Coconut oil increases HDL-c and decreases triglycerides in wistar rats

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ABSTRACT. Changes in body composition and serum lipid profile in rats, supplemented with coconut oil, are evaluated and compared to other lipid sources. Female Wistar rats received by gavage 1 mL kg-1 of saline, soybean oil, lard or coconut oil during 21 days. At the end of the study period, body composition, food intake, feces, urine, organ weight and serum lipid profile were assessed. No statistical differences between the groups were found in body composition, food intake, fecal and urinary analysis, and organ weight. In the case of plasma lipid concentrations, coconut oil and lard raised total cholesterol levels, without changes in LDL levels. On the other hand there was no change in total cholesterol levels in the soybean oil group. HDL fraction increased in all groups when compared to that in the saline group; this increase was more significant in the coconut oil group. There was significant reduction of serum triglycerides only in the coconut oil group when compared to the saline group. Supplementation with coconut oil did not interfere in weight and body composition of the animals used in current study, but revealed significant effect on the increase of HDL-c levels and decrease of serum triglycerides.

Keywords: dyslipidemias, body composition, oils.

Óleo de coco aumenta o HDL-c e reduz os triglicerídeos séricos em ratas Wistar

RESUMO. Analisar as alterações na composição corporal e no perfil lipídico sérico de ratas suplementadas com óleo de coco, comparativamente a outras fontes lipídicas. Foram utilizadas ratas Wistar, as quais receberam via oral 1 mL kg-1 de solução salina, óleo de soja, gordura de porco ou óleo de coco por 21 dias. Ao término avaliaram-se a composição corporal, o consumo alimentar, as fezes, a urina, o peso de órgãos e o perfil lipídico sérico. Não houve diferença estatística entre os grupos com relação à composição corporal, ao consumo alimentar, à análise de fezes e à urina, e peso de órgãos.QUanto às concentrações de lipídios plasmáticos, o óleo de coco e a gordura de porco elevaram o colesterol total, sem alteração dos níveis de LDL, quando comparados ao grupo salina. Em todos os grupos foi observada elevação da fração HDL, sendo mais significativa no grupo óleo de coco, quando comparados ao grupo salina. Ainda, se comparado ao grupo salina, apenas no grupo óleo de coco houve redução significativa das concentrações séricas de triglicerídeos. A suplementação com óleo de coco não interferiu no peso e composição corporal dos animais em estudo. No entanto esta suplementação apresentou importante efeito no aumento do HDL-c e na redução dos triglicerídeos séricos.

Palavras-chave: dislipidemias, composição corporal, óleos.

Introduction

According to the World Health Organization (WHO), cardiovascular diseases (CVDs) are the leading cause of death worldwide, accounting for 30% of global deaths, with almost rates in Brazil (Organização Mundial da Saúde [OMS], 2014). Studies show that deaths from CVDs could be prevented in 50 to 70% of patients if they comply with dietary recommendations and changes in lifestyle, or rather, in the control of obesity, metabolic syndrome and blood pressure, all of which have an impact in triggering these diseases (Sposito, Caramelli, Fonseca, & Bertolami, 2007). Thus, it may be observed that actions related to food and nutrition intervene in the prevention and control of such diseases and may modify the epidemiological profile mentioned above (Coltro et al., 2009).

Currently, dietary patterns have been altered as a result of nutritional transition process. It is a shift characterized by sequential changes in diet patterns.
and body composition of individuals, which, in turn, is the result of social, economic, demographic, technological and cultural changes that have affected the population’s lifestyle and health profile health. (Sposito et al., 2007; Coutinho, Gentil, & Toral, 2008). In fact the literature reports that the amount and type of dietary fat play a significant role in the onset of cardiovascular risk factors by causing elevated lipid concentrations and serum lipoproteins (Santos et al., 2013).

It is a well-known fact that food sources, rich in certain types of fatty acids, impact normal body metabolism (Almeida, Queiroz, Queiroz, Costa, & Matta, 2009). Saturated fatty acids and cholesterol, mainly present in animal fats, trigger changes in lipid metabolism, favoring the emergence of CVD. Moreover, oils rich in polyunsaturated fatty acids, present in corn oil and fish, for example, promote the reduce blood cholesterol in patients with atherosclerosis (Teitelbaum & Walker, 2001). In addition, omega-3 fatty acids (n-3) in the diet alter the metabolism of various organs by its conversion into eicosanoids, prostaglandins, leukotrienes, lipoxins and thromboxanes (Luz, Silva, Marques, Luciano, & Souza, 2012).

Coupled to the above information, coconut oil has recently been popularly attributed with several functional, especially related to better body composition. Since coconut has a vegetable origin, it has a high content of saturated fatty acids (Jayadas & Nair, 2006), mainly consisting of triglycerides with low degree of unsaturation, comprising two major fatty acids, lauric acid and myristic acid (Pham, Casa, Gregorio, & Kwon, 1998). Saturated fats, rich in lauric acid, provide a more favorable lipid profile of a fat rich in trans fatty acids. Moreover, myristic and palmitic acids increase LDL-C and HDL-C, with a protective role for CVD (Pham et al., 1998; Santos et al., 2013).

It has been demonstrated that consumption of coconut oil exhibits anti-inflammatory, analgesic and antipyretic effects, reduces tissue lipid levels and enhances the anti-thrombotic effects associated with inhibition of platelet coagulation, promotes low cholesterol level, increases antioxidant activity and inhibits lipid peroxidation in mice (Nurul-Iman, Kamisah, Jaarin, & Qodriyah, 2013).

Current study evaluates the changes in body composition and serum lipid profile in rats supplemented with coconut oil and compared to other lipid sources.

Material and methods

Animals and diet

Current experiment was approved by the Ethics Committee on Animal Experimentation of the Federal University of Grande Dourados (UFGD) through protocol no. 009/2011, according to Law 11.794 of October 8, 2008 established by the National Board of Control of Animal Experimentation (Sociedade Brasileira de Ciência em Animais de Laboratório [Concea], 2013).

Wistar rats, 8 weeks old and weighing 150-200 g, were divided into four groups (n = 7 - 8), placed in metabolic cages and fed on rodent chow (Purina Labina®), with water ad libitum, for 21 days (Castro, Almeida, Ovidio, & Jordão, 2012). The groups were treated orally (by gavage) with saline, soybean oil (9 kcal mL⁻¹) (Tabela Brasileira de Composição dos Alimentos [Taco], 2011), lard (5.89 kcal mL⁻¹) (Taco, 2011) and coconut oil (8.46 kcal mL⁻¹) (according to the product label), purchased in supermarkets in Dourados, Mato Grosso do Sul State, Brazil. The daily dose was 1.0 mL kg⁻¹ (Gonçalves, Mariucci, & Vicentini, 2007).

The consumption of water and feed were quantified daily, as feces and urine, by means of a 0.0001 g precision analytical balance (GEHAKA AG200®), during the administration of the respective treatments. After 21 days, the animals were fasted for 12 hours, anesthetized with ketamine and xylazine (10:75 mg kg⁻¹) and euthanized by exsanguination of the inferior vena cava. The collected blood was centrifuged (4000 rpm x 10 min) and the serum was kept at -20°C until biochemical analysis.

Feed efficiency coefficient

The Feed Efficiency Coefficient for weight gain and food consumption during the entire experimental period was calculated by the formula: Weight gain (g)/Food consumption (g) in the same period (Weber, Freitas, Amancio, & Morais, 2010).

Urine and feces

Feces and urine were weighed daily on an analytical balance, accurate to 0.0001 (GEHAKA AG200®). After verification, the feces were frozen at -20°C and the urine discarded.

Liver weight and carcass

After euthanasia, the viscera were removed and the liver preserved for weighing. After weighting, the carcass was frozen at -20°C and the remainder discarded.

Concentration of fat in the carcass and feces

Fat was determined by Soxhlet method. The carcasses and feces were dried in an oven with air circulation at 60°C for 14 and 8 days, respectively.
They were then ground in a knife mill; each sample was then weighed and packaged in cartridges, using petroleum ether as solvent (Instituto Adolfo Lutz [IAL], 1985). Fat percentage was calculated by weight difference before and after extraction.

**Concentration of total blood cholesterol, HDL-C, LDL-C and triglycerides**

Total cholesterol, HDL-C, LDL-C and triglycerides, was calculated based on colorimetric enzymatic method for semi-automatic biochemical analysis system, according to manufacturer's instructions (Lab-test Diagnostic®).

**Statistical analysis**

Results are expressed as mean ± standard error of mean (SEM). The analysis of variance (ANOVA) was used to compare 3 or more groups. When differences were significant, the analysis was complemented by Tukey’s test or Newman-Keuls, using a 5% significance level. Calculations were performed with Graph Pad Instat program 3.02.

**Results and discussion**

The comparison of groups supplemented with saline, soybean oil, lard and coconut oil, with regard to the total weight gain and total food intake showed no statistical difference. However, feed efficiency coefficient revealed a significant difference between the group treated with lard and soybean oil (p < 0.05) and demonstrated that the soybean oil group was capable of retaining more energy than the lard group (Table 1).

**Table 1.** Total weight gain (G), food consumption (FC) and feed efficiency coefficient (FEC) in rats treated with different fat sources for 21 days.

<table>
<thead>
<tr>
<th></th>
<th>Saline</th>
<th>Soybean Oil</th>
<th>Lard</th>
<th>Coconut Oil</th>
</tr>
</thead>
<tbody>
<tr>
<td>G (g)</td>
<td>34.28 ± 3.93</td>
<td>43.00 ± 2.07</td>
<td>30.75 ± 3.62</td>
<td>30.28 ± 3.33</td>
</tr>
<tr>
<td>FC (g)</td>
<td>418.28 ± 16.37</td>
<td>413.50 ± 14.53</td>
<td>413.14 ± 16.85</td>
<td>398.75 ± 16.16</td>
</tr>
<tr>
<td>FEC</td>
<td>0.08 ± 0.00</td>
<td>0.10 ± 0.00</td>
<td>0.07 ± 0.01</td>
<td>0.07 ± 0.00</td>
</tr>
</tbody>
</table>

Rates as mean ± SEM using ANOVA followed by Tukey’s test. n = 7-8.

There was no statistical difference between groups with regard to weight of feces, volume of urine (Table 2), and water consumption (data not shown). The same occurred with regard to the weight of the organs analyzed (Table 3), and to fat percentage of carcasses and feces (Table 4).

**Table 2.** Weight of feces and urine volume in rats treated on different fat sources for 21 days.

<table>
<thead>
<tr>
<th></th>
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<th>Lard</th>
<th>Coconut Oil</th>
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<tbody>
<tr>
<td>Feces</td>
<td>181.18 ± 10.77</td>
<td>173.18 ± 9.52</td>
<td>178.50 ± 14.59</td>
<td>173.69 ± 12.50</td>
</tr>
<tr>
<td>Urine (mL)</td>
<td>130.91 ± 19.97</td>
<td>99.35 ± 11.80</td>
<td>91.38 ± 18.08</td>
<td>110.35 ± 24.96</td>
</tr>
</tbody>
</table>

Rates as mean ± SEM using ANOVA followed by Tukey’s test. n = 7-8.

The evaluation of serum lipids showed that coconut oil and lard increased the total blood cholesterol, with significant difference (p < 0.01), when compared to groups treated with saline and soybean oil. In the case of LDL-c, the group supplemented with soybean oil revealed lower rates when compared to those of other groups. HDL-c levels were significantly increased in the group treated with coconut oil when compared to those with saline, soybean oil and lard groups. Coconut oil also reduced blood triglycerides, with a significant difference from the other groups (p < 0.001) (Figure 1).

**Information on the fatty acid composition of studied oils and fats is essential to justify their mechanism of action and their activities at different parameters. The evaluation of the ratio of polyunsaturated fatty acids and saturated fatty acids (PUFA/SFA) in oil or fat source identifies the**

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**Table 3.** Weight of liver, spleen, kidney, heart and lung of rats treated on different fat sources for 21 days.

<table>
<thead>
<tr>
<th></th>
<th>Saline</th>
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<th>Lard</th>
<th>Coconut Oil</th>
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<tbody>
<tr>
<td>Liver (g)</td>
<td>7.27 ± 0.15</td>
<td>7.33 ± 0.34</td>
<td>7.01 ± 0.25</td>
<td>6.99 ± 0.21</td>
</tr>
<tr>
<td>Spleen (g)</td>
<td>0.49 ± 0.01</td>
<td>0.47 ± 0.01</td>
<td>1.02 ± 0.71</td>
<td>0.64 ± 0.01</td>
</tr>
<tr>
<td>Right kidney (g)</td>
<td>0.78 ± 0.01</td>
<td>0.80 ± 0.03</td>
<td>0.79 ± 0.02</td>
<td>0.78 ± 0.03</td>
</tr>
<tr>
<td>Left kidney (g)</td>
<td>0.77 ± 0.02</td>
<td>0.78 ± 0.03</td>
<td>0.78 ± 0.02</td>
<td>0.76 ± 0.02</td>
</tr>
<tr>
<td>Heart (g)</td>
<td>0.81 ± 0.02</td>
<td>0.84 ± 0.02</td>
<td>0.77 ± 0.02</td>
<td>0.81 ± 0.02</td>
</tr>
<tr>
<td>Lung (g)</td>
<td>1.09 ± 0.04</td>
<td>1.18 ± 0.05</td>
<td>1.84 ± 0.77</td>
<td>1.14 ± 0.03</td>
</tr>
</tbody>
</table>

Rates as mean ± SEM using ANOVA followed by Tukey’s test. n = 7-8.

**Table 4.** Percentage of lipid carcasses and feces in rats treated on different lipid sources for 21 days.

<table>
<thead>
<tr>
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<th>Lard</th>
<th>Coconut Oil</th>
</tr>
</thead>
<tbody>
<tr>
<td>Carcasses (%)</td>
<td>26.02 ± 2.36</td>
<td>22.74 ± 1.50</td>
<td>22.76 ± 1.02</td>
<td>21.80 ± 1.10</td>
</tr>
<tr>
<td>Feces (%)</td>
<td>7.32 ± 1.97</td>
<td>8.74 ± 2.62</td>
<td>8.02 ± 1.67</td>
<td>5.23 ± 0.82</td>
</tr>
</tbody>
</table>

Rates as mean ± SEM using ANOVA followed by Tukey’s test. n = 7-8.

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**Figure 1.** Profile of blood lipids rats supplemented with different lipid sources for 21 days. Concentration of A) total blood cholesterol; B) LDL-cholesterol; C) HDL-cholesterol; D) and triglycerides. n = 4-7. Rates as mean ± SEM with ANOVA, followed by Newman-Keuls test. Different letters mean significant difference in the comparison of the groups.
relationship between dietary lipids and lipid metabolism (Newman et al., 2002).

Coconut oil is comprised of approximately 44% saturated fatty acids and 66% unsaturated fatty acids (Machado, Chaves, & Antoniassi, 2006), with a composition similar to that of lard, which has a higher acid content because of a higher saturated fat percentage (45.05%) than unsaturated fatty acids (54.95%). The composition is the reverse of soybean oil, which has lower amounts of saturated fatty acids (14.19%) when compared to unsaturated fatty acids (85.49%) (Almeida, Queiroz, Costa, & Matta, 2011). This fact influences many physiological mechanisms, such as the regulation of the activity of pancreatic lipase and, as a result, the digestibility of lipids (Ricketts & Brannon, 1994). A high PUFA/SFA ratio increases fat digestibility (Ricketts & Brannon, 1994; Monsma, Gallaher, & Ney, 1996).

Another factor that also determines digestibility is the amount of stearic fatty acids (C18:0). Several studies indicate that high stearic fatty acids concentration in food sources increases fat digestibility (Monsma et al., 1996). Therefore, the above factor justifies the difference found in the Feed Efficiency Coefficient between the lard group and the soybean oil group. In addition to a high PUFA/SFA ratio, soybean oil contains small amounts of stearic acid (4%) and consequently, it is highly digestible.

Almeida, Queiroz, Costa, and Matta (2011) analyzed serum lipid abnormalities and liver histology of rats fed on different lipid sources (soybean oil, fish and lard, margarine and butter) and did not detect any statistical difference between the groups in weight gain and food intake, corroborating results in current study.

With respect to faeces weight and urine volume, there were no significant differences among the groups. Quinna et al. (2013) reported that a high-fat diet generated greater satiety and consequent reduction consumption of food, water and excretion consumption.

Further exploration of these mechanisms is necessary, although it is possible to infer that this effect is tied to the amount of ingested fat.

In general, excessive intake of fat induces its accumulation in such organs as the heart and liver (Quinna et al., 2013). In the case of the liver, fat accumulation is detected as hepatic steatosis, characterized by the accumulation of VLDL particles, which cause liver increase with a weight 5% higher than the reference rate (Almeida et al., 2011). However, treatment with different types of oils or fats in current study did not cause changes in the weight of liver, heart and other organs analyzed. Result shows that fat dose in current study was not so high to alter these organs.

The same happened with the carcasses which are an evaluation criterion of body composition (Rogero et al., 2008). There was no statistical difference when the study groups were compared. In a study by Ippagunta, Hadenfeldt, Miner, and Hargrave-Barnes (2011), whose objective was to determine if there were changes in lipolysis in rats to offer diets containing soybean oil, coconut oil or fat-free, followed by conjugated linoleic acid (CLA) for 10 or 12 days, a difference was found in body fat loss in the group with coconut oil and CLA. The above suggested that coconut oil increased lipolysis.

Although coconut oil is considered an important natural source of saturated fat, especially lauric acid (C12:0), its use results in more favorable lipid profile than solid fat which is rich in trans fatty acids (Mensink, Zock, Kester, & Katan, 2003; Roos, Schouten, & Katan, 2011). Coconut oil is rich in myristic and palmitic acids and increases LDL and HDL cholesterol (Micha & Mozaffarian, 2010), the latter having been observed in current study. The negative effects of these fatty acids appear to be the cause of the increased prevalence of CVDs, according to studies conducted in Asia, where coconut oil comprises up to 80% of the fat consumed in some regions (Kumar, 1997; Assunção, Ferreira, Santos, Cabral, & Florêncio, 2009).

In an experimental study with guinea pigs comparing coconut oil and olive oil and sunflower oil, there was a significant increase in LDL-c and triglycerides in the groups treated with coconut oil (Lecker, Matthan, Billheimer, Rader, & Lichtenstein, 2010). This result differs from that in current study where coconut oil significantly increased the HDL fraction, making it a powerful alternative food for the prevention and treatment of CVDs due to the low range of foods to achieve this goal. Furthermore, the group supplemented with coconut oil in current analysis showed decreased levels of triglycerides in the other groups, perhaps due to its composition: mainly fatty short and medium chain acids which are transported directly to the liver to undergo β-oxidation (Ooyama, Wu, Nosaka, Aoyama, & Kasai, 2008). Different results were reported in a study by Lecker, Matthan, Billheimer, Rader, & Lichtenstein (2010), which showed an increase in LDL-c and triglycerides.

With regard to the LDL fraction, even within the context of controversy in the literature, soybean oil is acknowledged as a food with powerful cardio-protective effects by significantly decreasing the serum concentrations of LDL-c. Therefore, comparing LDL-c rates with coconut oil would not
be feasible (Hoie et al., 2005) although coconut oil has a protective effect when compared with other lipid sources, such as lard (Santos et al., 2013).

Conclusion

Alteration in weight and body composition was not detected in the use of coconut oil. However, the use of the same showed a significant effect in increasing HDL-c, suggesting that the coconut oil may have a cardio-protective role when included in the diet. Animals supplemented with this oil also showed a significant reduction of serum triglycerides. Compared to other sources of saturated fat (in our case, lard), no harm with regard to total blood cholesterol exists when coconut oil is used. However, further studies are needed to clarify the possible mechanisms leading to the observed results.

References


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