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EVALUATION OF THE EFFECTS OF DIET WITH COCONUT OIL ON THE METABOLISM OF CARBOHYDRATES AND LIPIDS IN WISTAR RATS

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ABSTRACT

The present study Aimed to evaluate the effect of coconut oil on rats submitted to ahypercaloric and hyperlipidic diet. It has about 28 male Wistar rats divided into four groups, group I: animals fed a diet hypercaloric; group II: animals treated with the high calorie diet plus coconut oil; group III: animals treated with commercial ration and group IV: animals treated with commercial ration plus coconut oil. The animals Were weighed and had the glycemia Evaluated weekly throughout the experiment until the day of sacrifice. After 54 days of the experiment, blood was collected for the determination of the total cholesterol and fractions, triglycerides and glucose, and the atherogenic index and the Lee index Were calculated from the results. In addition, the histopathological analysis of the liver glycogen was Performed to quantify. It was Observed que the hypercaloric diet with or without coconut oil and commercial coconut oil did not alter the weight of the animals, consequently there was the induction of obesity in any of the groups, the use of the refrigerant During the 27 days may have contributed to this result. Total plasma cholesterol levels of the normal Also Remained Within limits, the DID triglyceride, HDL and glycemia levels. There was no risk for the development of cardiovascular diseases. In the histopathological analysis is hepatic glycogen deposition, it was shown That the animals treated with commercial ration with or without coconut oil had a higher occurrence of hepatocytes positive for glycogen stores, Whereas the groups with a hypercaloric and hypercaloric diet with coconut oil decrease in hepatocytes positive for PAS, as well as areas presenting degeneration. It was That Concluded the diet hypercaloric and coconut oil did not alter the weight of the animals, but there Were important biochemical Alterations such as the Increase of triglycerides, glucose and HDL in the animals.

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INTRODUCTION

Coconut is considered a food of numerous functions. The variety of products which can be exploited through it, such as coconut oil, coconut milk, coconut water, cool and dry coconut, flour, beauty products, among other uses. By this reason, it is recognized worldwide as a vital resource for plant all mankind (EMBRAPA, 2014).

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Coconut oil is extracted through the process of grinding and pressing coconut cold in nature, has no any additive added to the composition, the oil has an acidity <0.5% and is composed of 47% of lauric acid (RIBEIRO, 2017). The intake of fats and your kind suffers strong influence on metabolism, thus causing various risk factors for metabolic syndrome, such as resistance to insulin and boards, high blood pressure, for example. Also carbohydrates refined and rapid absorption exaggeratedly that are consumed can trigger overweight and obesity accordingly. With these changes, the individual may dyslipidemia display boards, thus further increasing the risk of developing

cardiovascular disease (SANTOS, 2013). However, among the various products used for the treatment of obesity, coconut oil has been a featured food, known to possess thermogenic effect, and considered by many, essential for weight loss (DAUBER, 2015). It is believed in many therapeutic utilities that oil can bring to humans and may be useful in cases such as hair loss, burns, control of cardiovascular diseases (DEBMANDAL, 2011). Studies have shown that coconut oil is often used in the treatment of obesity by having a high content of medium chain fatty acids, which are easy to oxidation and are not stored in adipose tissues, thus aiding in the reduction of the basal metabolic rate (LIAU, 2011). There are also studies whose diets have large amounts of coconut oil, and showing that coconut oil intake does not influence the occurrence of dyslipidemia or obesity (AMARASIRI, 2006). A study examining the relationship between chain length fatty acids, postprandial satiety and food intake in lean men concluded that there is no evidence that the length of the chain fatty acids has an effect on appetite or food intake (POPPITT, 2010). Ingested fatty acids can contribute as a risk factor for cardiovascular disease, being amply demonstrated in several experimental and population studies (LANDS, 2008). In a Danish study compared diets rich in medium chain fatty acids (MCFA) and long chain fatty acids (LCFA) and the results showed that there was increase in total plasma cholesterol, LDL cholesterol and HDL cholesterol while preferred diet rich in MCFA. Moreover, the same study showed that a diet rich in coconut oil has helped raise insulin levels (ASSUNÇÃO, 2009). Given the increasing demand for products used in order to acquire numerous benefits and due to lack of information that prove such benefits, this study aimed to evaluate the effect of coconut oil in rats subjected to calorie and fat diet.

MATERIALS AND METHODS

Animals and experimental groups: Twenty-eight male Wistar rats weighing approximately 250 - 260 g were acclimated in the vivarium of the trial room of Caratinga University Center for a period of one week before starting the experiment. The animals were fed ad libitum food and water. The experiment was designed and conducted in accordance with ethical as norms the search committee with animals. The rats were divided into four groups of seven animals each, as follows: Group 1, high calorie diet; (II) Group 2 calorie diet plus coconut oil; Group 3, with commercial diet diet; (IV) Group 4 rats diet with commercial diet and treated with coconut oil.

diets: The standard vivarium diet, ie the commercial feed has the energy value of 3.8 kcal / g (carbohydrate 70%, protein 20% and fat 10%) and was administered to the rats of group 3:04. The cafeteria diet was produced considering described in Table 1.

Each week, the animals were weighed to adjust the preparation of diets, which were produced also considering the distribution of macronutrients. Thus, the foods were weighed as shown in Table 1 and ground in processor for the manufacture of the pellets. Suitable amounts of coconut oil (1 ml / kg / body weight) were added to the mass of the high calorie diet given to group 2 (calorie diet + coconut oil). For the group that was fed the commercial diet plus coconut oil, the oil was poured in suitable amounts over the feed pellets to be fed to the animals. The portions of calorie diet were packed in plastic film and maintained on cooling and withdrawn each day, according to the need of the consumer / day.

Preparation of coconut oil: The coconut oil used to treat the animals was purchased and prepared at room temperature (25 ° C) before being administered to the animals. They were treated daily for 54 days of the experiment with 1 ml / kg / coconut oil weight orally mixed with feed.

Experimental Procedures: Before starting the treatment, all animals were evaluated by the glucose syrup puncture with the aid of a blood glucose meter. The measures of blood glucose were held once a week until the day of sacrifice.

Collection and analysis of the material: After 54 days the animals were anesthetized with sodium thiopental 40 mg / kg injected intraperitoneally and in the blood, liver, intestines, pancreas and heart were removed for analysis. For biochemical evaluation, blood samples were collected and sent to the Clinical Laboratory of Caratinga University Center for blood glucose levels and lipidemic profile. From the results of the total cholesterol was calculated and fractions atherogenic index obtained from the following calculation: total cholesterol / cholesterol - HDL. The results were compared using the referrals Dantas *et al* (2006) and the Vivarium of FMUSP (2008). For histopathological examination, the liver of the animals was removed and fixed in formalin Calcium 10% for 24 hours, then transferred to 70% ethanol and processed for paraffin embedding and histological slides of preparation. For each animal, two slides was prepared containing four fragments with a thickness of 5 um.

Table 1. Composition of calorie diet used during the experimental period (grams)

Ingredients	Grams	CHO	LWA	LIP
kind of biscuit cornstarch	*50*	31.67	4.33	5.67
The carbonated drink cola	*50*	5	0	0
Roasted peanuts	*50*	10.85	11.6	25.45
Chocolate powder	*50*	23.33	6.67	15
neutral cake ready	*50*	30.3	3.6	3.75
Biscuit type water and salt	*50*	34.85	4.5	6.60
Cheese dish type	*100*	0	29.32	26.18
kind of Oreo	*50*	37.6	4.05	6
Condensed milk	*150*	83.25	11.7	13.5
Expessante cornstarch	*200*	70	0	0
Albumin in lyophilized powder	*100*	23	80	0.67
Granulated sugar crystal	*200*	199	0	0
Lard	*5*	0	0	5
Total	*1105*	548.85	155.77	107.81

From the diet composition verifies that this consists of 57.94% carbohydrates, 16.45% protein and 25.61% fat. The total caloric value corresponds to 3.43 Kcal / g, or 343 kcal per 100g of feed.

The slides were stained with HE and the slides were stained by PAS method for quantifying glycogen.

Biometry: To assess biometrics animals were weighed once a week until the day of sacrifice. To calculate the weight change was used in the following formula:

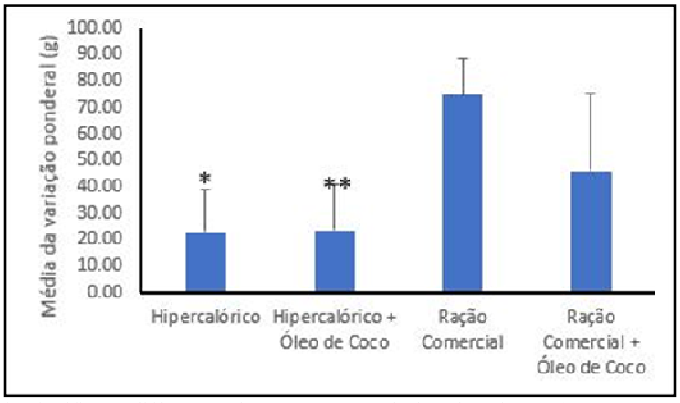
Weight change: Final weight (grams) - Initial Weight (grams) also evaluated the Lee index consisting of the cubic root of the division of the weight in grams nasoanal length in millimeters and multiplied by 1000. To be taken to correct analysis results, the values were considered below 0.300 are considered normal second Bernardis and Petterson (1968).

Statistical analysis: The results were analyzed using SigmaStat software by applying parametric tests. Differences were considered significant at P <0.05.

Ethical considerations: This project was submitted to the Ethics Committee on Animal Research of the University Center of Caratinga and approved by protocol number 001/2016.

RESULTS

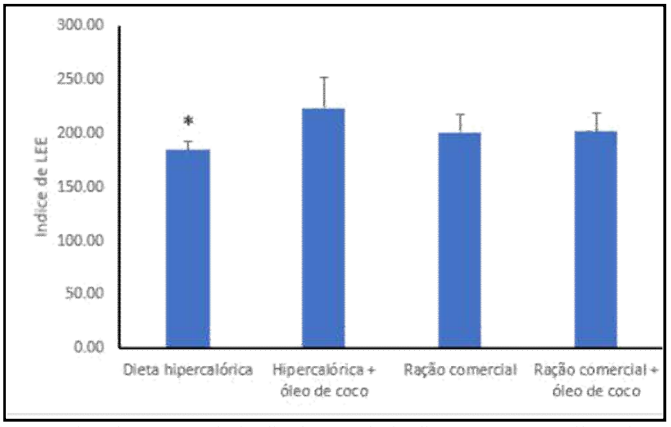
To evaluate the possible effects of coconut oil, animals received increased calorie diet or not with coconut oil 10 ml, later to produce the pellets, which were kept on refrigeration until ready to offer animals. The effects of induction of obesity by diet and calorie consumption of the role of coconut oil in the weight gain of the control was evaluated from weight change over the 54 days of the experiment and Lee index. These results can be seen in Figure 1 and Figure 2.



* Significant differences by ANOVA analysis of variance (F: 11.74, P: 0.0001) between the treated group and the group calorie diet diet with commercial diet (P <0.01 - Tukey method) ** Significant differences by ANOVA analysis of variance (F: 11.74, P: 0.0001) between the treated group calorie diet + coconut oil diet with commercial diet group (P <0.01 - Tukey method)

Figure 1. Average weight variation between the end of the experiment animals or non-induced obesity and treated or not with coconut oil

By analyzing Figure 1, it can be observed a significantly higher weight value in the group with the commercial chow diet (13,62g ± 74.71) compared to the group treated with the calorie diet (23.00 ± 15 90g) and the group that ingested the high calorie diet more coconut oil (23.57 ± 17,40g). Induction of animal obesity by high calorie diet was evaluated by Lee index and the results were shown in Figure 2. According Bernardis and Petterson (1968). Values for Lee index below 0.300 are considered normal. Considering that the average for the Lee index in all groups were below 0.300, the high calorie diet did not produce obesity in animals, in addition, animals fed high calorie diet plus coconut oil (222.71 ± 28.14) showed significantly higher rates than that seen in the group treated with calorie diet (184.15 ± 7.86). Considering the possible effect of coconut oil in preventing obesity with possible effects on the metabolism, we evaluated lipid levels, glucose and the atherogenic index of the different groups compared initially with Wistar rats to reference values obtained in other studies. The results may be seen in Table1.



*Comparing the mean calorie diet hypercaloric diet x + coconut oil (variance of Kruskal-Wallis test (H: 10.74, df 3, P: 0.013 by Dunn's method, and p <0.05)

Figure 2. Mean variation Lee index between animals with induced or obesity is not treated or not with coconut oil

Whereas plasma levels of total cholesterol, there is no significance in the comparison between the values obtained in the different groups, with all groups having an average of values below 100 mg / dL. When compared to the selected reference values, it is found that the total cholesterol levels of all groups are within the normal range for Wistar rats, as Dantas *et al* (2006) and the Vivarium of FMUSP (2008) But above the average found by the animal facilities of LTF - UFPB (2009). Regarding the findings related to HDL, there was no significant differences between the groups, but the values obtained in all of them are above the findings of Guimarães & Mazaro -UNIFESP (2004) But normal in relation to Caleiro findings (2012) (Table 1). Considering also the animal lipids described in Table 1, there were significant differences in the levels of cholesterol triglycerides, with the animals treated with increased commercial feed with coconut oil (90.14 ± 31.34 mg / dL) having values lower than observed in the groups fed with only the high calorie diet (188.14 ± 76,66mg / dL) and commercial feed (173.42 ± 45.79 mg / dL). When comparing these results with the reference values it appears that only the added animal feed group coconut oil to triglyceride levels were normal. When analyzing the records in table 1, there was no significant differences in values between groups atherogenic index, and they are all within the reference standards by Das *et al* (2006) and the values described for rats Vivarium of USP (2008). Considering the glucose values recorded in Table 1 identifies the occurrence of significant differences between groups. Thus, animals treated only with calorie diet (101.36 ± 5.14 mg / dL) had higher blood glucose levels to that observed in the group that was fed the commercial di*et al*one (82.38 ± 4.43 mg / dL) and that it was fed commercial diets with coconut oil (88.00 ± 6.20 mg / dL). Among the animals treated with high-calorie diet plus coconut oil (101.04 ± 5.16 mg / dL) also met blood glucose values of

the groups found in commercial and commercial feeds plus coconut oil. In comparison with the reference values all groups had lower values, suggesting normal levels of blood glucose. We also evaluated the deposition of glycogen in cytoplasm of hepatocytes and results are described in figures 3, 4, 5 and 6. The results of this analysis Histopathology showed that the animals treated with the commercial diet group showed only normal parenchyma formed by the hepatocyte cords interwoven sinusoidal capillaries that open in the central veins. There is also the technique of PAS there is a higher incidence of positive hepatocytes for glycogen storage in animals treated with commercial diet plus or not with coconut oil.

Among treated groups calorie diet with or without coconut oil, there appears to be a smaller distribution of hepatocytes positive for PAS, besides the occurrence of areas with respect degeneration. A Control fed commercial feed: observe hepatocytes cords PAS positive (arrow); 3B - Animals fed with commercial diet plus coconut oil: number of PAS-positive hepatocytes (arrows) and areas of degeneration with with fatty liver cells and PAS negative; 3C - Animals fed with high-calorie diet with various areas characterized as fatty degeneration and PAS negative mark (star). PAS few positive hepatocytes (arrows); 3D - Animal fed with high calorie diet plus coconut oil: negative PAS areas in hepatocytes with

Table 2. Biochemical parameters in male Wistar rats by comparing the values of three reference sources

parameters (unity)	*THIS STUDY RESULTS*				*Ref. 1*	*Ref 2*	*Ref 3*	*Ref 4*	*Ref 5*
	diet group calorie	Group calorie + coconut oil	commercial feed	commercial feed + Coconut oil					
Cholesterol (Mg / dL)	78.57 ± 9.73	83.71 ± 13.62	74.14 ± 5.93	68.00 ± 15.10	87 ± 18.1	62.2 ± 2.3	98.9 to 110.1	-	
Triglycerides (Mg / dL)	188.14 ± 76.66 *	163.85 ± 41.89	173.42 ± 45.79 **	90.14 ± 31.34	82 ± 24.7	98.0 ± 5.5	110 to 174.8	-	
Glucose (mg / dL)	101.36 ± 5,14Δ, ○	101.04 ± 5.16 ●, □	82.38 ± 4.43	88.00 ± 6.20	108 ± 17.4	117.4 ± 2.8	150 to 207.5	-	
HDL Cholesterol (Mg / dL)	52.86 ± 7.43	54.43 ± 7.30	51.71 ± 3.45	48.57 ± 8.40	-	-	-	16 ± 4.9	55.4 ± 5.5
Index therogenic	0.50 ± 0.17	0.53 ± 0.10	0.44 ± 0.13	0.39 ± 0.11	1.81	-	5.39	-	

*Significant difference in comparison Hypercaloric x commercial food coconut oil diet and the ANOVA analysis of variance (F: 4.98; P = 0.0081) and Tukey method P <0.0001

**Significant difference in comparison Commercial feed + x commercial food coconut oil by ANOVA analysis of variance (F: 4.98; P = 0.0081) and Tukey method P <0.0001

Δ significant difference in comparison Hypercaloric x commercial food by ANOVA analysis of variance (F: 22.85, P <0.0001) and Tukey method P <0.01

○ A significant difference in comparison between commercial food Hypercaloric x + coconut oil by ANOVA analysis of variance (F: 22.85, P <0.0001) and Tukey method P <0.01

● significant difference in comparison Hypercaloric x + coconut oil feed at commercial ANOVA analysis of variance (F: 22.85, P <0.0001) and Tukey method P <0.01

□ significant difference in comparison Hypercaloric coconut oil + x + commercial food coconut oil by ANOVA analysis of variance (F: 22.85, P <0.0001) and deTukey P <0.01 method

Reference 1 -Dantas et al (2006)[, Reference 2 - Vivarium of LTF - UFPB (2009)[12] Reference 3 - Vivarium of FMUSP (2008)[12] Reference 4 - Guimarães & Mazaro - UNIFESP (2004)[12], Reference 5 - Caleiro (2012).

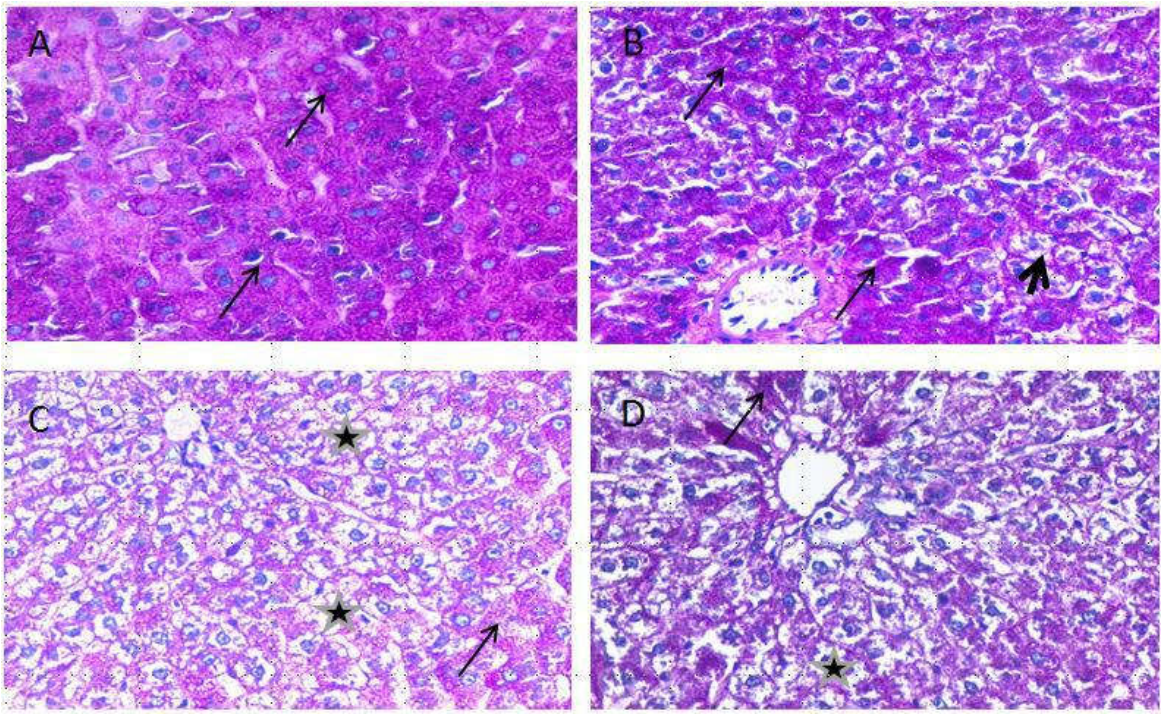


Figure 3. Liver sections stained with PAS glycogen for marking (400 times)

degeneration (star). In addition to the occurrence of some positive PAS hepatocytes (arrow).

DISCUSSION

The use of coconut oil in the experimental model proposed in our study showed no effect is in the control of obesity and metabolism of lipids and carbohydrates. It is believed that the supply of coolant for a period of 26 days may have contributed to the establishment of physiological changes in animals prevented to assess clearly the effects of coconut oil. This effect of chronic use of soda also interfered with the weight gain of the animals, since it was found that the calorie diet did not induce obesity in which is thus fed groups, in addition to having significantly thinner than those receiving commercial feed. Other authors described their work on the use of coolant may lead to slimming of rats as Oliveira *et al* (2017), that rats treated with commercial feed and liquid beverages as soda different type of 'glue', carbonated water and drinking water. They found that the animals treated with the refrigerant-type glue had during the experiment time, diarrhea, diuresis and increased weight loss compared to the other groups to analyze the esophagus and fragments observed intense inflammation. Mackenzie *et al*. (1992), to treat mice with Caramel IV, which is a component of soda, also identified this effect on inflammatory bowel. Diarrhea, increased diuresis and weight loss was also observed in our animals who used refrigerant, which led to the withdrawal of the diet in the twenty-sixth day of the experiment. Weight loss promoted in the first twenty-six days for refrigerant use should relate also to the fact that calorie diet did not have promoted obesity in animals, which is supported by the low values found for the Lee Index. This effect can relate to the results described by Santos *et al*. (2016), that to treat 31 animals divided into three groups: control, cola and carbonated water with daily doses of 150 ml of these liquid for evaluation of probable histopathological and biochemical changes in different organs of the digestive system, they observed changes in the liver, with areas of necrosis and steatosis, and in the large intestine, recorded regions with diffuse inflammation that ranged from moderate to severe. This impairment bowel contributed to malabsorption of nutrients with the consequent diarrhea and weight loss observed in our animals.

Although not been established obesity in mice, it was found that the calorie diet improved levels of triglycerides, blood glucose and HDL. This fact is consistent with published studies that say the cafeteria diet can alter the metabolism of lipids and carbohydrates. As in the study by Borba (2008). It tested the effect of a hyperlipidic-protein diet (HLP) with low carbohydrate content on body weight, organ weights, food consumption, serum biochemical parameters and histopathological changes in the liver of rats also reports an increase in triglyceride levels, and HDL in blood glucose. In another work of Yonn *et al*., (2008) there was also change log in the metabolism of lipids and carbohydrates to treat mice with fat diet. Our results are inconsistent regarding Caleiro findings (2012)[14]that evaluated the effect of coconut oil supplementation in 48 Wistar rats treated with high calorie diet. In this study, the author claims to have weight gain, accompanied by elevated triglycerides, LDL and HDL in animals treated only with the high calorie diet, however, treatment with coconut oil allowed a reduction in the levels of triglycerides and LDL and increased and HDL. The differences found between our results can be explained by the way that coconut oil was administered in our study it was offered at the same concentration used by Caleiro (2012)[14]Ie 1ml / kg, but given orally mixed in the feed and not by gavage. This may have affected the assessment of the effects of coconut oil. Narayanankutty *et al*. (2017), also evaluated the effect of coconut oil on the lipidêmico and glycemic profile of rats treated with high calorie diet. In its results describe that dyslipidemia and hyperglycemia-induced calorie diet were reversed with the use of coconut oil. In this experiment the authors also tendered coconut oil by gavage and extracted coconut oil almond in the laboratory. These different procedures might have contributed to the differences obtained in respect of our results. Thus, it is necessary to standardize for further studies the practice of gavage which can ensure consumer right amounts of coconut oil for animals and thus increasing the possibility of assessing their effects with less risk of bias.

The effect of high calorie diet on the liver described in our results was also observed by other authors such as Vera-Cruz *et al*., (2010), that evaluated the effect of green tea on body weight, glucose tolerance test (GTT) and the promotion of metabolic and morphological changes in the liver. It describes in its results the occurrence of macroscopic and microscopic changes in the liver of animals subjected to high calorie food including focal necrosis and accumulation of lipids suggesting hepatic steatosis. When you select the works to be used as reference value for the different variables analyzed in our study, the results were very different times among authors, as seen for HDL. About this variety in the biochemical, hematological factors and diet observed in experimental studies, says Dantas *et al*. (2006)[11]which can be determined by factors such as intra-species variations on geographical differences and handling. Therefore, the authors recommend that each laboratory animal facility or investigate and establish the reference values of normal control animals, according to lineage, sex, age of each species used.

Conclusion

Several factors related to experimental design defined in this study prevented to assess adequately the effects of coconut oil in animals subjected to high calorie diet. However, some results described draw attention as possible refrigerant effects included in the diet, which may have caused weight loss in animals due to severe diarrhea and increased diuresis. The impairment in glycogen stores and renal lesions were observed in groups treated with the high calorie diet. Moreover, it is necessary to evaluate in future studies the impact of this diet, including soda, renal function. Also recorded that despite the lower weight of animals treated with increased calorie diet or not with coconut oil, it was important biochemical changes such as increases in triglycerides and glucose levels in animals treated with a high calorie diet. Further work should be developed to determine the effects of coconut oil in animals subjected to high calorie diet, considering the form of administration by gavage.

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