seu trabalho**?
_____ horas _____ minutos

SEÇÃO 2 –
ATIVIDADE FÍSICA COMO MEIO DE TRANSPORTE

Estas questões se referem à forma típica como você se desloca de um lugar para outro, incluindo
seu trabalho, escola, cinema, lojas e outros.

2a. O quanto você andou na ultima semana de carro, ônibus, metrô ou trem?
_____dias por **SEMANA**
( ) nenhum - **Vá para questão 2c**

2b. Quanto tempo no total você usualmente gasta **POR DIA andando de carro, ônibus, metrôou trem**?
_____horas _____minutos

Agora pense **somente** em relação a caminhar ou pedalar para ir de um lugar a outro na ultima semana.

2c. Em quantos dias da ultima semana você andou de bicicleta por **pelo menos 10 minutos contínuos** para ir de um lugar para outro? (**NÃO** inclua o pedalar por lazer ou exercício)
_____ dias por **SEMANA**
( ) Nenhum - **Vá para a questão 2e**.

2d. Nos dias que você pedala quanto tempo no total você pedala **POR DIA** para ir de um lugar para outro?
_____ horas _____ minutos

2e. Em quantos dias da ultima semana você caminhou por **pelo menos 10 minutos contínuos**
para ir de um lugar para outro? (**NÃO** inclua as caminhadas por lazer ou exercício)
_____ dias por **SEMANA**
( ) Nenhum - **Vá para a Seção 3**.

2f. Quando você caminha para ir de um lugar para outro quanto tempo **POR DIA** você gasta? (**NÃO** inclua as caminhadas por lazer ou exercício)
_____ horas _____ minutos

SEÇÃO 3 –
ATIVIDADE FÍSICA EM CASA: TRABALHO, TAREFAS DOMÉSTICAS E CUIDAR DA FAMÍLIA

Esta parte inclui as atividades físicas que você fez na ultima semana na sua casa e ao redor da sua casa, por exemplo, trabalho em casa, cuidar do jardim, cuidar do quintal, trabalho de manutenção da casa ou para cuidar da sua família. Novamente pense ***somente*** naquelas atividades físicas que você faz **por pelo menos 10 minutos contínuos**.

3a. Em quantos dias da ultima semana você fez atividades **moderadas** por pelo menos 10 minutos como carregar pesos leves, limpar vidros, varrer, rastelar **no jardim ou quintal.**
_____dias por **SEMANA**
( ) Nenhum - **Vá para questão 3b**.

3b. Nos dias que você faz este tipo de atividades quanto tempo no total você gasta **POR DIA** fazendo essas atividades moderadas **no jardim ou no quintal**?
_____ horas _____ minutos

3c. Em quantos dias da ultima semana você fez atividades **moderadas** por pelo menos 10 minutos como carregar pesos leves, limpar vidros, varrer ou limpar o chão **dentro da sua casa**.
_____ dias por **SEMANA**
( ) Nenhum - **Vá para questão 3d.**

3d. Nos dias que você faz este tipo de atividades moderadas **dentro da sua casa** quanto tempo no total você gasta **POR DIA**?
_____ horas _____ minutos

3e. Em quantos dias da ultima semana você fez atividades físicas **vigorosas no jardim ou quintal** por pelo menos 10 minutos como carpir, lavar o quintal, esfregar o chão:
_____ dias por **SEMANA**
( ) Nenhum - **Vá para a seção 4.**

3f. Nos dias que você faz este tipo de atividades vigorosas **no quintal ou jardim** quanto tempo no total você gasta **POR DIA**?

_____ horas _____ minutos

SEÇÃO 4 -
ATIVIDADES FÍSICAS DE RECREAÇÃO, ESPORTE, EXERCÍCIO E DE LAZER.

Esta seção se refere às atividades físicas que você fez na ultima semana unicamente por recreação,
esporte, exercício ou lazer. Novamente pense somente nas atividades físicas que faz **por pelo menos 10 minutos contínuos**. Por favor, **NÃO** inclua atividades que você já tenha citado.

4a. **Sem contar qualquer caminhada que você tenha citado anteriormente**, em quantos dias da
ultima semana você caminhou **por pelo menos 10 minutos contínuos no seu tempo livre**?
_____ dias por **SEMANA**
( ) Nenhum - **Vá para questão 4b**

4b. Nos dias em que você caminha **no seu tempo livre**, quanto tempo no total você gasta **POR DIA**?
_____ horas _____ minutos

4c. Em quantos dias da ultima semana você fez atividades **moderadas no seu tempo livre** por pelo menos 10 minutos, como pedalar ou nadar a velocidade regular, jogar bola, vôlei, basquete, tênis :
_____ dias por **SEMANA**
( ) Nenhum - **Vá para questão 4d.**

4d. Nos dias em que você faz estas atividades moderadas **no seu tempo livre** quanto tempo no
total você gasta **POR DIA**?
_____ horas _____ minutos

4e. Em quantos dias da ultima semana você fez atividades **vigorosas no seu tempo livre** por pelo menos 10 minutos, como correr, fazer aeróbicos, nadar rápido, pedalar rápido ou fazer Jogging:
_____ dias por **SEMANA**
( ) Nenhum - **Vá para seção 5.**

4f. Nos dias em que você faz estas atividades vigorosas **no seu tempo livre** quanto tempo no total você gasta **POR DIA**?
_____ horas _____ minutos

SEÇÃO 5 –
TEMPO GASTO SENTADO

Estas últimas questões são sobre o tempo que você permanece sentado todo dia, no trabalho, na escola ou faculdade, em casa e durante seu tempo livre. Isto inclui o tempo sentado estudando, sentado enquanto descansa, fazendo lição de casa visitando um amigo, lendo, sentado ou deitado assistindo TV. Não inclua o tempo gasto sentando durante o transporte em ônibus, trem, metrô ou carro.

5a. Quanto tempo no total você gasta sentado durante um **dia de semana**?
_____horas ____minutos

5b. Quanto tempo no total você gasta sentado durante em um **dia de final de semana**?
_____horas ____minutos

CENTRO COORDENADOR DO IPAQ NO BRASIL– CELAFISCS - INFORMAÇÕES ANÁLISE, CLASSIFICAÇÃO E COMPARAÇÃO DE RESULTADOS NO BRASIL
011-42298980 ou 42299643.
celafiscs@celafiscs.com.br
www.celafiscs.com.br IPAQ Internacional:
www.ipaq.ki.se

7.3 Anexo 3: CLASSIFICAÇÃO DO NÍVEL DE ATIVIDADE FÍSICA IPAQ

1. MUITO ATIVO: aquele que cumpriu as recomendações de:
a) VIGOROSA: ≥ 5 dias/sem e ≥ 30 minutos por sessão
b) VIGOROSA: ≥ 3 dias/sem e ≥ 20 minutos por sessão + MODERADA e/ou CAMINHADA: ≥ 5 dias/sem e ≥ 30 minutos por sessão.

2. ATIVO: aquele que cumpriu as recomendações de:
a) VIGOROSA: ≥ 3 dias/sem e ≥ 20 minutos por sessão; **ou**
b) MODERADA ou CAMINHADA: ≥ 5 dias/sem e ≥ 30 minutos por sessão; ou
c) Qualquer atividade somada: ≥ 5 dias/sem e ≥ 150 minutos/sem (caminhada + moderada + vigorosa).

3. IRREGULARMENTE ATIVO: aquele que realiza atividade física porém insuficiente para ser classificado como ativo pois não cumpre as recomendações quanto à freqüência ou duração. Para realizar essa classificação soma-se a freqüência e a duração dos diferentes tipos de atividades (caminhada + moderada + vigorosa). Este grupo foi dividido em dois sub-grupos de acordo com o cumprimento ou não de alguns dos critérios de recomendação:

IRREGULARMENTE ATIVO A: aquele que atinge pelo menos um dos critérios da recomendação quanto à freqüência ou quanto à duração da atividade:
a) Freqüência: 5 dias /semana **ou**
b) Duração: 150 min / semana

IRREGULARMENTE ATIVO B: aquele que não atingiu nenhum dos critérios da recomendação quanto à freqüência nem quanto à duração.

4. SEDENTÁRIO: aquele que não realizou nenhuma atividade física por pelo menos 10 minutos
contínuos durante a semana.

Exemplos:

Indivíduos	**Caminhada**	**Classificação**
1	Sedentário	
2	-	Irregularmente Ativo A
3		
4	Ativo	
5	Ativo	
6	Muito Ativo	
7	Muito Ativo	

7.4 Anexo 4: VAS para análise das sensações subjetivas do apetite

VAS **ID: _____ Tempo: _____ Data:_____ Horário:_____** Favor marcar nas escalas abaixo o que melhor reflete a sua resposta para cada uma das questões:		
Estou sem fome alguma	**Como está sua fome agora?** _____	**Eu nunca estive com tanta fome**
Eu estou completamente vazio	**Quão satisfeito você se sente agora?** _____	**Não aguento comer mais nada**
Nenhum pouco cheio	**Quão saciado (cheio) você se sente agora?** _____	**Completamente cheio**
Nada	**Quanto você acha que comeria agora?** _____	**Muito**
Sim, muito	**Você gostaria de comer alguma coisa doce agora?** _____	**Não, nenhum alimento doce**
Sim, muito	**Você gostaria de comer alguma coisa salgada agora?** _____	**Não, nenhum alimento salgado**
Sim, muito	**Você gostaria de beliscar algum alimento agora?** _____	**Não, nenhum**
Sim, muito	**Você gostaria de comer alguma coisa gordurosa agora?** _____	**Não, nenhum alimento gorduroso.**

7.5 Anexo 5: VAS para análise da palatabilidade das bebidas teste

VAS **ID: _____ Tempo: _____ Data:_____ Horário:_____** Favor marcar nas escalas abaixo o que melhor reflete a sua resposta para cada uma das questões:		
Ruim	**Aparência Visual** _____	**Boa**
Ruim	**Aroma (cheiro)** _____	**Boa**
Ruim	**Sabor** _____	**Bom**
Nenhum	**Gosto Residual** _____	**Muito**
Ruim	**Palatabilidade** _____	**Boa**

7.6 Anexo 6: Parecer do Comitê de Ética em Pesquisa com Seres Humanos

UNIVERSIDADE FEDERAL DE VIÇOSA - UFV

PARECER CONSUBSTANCIADO DO CEP

DADOS DO PROJETO DE PESQUISA

Título da Pesquisa: Efeito do consumo de diferentes fontes lipídicas associadas à dieta hipocalórica no controle do excesso de peso corporal

Pesquisador: MARIA DO CARMO GOUVEIA PELUZIO

Área Temática:

Versão: 2

CAAE: 37558514.5.0000.5153

Instituição Proponente: Departamento de Nutrição e Saúde

Patrocinador Principal: FUNDACAO DE AMPARO A PESQUISA DO ESTADO DE MINAS GERAIS

DADOS DO PARECER

Número do Parecer: 892.467

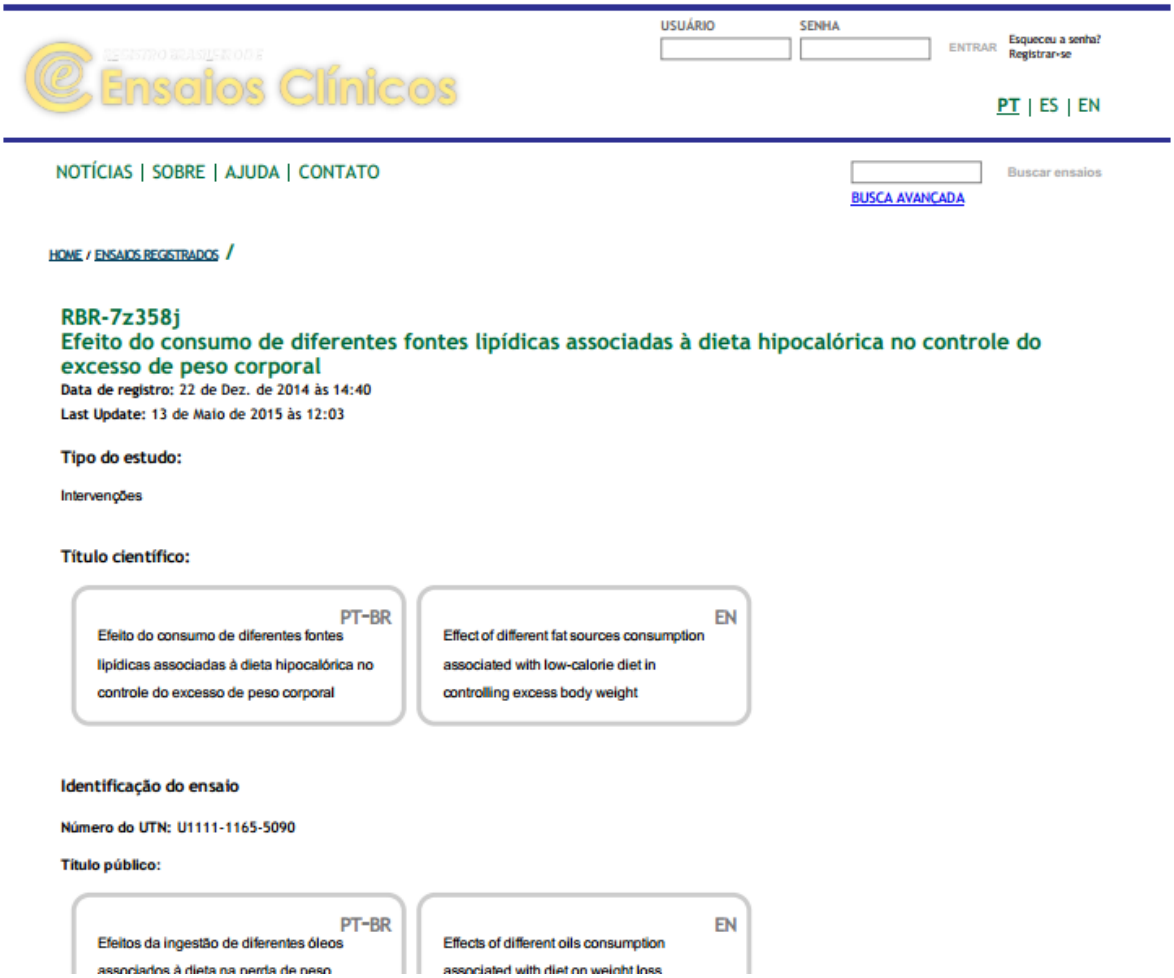
Data da Relatoria: 09/12/2014

Apresentação do Projeto:

O projeto PB_352501 trata-se de um projeto guarda-chuva, do qual podem originar outros projetos que utilizarão a mesma base de dados. O projeto propõe a realização de um ensaio clínico randomizado, duplo cego, com duração de 12 semanas consecutivas, que envolverá 66 mulheres de 20 a 40 anos de idade, as quais serão alocadas aleatoriamente em três grupos experimentais com dieta hipocalórica (-500kcal/dia)(n=22): óleo de coco (OC), Azeite de Oliva (AO) e óleo de soja (OS - controle). O tamanho amostral foi calculado conforme proposto por Mera et al (1998), adotando-se uma diferença de 10% no peso corporal. A escolha do peso corporal como variável principal baseou-se no impacto que esse indicador exerce nas complicações da obesidade. A diferença de 10% foi adotada já que, o consenso da SociedadEspañola para El Estudio de La Obesidad– SEEDO (SEEDO, 2007) recomenda como intervenção terapêutica a perda de 10% do peso corporal para indivíduos com IMC de 27 – 34,9 kg/m2 para que haja redução da morbidade e mortalidade nesta população, e percentual de gordura corporal maior que 30%. Ao primeiro contato com as possíveis voluntárias, será aplicado um mini questionário contendo perguntas sobre idade, peso habitual e estatura, para cálculo do IMC, hábito de fumar, histórico de doenças, gestação ou lactação, avaliando-se assim, se as voluntárias encontram-se dentro do perfil desejado. Caso atendam aoscritérios iniciais, as mesmas serão encaminhadas para a

Endereço: Universidade Federal de Viçosa, Edifício Arthur Bernardes, piso inferior
Bairro: Campus Universitário **CEP:** 36.570-900
UF: MG **Município:** VICOSA
Telefone: (31)3899-2492 **Fax:** (31)3899-2492 **E-mail:** cep@ufv.br

7.7 Anexo 7: Registro Brasileiro de Ensaios Clínicos



USUÁRIO SENHA ENTRAR Esqueceu a senha? Registrar-se

Ensaios Clínicos

PT | ES | EN

NOTÍCIAS | SOBRE | AJUDA | CONTATO

Buscar ensaios

BUSCA AVANÇADA

HOME / ENSAIOS REGISTRADOS /

RBR-7z358j

Efeito do consumo de diferentes fontes lipídicas associadas à dieta hipocalórica no controle do excesso de peso corporal

Data de registro: 22 de Dez. de 2014 às 14:40

Last Update: 13 de Maio de 2015 às 12:03

Tipo do estudo:

Intervenções

Título científico:

PT-BR

Efeito do consumo de diferentes fontes lipídicas associadas à dieta hipocalórica no controle do excesso de peso corporal

EN

Effect of different fat sources consumption associated with low-calorie diet in controlling excess body weight

Identificação do ensaio

Número do UTN: U1111-1165-5090

Título público:

PT-BR

Efeitos da ingestão de diferentes óleos associados à dieta na perda de peso

EN

Effects of different oils consumption associated with diet on weight loss

7.8 Anexo 8: TCLE

Universidade Federal de Viçosa – UFV
Centro de Ciências Biológicas e da Saúde
Departamento de Nutrição e Saúde

TERMO DE CONSENTIMENTO LIVRE ESCLARECIDO

Convidamos você a participar do estudo "EFEITO DO CONSUMO DE DIFERENTES FONTES LIPÍDICAS ASSOCIADAS À DIETA HIPOCALÓRICA NO CONTROLE DO EXCESSO DE PESO CORPORAL", cujo objetivo é conhecer a resposta metabólica, inflamatória, da saciedade e da microbiota intestinal frente a diferentes óleos.

Para tal, você deverá comparecer por quatro vezes no Laboratório de Metabolismo Energético e Composição Corporal (LAMECC), no Departamento de Nutrição e Saúde da Universidade Federal de Viçosa. Em todas as visitas serão realizadas aplicação de um questionário, medidas de peso, altura, perímetro da cintura e quantidade de gordura corporal, além da avaliação do metabolismo energético e exame de sangue. Nestas visitas, será oferecido a você um café da manhã, que deverá ser consumido no LAMECC. Após tomar o café da manhã, você deverá permanecer conosco por mais 3 horas para avaliação do metabolismo energético. Ainda, será pedido que você nos entregue amostras de fezes e urina que você coletará na sua casa.

Além disso, iremos prescrever uma dieta para perda de peso e fornecer o óleo que estamos avaliando, junto com várias receitas, sem custo algum. Você deverá consumir este óleo todos os dias, na sua casa, por um período de 3 meses sem interrupções. Antes de iniciar e quando finalizar o período de acompanhamento, o cirurgião da nossa equipe irá realizar um pequeno procedimento para retirar 4g de gordura da sua barriga, sob efeito de anestesia local, no Hospital São Sebastião.

Quanto aos riscos do estudo, a extração de sangue pode ser dolorosa e causar hematomas (roxo) no local da punção (picada), como qualquer outra coleta de sangue que você possa ter feito no passado. Para minimizar, contamos com um profissional especializado e treinado neste procedimento, que tomará todas as precauções possíveis para que você não sinta nada. Além disso, toda a coleta será realizada com matérias descartáveis para que não haja risco de contaminação.

A retirada de uma pequena amostra de gordura da barriga só causará dor na hora da picada da anestesia e o local poderá ficar roxo. Para minimizar, o nosso cirurgião fará o procedimento de forma bem lenta e de acordo com a tolerância de cada um. Informações sobre os cuidados a serem tomados depois do procedimento também serão oferecidas. Todo o material utilizado será descartável

Os questionários serão aplicados em local reservado e de modo individual. Caso você se sinta constrangido com alguma pergunta, você pode optar por não respondê-la e passar para a seguinte.

Para a coleta de fezes e urina, serão fornecidos coletores descartáveis e recipientes opacos para armazenamento, evitando assim a visualização do conteúdo e possíveis constrangimentos.

As outras medidas não causam risco em potencial, pois são técnicas não invasivas. Para minimizar qualquer risco e/ou desconforto, a coleta de sangue e demais medições serão realizadas por profissionais treinados, em ambiente tranquilo e adequado, utilizando-se de

técnicas padronizadas e preconizada na literatura científica. Além disso, você poderá se recusar a participar de qualquer etapa do estudo caso não se sinta confortável, sem que isso acarrete nenhum problema para você.

Você receberá um relatório dos resultados da avaliação nutricional e dos exames bioquímicos realizados com as devidas orientações nutricionais e, ou, encaminhamento para seu médico, caso necessário.

As amostras e questionários coletados no presente estudo serão guardados e utilizados em 02 projetos de doutorado e 02 de mestrado que abordarão os temas: gasto energético, sensação de fome e saciedade, metabolismo do tecido adiposo, permeabilidade e microbiota intestinal, estresse oxidativo e perfil inflamatório. Os resultados destes estudo serão apresentados, comunicados e/ou publicados no meio científico, mas sempre preservando sua confidencialidade e privacidade.

Você não terá nenhum gasto por sua participação nesse estudo, ao mesmo tempo em que não receberá nenhum tipo de remuneração. Você poderá se recusar a participar ou sair do estudo a qualquer momento depois de dar o seu consentimento, e esta atitude não lhe trará prejuízos no futuro. Em qualquer momento, você poderá fazer perguntas sobre o estudo ou esclarecer dúvidas. Você poderá entrar em contato com Flávia Xavier Valente ou Flávia Galvão Cândido, Prof. Maria do Carmo Gouveia Peluzio ou Prof. Rita de Cássia para esta finalidade nos telefones: (31-3899-3388 / 31-3899-2111 / 31-8662-0687).

Ao assinar este documento, confirmo que me foi explicado o objetivo, os procedimentos aos quais serei submetido, os riscos e os benefícios potenciais que eu possa experimentar, e os possíveis destinos dos resultados que serão obtidos neste estudo. As perguntas que foram feitas foram satisfatoriamente respondidas, li e compreendi este termo de consentimento, ficando em meu poder uma cópia do mesmo. Ainda sim, em caso de dúvidas não esclarecidas de maneira adequada pelo pesquisador responsável, de discordância com procedimentos ou irregularidade de natureza ética posso buscar auxílio junto ao **Comitê de Ética em Pesquisa com Seres Humanos da Universidade Federal de Viçosa** localizado no campus Viçosa, prédio Arthur Bernardes, sala 04**, Telefone: (31) 3899-2492, e-mail: cep@ufv.br.** Este termo está de acordo com a Resolução 466 do Conselho Nacional de Saúde, de 12 de dezembro de 2012 e foi redigido em duas vias.

Portanto, assino e dou meu consentimento para participar deste estudo.

Viçosa, _____ de _____ de 2015.

Identificação do voluntário da pesquisa

Nome:_____

Telefone_____Celular:_____e-mail:_____

Endereço: _____

_____ _____

Voluntário Pesquisador

_____

Profª. Dra. Maria do Carmo Gouveia Peluzio

Coordenadora do Projeto

DNS/UFV- 3899-2111