A coconut extra virgin oil-rich diet increases HDL cholesterol and decreases waist circumference and body mass in coronary artery disease patients

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Abstract

Introduction: saturated fat restriction has been recommended for coronary arterial disease, but the role of coconut oil (Cocos nucifera L.) extra virgin, lauric acid source in the management of lipid profile remains unclear.

Objective: to evaluate the effect of nutritional treatment associated with the consumption of extra virgin coconut oil in anthropometric parameters and lipid profile.

Methods: we conducted a longitudinal study of 116 adults of both sexes presenting CAD. Patients were followed in two stages: the first stage (basal-3 months), intensive nutritional treatment. In the second stage (3-6 months), the subjects were divided into two groups: diet group associated with extra virgin coconut oil consumption (GDOC) and diet group (DG). Held monthly anthropometric measurements: body mass, waist circumference (WC), neck circumference (PP), body mass index (BMI). Gauged to collected blood pressure and blood samples were fasted for 12 hours, for total cholesterol analysis and fractions apoproteins (Apo A-1 and B), glucose, glycated hemoglobin (HbA1C), insulin (I). Comparing the averages at the beginning and end of the study employing the paired Student t-independent. And set the diastolic blood pressure by BMI using ANOVA. Analyses were performed using the SPSS statistical package, being significant p < 0.05.

Results: the mean age of the population was 62.4 ± 7.7 years, 63.2% male, 70% elderly, 77.6% infarcted, 52.6% with angina, hypertension and dyslipidemia 100%. In the first stage the nutritional treatment reduced body weight, WC, BMI and PP and insulin concentrations, HbA1C, HOMA-IR and QUICK, without changing the other parameters. In the second stage of the study, it was observed that the GDOC maintained the reduction of body mass, BMI, WC, with a significant difference between groups for DC (-2.1 ± 2.7cm; p < 0.01).

In addition, there was an increase in HDL-C concentrations, Apo A, with significant difference in GD, only for HDL-C (3.1 ± 7.4 mg/dL; p = 0.02).

**Conclusion:** it was observed that the nutritional treatment associated with extra virgin coconut oil consumption reduced the CC and increased HDL-C levels in patients with CAD.


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Key words: **Coronary artery disease. Nutritional treatment. Secondary prevention and extra virgin coconut oil.**

### Introduction

Secondary prevention for patients with coronary artery disease (CAD) aims to avoid new cardiovascular events. The change towards a healthier lifestyle presents a 44% decrease in mortality from CAD. Recent guidelines emphasize the necessity of reducing visceral fat, and controlling blood pressure and dyslipidemia. The adoption of a dietary pattern based on good sources of mono- and polyunsaturated fat, fiber, fruit, vegetables, whole grains, olive oil and nuts results in the decrease of risk factors for cardiovascular disease. On the other hand, according to the National Health and Nutrition Examination Survey (2007-2011), conducted on 759 individuals with CAD, data showed very low compliance to the nutritional and clinical treatment, with only 20% displaying adequate body weight, and 59% having lipid profile control.

New therapeutic targets are necessary to increase compliance to dietary treatments. Due to this necessity, the effects of functional foods have been studied. Although no consensus exists over the subject, functional foods appear to exert some beneficial action on lipid profile and promote better compliance to dietary treatment.

In this context, extra virgin coconut oil (Cocos nucifera L.), extracted from the fresh coconut pulp, has been acknowledged for its high proportion of medium-chain fatty acids (MCFA), lauric acid (source of vitamin E), and polyphenols with antioxidant activity. The scientific literature has shown benefits of extra virgin coconut oil to the reduction of body fat, but there is still controversy over its effects on lipid profile, since it is a source of saturated fat. Thus, the aim of this study was to evaluate the effect of a diet rich in coconut oil concerning the improvement of lipid profile and anthropometric measurements.

### Methods

**Study subjects and design**

We conducted a nonrandomized 6-month clinical trial, with 360 patients being initially screened. The study included patients of both genders aged 45-85 years on secondary prevention of CAD (myocardial infarction and/or stable angina), with the use of lipid-lowering drugs for longer than six months, seen at an outpatient department of a specialized cardiology hospital during January-September, 2012. It excluded those who had coronary artery bypass grafting and previous cardiovascular event within less than 6 months, those who had chronic renal failure with creatinine levels greater than 1.2 mg/dL, patients using coconut oil, food supplements, and those who suffered from liver diseases.

From the screened population, 136 patients met the eligibility criteria for the three-month run-in phase in order to homogenize or standardize their food intake. From the third month the allocation was performed for two intervention groups: diet group (DG) (n = 22), who remained only with diet, and another group that besides diet received extra virgin coconut oil (CODG) (n = 92). The study details are better shown in figure 1.

Patients were seen in a monthly basis at the clinical nutrition department of a specialized hospital where they received intensive dietary treatment with periodic phone calls to assess compliance. In addition, all patients were provided with a telephone number to contact to dispel doubts whenever necessary. Socio economic and demographic data, information on past medical history and present illness, drug therapy, and physical exercise were collected. In each visit, 12-hour fasting blood sample was drawn, 24-hour dietary recall was obtained, anthropometric assessment was made and systemic blood pressure (BP) was measured. At the beginning of the run-in period, all patients were given a adequate nutritional status diet and instructed to follow it until the end of the study.

The experimental protocol was approved by the Research Ethics Committee of Instituto Nacional de Cardiología (INC)-RJ under no. 0305/2010, and its National Clinical Trial (NCT) number is 01962844. All the volunteers were informed about the procedures they would undergo during the research, and signed the statement of informed consent (SIC).
Anthropometric measurements, physical activity and blood sampling

The anthropometric measurements body mass (kg) and height (m) were taken using a digital platform scale coupled with a stadiometer (Filizola®). BMI was calculated by dividing body mass (kg) by height (m) squared, classified according to the World Health Organization (WHO).

WC was measured at the midpoint between the last rib and the iliac crest. NC was measured with the subject standing with the head positioned in the Frankfort horizontal plane, the upper edge of the tape was placed under the cricoid cartilage and applied perpendicularly around the neck. Blood pressure was measured twice in the right arm by the trained investigator, with a mercury sphygmomanometer and stethoscope after subjects had rested for a minimum of 10 minutes.

Physical activity was considered when patients trained at least once a week. Physical exercise was assessed as metabolic equivalent of task (MET) expressed in kcal/day. Patients were considered sedentary when they did not perform physical exercise, or when they exercised with caloric expenditure below 3 METs and a frequency of less than two times per week. Patients were advised to keep the level of habitual physical activity.

Blood samples were drawn after 12 hours of overnight fasting. The samples were taken in blood collection vacuum tubes containing heparin. The collection tubes were then centrifuged for 15 min at 4°C and 3,000 rpm. TG, TC, and HDL-C and LDL-C were analyzed. Serum levels of ApoA-1 and ApoB were measured by
immunoturbidimetric assay. Fasting plasma glucose was measured by the spectrophotometric method using the glucose oxidase/peroxidase. The glycated hemoglobin (HgA1c), by turbidimetric immunoassay. All analyses were performed at the clinical laboratory of INC (Rio de Janeiro, Brazil) through the automated method (ARCHITECT ci8200, Abbott, Abbott Park, IL, USA) using commercial kits (Abbott ARCHITECT ci8000®, Abbott Park, IL, USA).

**Diet design and supplementation**

The diet was prescribed during the run-in period according to the dietary habits of volunteers and nutritional recommendations for individuals with dyslipidemia. The total energy expenditure was calculated considering the recommendations of the Dietary Reference Intake, 2005, and of the National Cholesterol Education Program - Adult Treatment Panel III (NCEP ATPIII) (2002) considering the current BM. At each visit, a 24-hour recall was used to assess patient compliance to the offered nutritional treatment. In order to assess changes in the habitual dietary pattern, baseline 24-hour recalls were compared to those three months after intervention. Data were analyzed using the computer program Food Processor Version 7.2 (Esha Research, Salem, USA, 1998).

The CODG received extra virgin coconut oil in sachets containing 13 mL (30 units per month), totaling 90 sachets per patient. Patients were instructed to consume one sachet per day, alone or added to fruit, without subjecting it to heat.

Coconut oil was donated by COPRA Food Industry, Maceió, AL, Brazil.

The composition of fatty acids of coconut oil was obtained by the Analytical Chemistry Organic Laboratory of Centro de Pesquisas e Desenvolvimento Leopoldo Américo Miguez de Mello (CENPES)/Rio de Janeiro, RJ, Brazil. Vitamin E and phytosterols contents were determined by the Instituto de Tecnologia de Alimentos/Centro de Ciências e Qualidade de Alimentos, Campinas, São Paulo, SP, Brazil (Table I).

**Statistical analysis**

The results were expressed as percentage and mean ± standard deviation (SD). The chi-square test (c² test) was performed to compare categorical variables between groups. Kolmogorov-Smirnov adhesion test was performed.

Paired Student’s t-test or Wilcoxon Signed Ranks was used to assess changes in anthropometric and biochemical variables after the intervention period in each group. While the effect of the intervention groups was evaluated by Student t test independent or Mann-Whitney U test according to the distribution of variables.

The difference between DG and CODG was evaluated by Student’s t-test. Through the analysis of variance for repeated measures, DBP was adjusted for BMI, and the development of HDL-C and WC in CODG and DG was evaluated. All analyses were performed using SPSS, version 20.0. When p < 0.055, the finding was considered statistically significant.

**Results**

One hundred and thirty-six patients were included in the study. Among them, one hundred and fourteen (85.3%) managed to complete the run-in period. The majority of the participants who abandoned the study

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### Table I

**Nutrient composition of a serving of coconut oil**

| Energy, kcal/kJ | 127/533 | Composition of fatty acids (%/100g) | C6:0 Caproic | – | Brassicasterol | 5.61 (0.11)b |
| Carbs, g | 0 | C8:0 Caprylic | 7.0 | Campesterol | 13.75 (0.53)b |
| Protein, g | 0 | C10:0 Capric | 6.0 | Stigmasterol | 32.43 (0.33)b |
| Total Fat, g | 14 | C12:0 Lauric | 48.0 | Alpha-tocopherol | ND < 0.02a |
| Saturated fat, g | 13 | C14:0 Myristic | 19.0 | Beta-tocopherol | ND < 0.02a |
| Trans fat, g | 0 | C16:0 Palmitic | 9.0 | Delta-tocopherol | NDa |
| Monounsaturated fats, g | 0.8 | C18:1 Searic | 3.0 | Vitamin E (IU/100 g) | ND < 300a |
| Polyunsaturated fats, g | 0.2 | C18:1 PUFA 9 Oleic | 8.0 | Vitamin E (IU/100 g) | ND < 300a |
| Cholesterol, mg | 0 |  |  |  |  |
| Fiber, mg | 0 |  |  |  |  |
| Sodium, mg | 0 |  |  |  |  |

1Composition held in 15ml of extra virgin coconut oil = 1 tablespoon.
2Source: Laboratory of Analytical Chemistry Organic Cenpes/Rio de Janeiro, RJ, Brazil.
3Source: Institute of Food Technology, Science Center and Food Quality, Campinas, São Paulo, SP, Brazil Laboratory.
4ND: not detected value; bAverage and estimated standard deviation; mg/dL: milligram per deciliter; IU: international unit.
did not return after the baseline visit due to scheduling difficulties (Fig. 1).

The main characteristics of the population are shown in Table II; there was no significant difference between the studied groups since the beginning of intervention. The mean age of the studied population was 62.4 ± 7.7 years, with 70% of elderly individuals, and 63.2% of males. There were 100% hypertensive and 94.5% dyslipidemic patients on regular medication to control these diseases.

During the run-in period, there was significant decrease in body mass (BM), body mass index (BMI), neck circumference (NC), waist circumference (WC) and glycemic profile (data not shown).

Table III shows the effect of an extra virgin coconut oil-rich diet on anthropometric data and on the blood pressure (BP) after three months of intervention. Data showed that the CODG significantly decreased their BM, BMI, NC, WC, with a statistical difference between the groups for WC (-2.1 ± 2.7; p < 0.01) (Fig. 2A). We also observed the reduction of diastolic blood pressure (DBP) in the CODG; however, after adjustment for BMI, no significant difference was found (data not shown). There was no significant difference in physical activity between both groups at the beginning and during the phases of the study (data not shown).

CODG presented an increase on serum concentrations of HDL-C (CODG: 3.1 ± 7.4 mg/dL; p < 0.01 vs. DG: -1.2 ± 8.5 mg/dL; p = 0.52) and apoprotein A (apoA) (CODG: 4.7 ± 12.7; p = 0.01 vs. DG: -3.9 ± 2.7; p = 0.27).

We notice the effect of a coconut-rich diet on the levels of HDL-C in figure 2B. We may also observe a small increase on the concentrations of apoprotein B (apoB) in the CODG, however with no difference on serum concentrations of low-density lipoprotein cholesterol (LDL-C) and total cholesterol (TC).

The dietary assessment, undertaken through the 24-hour recalls, showed decrease in the total energy expenditure (-748.9 ± 1110.6 kcal; p < 0.01), lipids (-4.1 ± 11.4; p < 0.01), saturated fat (-2 ± 5.1%; p < 0.01), cholesterol (-70.9 ± 199.1 mg/dL; p < 0.01) and sodium (-814.5 ± 1583.2 mg/dL; p < 0.01), after the run-in period (data not shown). After the run-in period, the CODG presented increased intake of lipids and saturated fatty acids, with reduced carbohydrate intake. There was no modification in the DG group. The statistical analyses showed no difference between the groups.

Discussion

The results of this study show that the inclusion of 13 mL of extra virgin coconut oil in a diet increases significantly the HDL-C levels and decreases the WC. Previous studies involving the intake of coconut oil have linked it to the reduction of abdominal fat, as the

<table>
<thead>
<tr>
<th>Variables</th>
<th>CODG (n = 2)</th>
<th>DG (n = 22)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, years</td>
<td>62.5±8.02</td>
<td>63.2±11.5</td>
<td>0.941</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>79.7±15.7</td>
<td>79.59±14.1</td>
<td>0.961</td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td>29.9±5.8</td>
<td>29.7±5.2</td>
<td>0.821</td>
</tr>
<tr>
<td>Inactivity, % (n)</td>
<td>71.1 (66)</td>
<td>81.8 (18)</td>
<td>0.111</td>
</tr>
<tr>
<td>Diabetes mellitus type 2, % (n)</td>
<td>50 (46)</td>
<td>36.4 (8)</td>
<td>0.441</td>
</tr>
<tr>
<td>Hypoglycemic, % (n)</td>
<td>48.9 (45)</td>
<td>40.9 (9)</td>
<td>0.501</td>
</tr>
<tr>
<td>Angina, % (n)</td>
<td>46.7 (43)</td>
<td>40.9 (9)</td>
<td>0.801</td>
</tr>
<tr>
<td>Acute myocardial infarction, % (n)</td>
<td>77.2 (71)</td>
<td>77.3 (17)</td>
<td>0.151</td>
</tr>
<tr>
<td>C-Total, mg/dL</td>
<td>177.5±51.8</td>
<td>176.9±68.6</td>
<td>0.961</td>
</tr>
<tr>
<td>LDL-col, mg/dL</td>
<td>108.3±45.1</td>
<td>114.5±55.5</td>
<td>0.581</td>
</tr>
<tr>
<td>HDL-col, mg/dL</td>
<td>37.5±9.2</td>
<td>37.5±9.3</td>
<td>0.961</td>
</tr>
<tr>
<td>Triglycerides, mg/dL</td>
<td>153.7±71.2</td>
<td>153.0±68.9</td>
<td>0.711</td>
</tr>
<tr>
<td>SBP, mmHg</td>
<td>129.0±19.0</td>
<td>128.1±15.9</td>
<td>0.841</td>
</tr>
<tr>
<td>DBP, mmHg</td>
<td>77.8±11.5</td>
<td>81.3±9.9</td>
<td>0.182</td>
</tr>
</tbody>
</table>

Abbreviations: SBP: SBP; DBP: diastolic blood pressure; C-total: total cholesterol; HDL-C: high density lipoproteins cholesterol; LDL-C: low density lipoprotein cholesterol; TG: triglycerides.
1Results are expressed as mean ± SD or percentage.
2T-Student test between CODG and DG.
3Chi-square test between the groups, statistically significant for p < 0.05.
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200935, by comparing refined coconut oil and refined soy oil, did not find any benefit in the lipid profile, and Liau et al. 201126 did not find any effect either. Two other studies with isolated MCT also found no important change in the lipid profile30,31.

Experimental studies in which animals were fed diets supplemented with virgin coconut oil showed increased levels of HDL-C and decreased levels of LDL-C, TG and TC37. Authors credited the results to the action of polyphenols and vitamin E, present in the virgin coconut oil. Besides, saturated fat is known to have a role in the improvement of HDL-C levels by increasing the activity of lecithin cholesterol acetyltransferase (LCAT)38. The elevation of HDL-C levels, with no change in LDL-C levels, in our population of chronic CAD patients was highly significant, for evidence point that normal concentrations of HDL-C are associated with minor risk of non-lethal infarct39 and low concentrations of HDL-C are strong predictors of infarct40. And further, that the lower the LDL-col lower the cardiovascular morbidity and mortality41.

Table V
Dietary characteristics during the intervention

<table>
<thead>
<tr>
<th>Variables</th>
<th>CODG (n = 92)</th>
<th>DG (n = 22)</th>
<th>p</th>
<th>Baseline</th>
<th>∆T1</th>
<th>p</th>
<th>Baseline</th>
<th>∆T2</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Energy, kcal</td>
<td>1508.3±669.6</td>
<td>76.5±707.2</td>
<td>0.31</td>
<td>1580.3±565.5</td>
<td>-0.5±858.2</td>
<td>0.98</td>
<td>0.69</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PTN, %VET</td>
<td>24.1±7.1</td>
<td>-1.8±9.8</td>
<td>0.09</td>
<td>25.3±8.5</td>
<td>-1.7±12.5</td>
<td>0.58</td>
<td>0.97</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CHO, %VET</td>
<td>55.5±9.6</td>
<td>-3.0±11.2</td>
<td>0.01*</td>
<td>55.1±11.2</td>
<td>-2.9±16.7</td>
<td>0.49</td>
<td>0.98</td>
<td></td>
<td></td>
</tr>
<tr>
<td>LIP, %VET</td>
<td>20.1±8.5</td>
<td>4.5±9.6</td>
<td>&lt;0.01*</td>
<td>20.1±9.2</td>
<td>4.9±18.2</td>
<td>0.29</td>
<td>0.89</td>
<td></td>
<td></td>
</tr>
<tr>
<td>SFA, %</td>
<td>6.5±3.6</td>
<td>5.9±4.8</td>
<td>&lt;0.01*</td>
<td>6.4±2.6</td>
<td>3.7±10.3</td>
<td>0.16</td>
<td>0.17</td>
<td></td>
<td></td>
</tr>
<tr>
<td>MFA, %</td>
<td>6.2±4.1</td>
<td>0.2±5.4</td>
<td>0.07</td>
<td>6.9±4.4</td>
<td>0.5±6.8</td>
<td>0.78</td>
<td>0.87</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PFA, %</td>
<td>3.4±2.6</td>
<td>-0.6±3.3</td>
<td>0.12</td>
<td>3.3±2.8</td>
<td>-0.2±3.8</td>
<td>0.85</td>
<td>0.89</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cholesterol, mg/d</td>
<td>179.8±122.6</td>
<td>-10.9±170.7</td>
<td>0.54</td>
<td>212.8±137.0</td>
<td>54.9±288.6</td>
<td>0.44</td>
<td>0.20</td>
<td></td>
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</tr>
<tr>
<td>Sodium, mg/d</td>
<td>1311.2±768.7</td>
<td>-57.7±894.7</td>
<td>0.54</td>
<td>1251.4±680.8</td>
<td>368.1±1092.0</td>
<td>0.18</td>
<td>0.06</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fiber, g/d</td>
<td>26.9±16.6</td>
<td>-0.3±18.7</td>
<td>0.86</td>
<td>25.7±11.0</td>
<td>-6.8±10.3</td>
<td>0.01*</td>
<td>0.19</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Abbreviation: PTN: protein; CHO: carbohydrate; LIP: lipids; SFA: saturated fatty acids; MFA: monounsaturated fatty acids; PFA: polyunsaturated fatty acids.

1Results expressed in mean ± SD.

∆T1 = (3 months-baseline); ∆T2 = (3 months-baseline).

*Within 3 months statistically significant (p < 0.05).

Fig. 2.—Evolution of WC (A) and HDL-C (B) between the groups (DG x CODC) during the three-month intervention. *Test of analysis of variance for repeated measures, p < 0.05.

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were highly significant and unprecedented in this group of patients with chronic coronary disease. The intake of this kind of fat meets strong opposition from people in general, although studies have not proved the association between the intake of saturated fat and cardiovascular disease or CAD\textsuperscript{42}. Also, considering a specific population that regularly used this coconut oil, there was no positive association with the onset of cardiovascular disease\textsuperscript{10}.

Our study presents some limitations: small sample size in the diet group, absence of randomization when allocating patients to nutritional intervention. However, it is noteworthy that CODG and DG were comparable in relation to anthropometric and biochemical data.

**Conclusion**

Nonpharmacological interventions are essential for risk factor control in secondary prevention among patients with coronary disease. Our study showed that a diet rich in extra virgin coconut oil seems to favor the reduction of WC and the increase of HDL-C concentrations, aiding with secondary prevention for CAD patients.

**Acknowledgment**

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**Authors’ contributions**

DAC, GMMO, ABM, RRL and GR were responsible for the study conception and design, and the drafting of the manuscript; DAC, GMMO, ABM, RRL and GR participated in the analysis and interpretation of data; GMMO, ABM and GR critically revised the article for intellectual content; RRL developed the statistics; all authors are accountable for the final approval of the manuscript. None of the authors had a conflict of interest.

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