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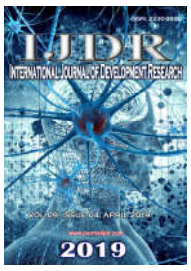

RESEARCH ARTICLE OPEN ACCESS

COCONUT OIL ACTION ON THE HEPATIC FUNCTION OF RATS Wistar SUBMITTED TO THE DIET HYPERCALORIC

[1]Lamara Laguardia Valente Rocha, [1]Farliany Alves Cimini Azevedo, [1]Wanessa Soares Luiz Silva, [1]Juscélio Clemente de Abreu, [2]Verônica Spani Amado, [1]Bruno Augusto de Rezende and *[1]Daniel Rodrigues Silva

[1]University Center of Caratinga
[2]Nurse of the Family Health Strategy of the State of Espírito Santo-Brazil

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ABSTRACT

Objective: To evaluate the effect of coconut oil on animals submitted to the hypercaloric diet considering its possible hepatoprotective effect. Method: Seven animals Were included in each of the experimental groups, totaling 28 animals: G1 (hypercaloric diet); G2 (+ hypercaloric coconut oil); G3 (control: commercial ration) and G4 (commercial diet + coconut oil) and sacrificed after 54 days of diet. Blood collection was Performed to Obtain the serum levels of transaminases, and tissue for histological analysis of the liver.Results: The hypercaloric diet with or without coconut oil did not please weight gain. However, the group fed with hypercaloric and coconut oil ration (9:30 22:49 ± grams) had Significantly higher than the visceral fat G3 (14.07 ± 4.11 grams) and G4 (13:01 3:53 ± grams). There was significant difference in the serum AST values between groups, but the greater G2 presented expression (223.00 ± 106.18U / L). Only animals fed commercial feed supplemented with coconut oil (72.00 ± 19:00 U / L) presented significant differences in relation to ALT levels When Compared to the hypercaloric diet groups (43.43 ± 19.12 U / L) and high calorie diet with coconut oil (46.43 ± 15:02 U / L). The liver sections of the animals submitted to the diet hypercaloric presented histopathological differences with each other by optical microscopy When Analyzed. Conclusion: Our results suggest of coconut oil did not present a hepatoprotective effect, however, other studies need to be developed to better elucidate this relationship. Only animals fed commercial feed supplemented with coconut oil (72.00 ± 19:00 U / L) presented significant differences in relation to ALT levels When Compared to the hypercaloric diet groups (43.43 ± 19.12 U / L) and high calorie diet with coconut oil (46.43 ± 15:02 U / L). The liver sections of the animals submitted to the diet hypercaloric presented histopathological differences with each other by optical microscopy When Analyzed. Conclusion: Our results suggest que coconut oil did not present a hepatoprotective effect, however, other studies need to be developed to better elucidate this relationship. Only animals fed commercial feed supplemented with coconut oil (72.00 ± 19:00 U / L) presented significant differences in relation to ALT levels When Compared to the hypercaloric diet groups (43.43 ± 19.12 U / L) and high calorie diet with coconut oil (46.43 ± 15:02 U / L). The liver sections of the animals submitted to the diet hypercaloric presented histopathological differences with each other by optical microscopy When Analyzed. Conclusion: Our results suggest que coconut oil did not present a hepatoprotective effect, however, other studies need to be developed to better elucidate this relationship. The liver sections of the animals submitted to the diet hypercaloric presented histopathological differences with each other by optical microscopy When Analyzed. Conclusion: Our results suggest que coconut oil did not present a hepatoprotective effect, however, other studies need to be developed to better elucidate this relationship. The liver sections of the animals submitted to the diet hypercaloric presented histopathological differences with each other by optical microscopy When Analyzed. Conclusion: Our results suggest que coconut oil did not present a hepatoprotective effect, however, other studies need to be developed to better elucidate this relationship.

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INTRODUCTION

The non-alcoholic fatty liver disease (NAFLD) is a condition of high prevalence, characterized by lipid accumulation in the liver when it exceeds 5-10% by weight (CALEIRO, 2012). The worldwide occurrence of fatty liver appears to be the most common liver disease in the Western world, physical inactivity, high anthropometric indices and obesity contribute to the emergence and development of NAFLD (FESTI, 2004). The liver diseases affect a very important organ in maintaining homeostasis, and its functionality influenced by por multiple genetic and environmental factors (MAGER, 2008). Currently, the condition is recognized as the hepatic component of the metabolic syndrome due to its strong association with obesity, dyslipidemia, hypertension and insulin resistance index (HOMA-IR) (TAKAHASHI, 2011) may vary from steatosis to steatohepatitis, fibrosis and cirrhosis (BAFFY, 2012). NAFLD occurs initially by lipid accumulation in the cytoplasm of hepatocytes, mainly in the form of triglycerides, and its prevalence increases considerably with the regionalization of the central body, one of the main factors of changes and metabolic comorbidities (CUPPARI, 2014 and VOLTERA, 2008). Already, the exacerbated metabolism of triglycerides, form reactive oxygen species may progress to liver inflammation (DIEHL, 2009). This inflammatory activity when progressive leads to the development of non-alcoholic steatohepatitis (NASH), characterized by the presence of steatosis and inflammation (VITAGLIONE, 2004).

The last stage is the most severe form of NASH, steatosis in which hepatocellular damage is associated with fibrosis and necrosis, can lead to cirrhosis and hepatocellular carcinoma (GUPTE, 2004). Several studies indicate an association between obesity and metabolic disorders, in particular the intake of high fat diet. In patients with hepatic fatty infiltration, Silva and Ribeiro (2016) observed high values of waist circumference (95%), triglycerides (46.4%), glucose (35.9%) and Assessment Model Index homeostasis (HOMA-IR, 33.3%), suggesting then verified that the liver changes are influenced by consumption of a high fat diet (SILVA, 2016). Regarding the distribution of nutrients, it also observed that fat is a major component of the human diet and the combination of fatty acids may not only influence the phospholipid profile of cell membranes, but also the composition of the triglycerides stored molecules in hepatocytes and adipocytes (ALMEIDA, 2013). The accumulation of lipids, and lead to major histopathological changes, may be associated with liver of enzime elevation. Several biochemical markers can be used paraavaliar liver changes. The transaminases aspartate aminotransferase (AST) and alanine aminotransferase (ALT) are the most frequently used parameters (STRANGES, 2004). AST is found mainly in the heart, liver, skeletal muscle and kidney, with distribution of both mitochondrial and in the cytoplasmic forms of cells, while the ALT is prevalent in the liver and to a lesser extent in the kidney, being exclusively cytoplasmic. It was shown that the elevation in serum liver enzymes, particularly ALT, has great importance as a specific marker of liver damage caused by injury or permeability changes in hepatocytes (LAI, 1995).

With all the risk factors and comorbidities that relate to obesity, the world population has shown growing concern about food, which encourages the search for food with hepatoprotective capacity, mainly because of the synthetic products carry adverse effects, where it has shown a substantial increase in intake of foods of plant origin (COTRIM, 2006). In the media, there is a great release on the beneficial effects of coconut oil (Cocos nucifera L.) in the form of metabolism to improve the lipid profile and reduce oxidative stress associated with obesity and dyslipidemia, as well as in the treatment of chronic diseases, such as liver disease, heart disease and cancer (DAUBER, 2015). Coconut oil is composed mostly (about 60 to 63%), medium chain triglycerides (MCT) 8 to 12 carbon atoms such as caprylic (C8: 0), capric (C10 : 0) and lauric (C12: 0), this oil also presents around 90% of saturated fatty acids, with a higher proportion of those 40 to 60%, represented by lauric acid (C12: 0) (RODRIGUES, 2005). Proponents of coconut oil consumption are based on the theory that the MCFA are easily oxidized lipids and not stored in adipose tissue, compared to LCFA, giving it effective in treatment against obesity (CRUZ, 2016 and MELLO, 2013). The use of coconut oil as phytonutrient have caused much discussion and controversy. Mello *et al*., (2013), points out that even beneficial to lipid profile, coconut oil because abdominal fat, a predictor of metabolic diseases (STROHER, 2017). Faced with the contradictory effects in relation to the actions of coconut oil in the metabolism is necessary development work to assist the understanding of these mechanisms. Coconut oil is composed mostly (about 60 to 63%), medium chain triglycerides (MCT) 8 to 12 carbon atoms such as caprylic (C8: 0), capric (C10 : 0) and lauric (C12: 0), this oil also presents around 90% of saturated fatty acids, with a higher proportion of those 40 to 60%, represented by lauric acid (C12: 0) (RODRIGUES, 2005). Proponents of coconut oil consumption are based on the theory that the MCFA are easily oxidized lipids and not stored in adipose tissue, compared to LCFA, giving it effective in treatment against obesity (CRUZ, 2016 and MELLO, 2013) The use of coconut oil as phytonutrient have caused much discussion and controversy. Mello *et al*., (2013), points out that even beneficial to lipid profile, coconut oil because abdominal fat, a predictor of metabolic diseases (STROHER, 2017). Faced with the contradictory effects in relation to the actions of coconut oil in the metabolism is necessary development work to assist the understanding of these mechanisms.

MATERIALS AND METHODS

Animals and experimental groups: Twenty-eight male Wistar rats weighing approximately 250 - 260 g were acclimated in the trial room of the University Center of Physiology Laboratory of Caratinga for a period of one week before starting the experiment. The animals were fed ad libitum food and water. The rats were divided into four groups of seven animals each, as follows: (G1) Group 1 mice fed with high-calorie diet; (G2) Group 2 rats hypercaloric diet and treated with coconut oil; (G3) Group 3, control rats and with commercial diet (G4) group 4 subject to commercial diet and treated with coconut oil.

diets: The standard vivarium diet, or the diet commercial show the energy value of 3.8 kcal / g (carbohydrate 70%, protein 20% and fat 10%) and was administered to the rats of group 3, and 4. The cafeteria diet was produced considering described in Table 1. The from the diet composition it is verified that 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 ração.

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

Ingredients	Grams	CHO	PTN	LIP
Cornflour biscuit	50	31,67	4,33	5,67
Coke	50	5	0	0
Roasted peanuts	50	10,85	11,6	25,45
Chocolate	50	23,33	6,67	15
Cake ready	50	30,3	3,6	3,75
Salty Craker	50	34,85	4,5	6,60
Cheese plate	100	0	29,32	26,18
Stuffedk cookie	50	37,6	4,05	6
Condensed milk	150	83,25	11,7	13,5
Cremogema	200	70	0	0
Albumin powder	100	23,0	80	0,67
Granulated sugar	200	199,0	0	0
Lard	5	0	0	5,00
Total	1105	548,85	155,77	107,81

A each week the animals were weighed in preparation for the adjustment of diets which were manufactured also considering the distribution of macronutrients. Thus, the foods were weighed as shown in Table 1 were milled in the processor for making 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.

Biometrics reviewed: To evaluate the biometric data of the animals was measured weight of the animal once a week until the day of sacrifice. To calculate the weight change used the following formula:

Weight change: Final weight (grams) Initial -Weight (grams): After 54 days the animals were anesthetized with sodium thiopental 40 mg / kg intraperitoneally injected in to remove the visceral fat. To calculate the visceral mass, pulled up and weighed to the epididymis fat and that deposited on the kidneys of the animal. The visceral fat was then estimated as:

Visceral Fat Weight (g): Epididymal fat (g) + kidney fat (g)

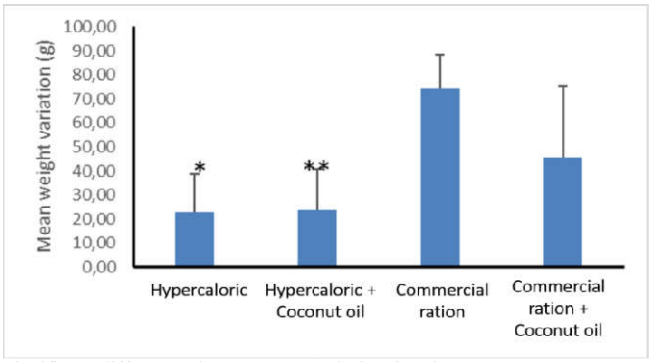
Collection and analysis of the material: After euthanasia, the liver was removed for histological studies. Samples of organs passed through the appropriate stages of histological processing and fixation, dehydration, paraffin embedding, the formation of paraffin blocks microtome in a thickness of 5µm, length of bath cuts, transfer to the blade, drying in a heating stage, Hematoxylin-eosin staining, and mounting of the slides for histopathological evaluation using the optical microscope coupled with a digital camera at 40x magnification rating for the presence of steatosis, according to Abbas *et al*. and Mello and Alves. For biochemical evaluation, blood samples were collected and sent to the Clinical Analysis Laboratory of the University Center of Caratinga. Transaminases to dosage, Serum ALT and AST were measured using commercial kits (Bioclin®, Belo Horizonte, MG.

Statistical analysis: Results were expressed as mean values and the standard deviation (SD). For the analysis of histological findings was used Kruskal-Wallis test. When there was no statistical difference was applied Mann-Whitney analysis to test pairs. To determine the significance of the other results it was used analysis of variance (ANOVA) for between group analysis and Newman-Keuls between two groups for analysis. The value of P <0.05 was considered significant.

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

RESULTS

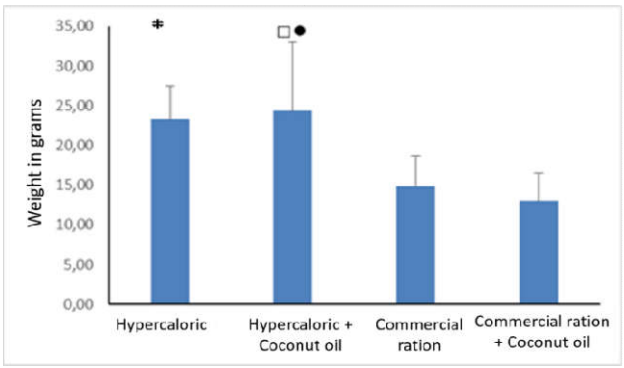
In the present study we evaluated the effect of coconut oil in animals subjected to high calorie diet considering its potential hepatoprotective effect.



* 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

Seven animals were included in each experimental group a total of 28 animals whose liver biopsy was evaluated, as well as the transaminase. Another parameter evaluated was related to weight variation and obtained the results shown in Figure 1. The results presented in Figure 1 you can see that the high calorie diet did not favor weight gain of the animals, either with the combination or not with coconut oil, not inducing obesity. It also appears that the animal groups fed with added caloric diet (23.57 ± 17,40g) or not (23.00 ± 15,90g) of coconut oil had significantly less weight than that observed in those animals treated with commercial d*iet al*one (74.71 ± 13,62g). However, compared to visceral fat gain, the animals in group 1 (high calorie diet) and group 2 (high calorie diet + coconut oil) showed significant increase compared to animals that only fed the commercial diet or increased commercial feed with coconut oil (Figure 2).



* Significant difference in comparing calorie diet + x commercial feed coconut oil by analysis of variance (F: 7.17, p: 0.0020, Tukey method p <0.05).
□ significant difference when comparing calorie diet + x commercial coconut oil feed by analysis of variance (F: 7.17, p: 0.0020, Tukey method p <0.05).
● significant difference when comparing calorie diet coconut oil + x + commercial feed coconut oil by analysis of variance (F: 7.17, p: 0.0020, Tukey method p <0.05).

Figure 2. Average weight in grams of the total visceral fat of the mice fed with high-calorie diet or not and treated or not with high calorie diet

Among the animals that were fed a high-calorie diet weight was visceral fat (21.78 ± 5.45) g, with significantly greater than that observed in the group fed on commercial diet plus coconut oil (1.13 ± 3.53 grams).

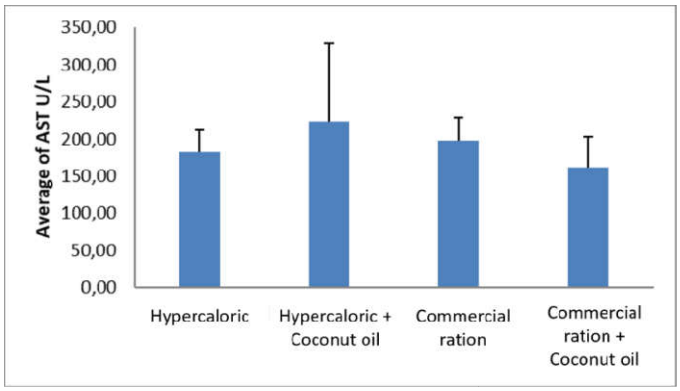
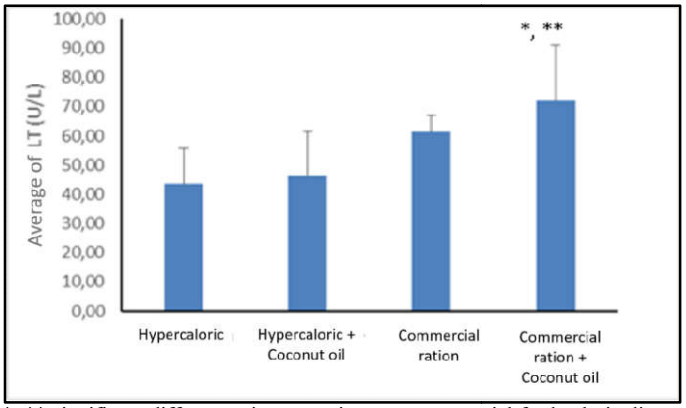


Figure 3. AST mean plasma values between rats fed with high-calorie or not and treated or not with coconut oil diet

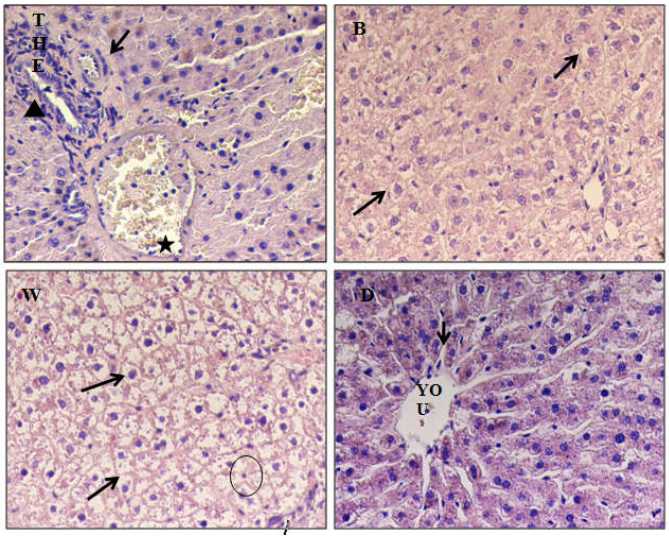
The group fed with high-calorie feed and coconut oil (22.49 grams ± 9.30) was significantly higher than the visceral fat of the animals from the group fed only commercial diet (14.07 ± 4.11 g) and commercial diet with coconut oil (01.13 ± 3.53 grams). When considering data related to liver function was evaluated the serum levels of transaminases, including AST, whose results are depicted in Figure 3.According to the results described in Figure 3, AST values at the different groups is presented not significantly different, ranging from 161.14 ± 42,14U / L (commercial diet + coconut oil) 223.00 ± 106,18U / L (+ calorie coconut oil). It was also evaluated the serum levels of ALT enzyme and the results were recorded in Figure 4.



*, ** significant differences in comparisons: x commercial feed calorie diet + coconut oil (Turkey method, p <0.01) and more than xração calorie commercial coconut oil + coconut oil (Turkey method, p <0, 05) test by ANOVA (F: 6.46, p: 0.0026).

Figure 4. Mean ALT values in plasma from mice fed with high-calorie or not and treated or not with coconut oil diet

At shown in Figure 4 it can be seen that only those animals fed with commercial diet plus coconut oil (72.00 ± 19.00 U / L) that is significantly different compared to ALT levels compared to the diet groups hypercaloric (43.43 ± 12.19 U / L) and high calorie diet with coconut oil (46.43 ± 15.02 U / L). Also evaluated in this study liver condition from the histopathological analysis of animal body fragments of different groups (Figure 5). In the biopsy of animals fed only with commercial diet there has been no change or hepatic lesion with all animals showing normal parenchyma with continuous mass hepatocytes with rounded cores, apparent power plants and nucleoli, forming cell cords around the central veins, the network sinusoid capillaries, whose walls were observed the presence of Kupffer cells.



See Appendix Figure 05 High Resolution
Figure 5. liver sections stained with hematoxylin-eosin (400 times)

A- 5 Control fed with commercial feed: Observe the normal parenchyma with well distributed hepatocytes with central round nuclei and evident nucleoli. The door system checks the presence of bile duct (triangle), hepatic artery (arrow) and vein (star).
5B Group + calorie coconut oil diet: Note the loss of the normal front of the control architecture. Hepatocytes exhibiting lipid vacuoles in the cytoplasm (arrows).
5C-Animals subjected to high calorie diet. Presence of hepatic degeneration, areas with ballooning of hepatocytes (arrows), with the presence of lipid droplets suggesting steatosis, besides the occurrence of nuclei with varying sizes and shapes, and pyknotic with condensed chromatin (circle). steatosis areas.
5D-Group commercial diet + coconut oil. The presence of central vein (CV) with strands of hepatocytes ecapilares sinusoids with Kupffer cells. steatosis areas.

The door system verified the presence of the bile duct, hepatic artery and portal vein. In calorie diet groups treated with or without the presence of coconut oil, we observed changes suggestive of liver steatosis, by analyzing the material stained with hematoxylin-eosin. Among the structural changes observed in these animals were identified areas of ballooning of hepatocytes, with the presence of droplets suggesting steatosis, besides the occurrence of nuclei with sizes and shapes, with more condensed and pyknotic chromatin. Some cells showed very disorganized, it is not possible to verify the presence of solid mass cords of hepatocytes, including no core, suggesting necrotic areas.

DISCUSSION

In this study, we sought to identify the effects of coconut oil on the possible changes in Wistar rat liver (Rattus norvegicus) after being subjected to high calorie diet. Our results show that coconut oil has not hepatoprotective effect, not interfering with the installation of the observed hepatoesteatose. The absence of the beneficial effects of coconut oil noted here confirms other results as revised by Santos *et al.* (2013), state that although coconut oil brings benefits over the elevation of HDL, its hypercholesterolemic effect can It is confirmed by other studies (SANTOS, 2011). Lecker *et al.*(2010) developed a study of guinea pigs compared the use of coconut oil with olive oil and sunflower oil so obtained, results in the group

treated with coconut oil showed a significant increase in HDL and triglyceride fraction does not (Lecker, 2015). Hann, and Martins Dias (2014) developed a study with the objective of identify key evidence of the use of coconut oil, safflower oil and conjugated linoleic acid in body fat reduction. In this systematic review, claim that no evidence was found to ensure that coconut oil decrease body fat (HANN, 2014). However, to better analyze the results found in our study is necessary to assume a possible bias on the intake of the necessary amounts of coconut oil, since this supplement was not offered by gavage, which may have interfered in our findings. Regarding the changes in body weight, it was found that animals fed increased calorie diet or not with coconut oil, showed no weight gain, with the exception of the control group treated with commercial diet. This fact can be justified by the rejection presented by rats compared to solid diet, consumed coolant preferably over the initial 27 days of the experiment. They showed signs of clinical disorders such as diarrhea, diuresis and increased weight loss. This may be related to intestinal involvement refrigerant and be the cause of weight loss. This hypothesis presents consistent with that described by (Santos *et al*, 2016; Machenzie *et al*, 1992), where the relation between refrigerant intake and weight has not been established.

Despite the absence of weight gain observed in our animals, there was an increase of visceral fat among those that fed on high calorie diet, especially among the group put on high calorie diet + coconut oil. These findings are consistent with the diagnosis of metabolic syndrome, which also includes fatty liver disease. [27] Furthermore, Piano *et al*., 2015 found that adolescents with visceral obesity and elevated levels of HOMA-IR, had a higher risk of developing NAFLD noting that every increase of 1 cm in visceral adipose tissue was associated with a two times higher risk of NAFLD in obese adolescents, confirming that central adiposity is associated with chronic low-grade inflammation, which accelerate insulin resistance and fat accumulation hepatocellular (DÂMASO, 2008). Yet, in experimental work conducted with Wistar rats, male and female, underwent cafeteria diet (Caleiro, 2012), found that supplementation with coconut oil in obese mice showed lipid-lowering effects, predominantly in males. However, the practice was not effective in reducing body weight in both genders, and promote greater accumulation of abdominal fat in males, a risk factor for heart disease. Therefore, it is inferred, consider what the desired benefit, gender and the possible effects that this supplement may produce (CALEIRO, 2012). Therefore, many authors suggest that abdominal lipid accumulation, regardless of the total of the individual adiposity is related to ectopic lipid deposition, especially in the hepatocyte, and therefore predictive factor in the pathogenesis of NAFLD (CAVE, 1995). However, Liau and employees, (2011), noted reduction of abdominal fat in humans supplemented with virgin coconut oil, however, it is noteworthy that supplementation was short compared to the addressed protocol (only 30 days) (MUNHOZ, 2017). Animal studies have shown a strong association between dietary composition and the development of hepatic steatosis, also shown in our work. So it has been reinforced that inadequate diets can promote NAFLD in humans. The mechanisms by which the diet may play this role include modulation TAG accumulation in the liver and regulation of antioxidant activity as well as changes in insulin sensitivity and triglyceride metabolism (CAVE, 2007). Patients with fatty liver disease often discover by chance, through imaging studies to investigate other diseases. The suspicion is generally associated with elevated levels of markers of liver injury (AST, aspartate aminotransferase, and ALT, alanine aminotransferase) in the absence of alcohol and exclusion of other liver diseases. However, these injury markers may show normal levels (CAVE, 2007). Being a slow developing disease, patients with steatosis individuals are for the most part, asymptomatic and only about 20-50% show changes in transaminases, which can make diagnosis difficult (DÂMASO, 2008). In experimental work carried out on rats submitted to high calorie diet, Mello., (2016) evidenced by serum analysis of the enzymes aspartate aminotransferase (AST) and alanine aminotransferase (ALT) that rats fed with cafeteria diet showed one lipid percentage higher than the group fed the control diet. However, this accumulation of lipids was insufficient to cause changes in serum enzymes analyzed, showing no one liver damage frame, corroborating our findings (MUNHOZ, 2017). Analysis evidenced by serum enzymes aspartate aminotransferase (AST) and alanine aminotransferase (ALT) that rats fed with cafeteria diet showed a lipid percentage higher than the group fed the control diet. However, this accumulation of lipids was insufficient to cause changes in serum enzymes analyzed, showing no one liver damage frame, corroborating our findings (MUNHOZ, 2017), Analysis evidenced by serum enzymes aspartate aminotransferase (AST) and alanine aminotransferase (ALT) that rats fed with cafeteria diet showed a lipid percentage higher than the group fed the control diet. However, this accumulation of lipids was insufficient to cause changes in serum enzymes analyzed, showing no one liver damage frame, corroborating our findings (MUNHOZ, 2017). Comparing the results for the transaminase in our study, with reference AST levels to male Wistar rats, healthy, weighing between 140 to 310g showed by Lima *et al*, (61-210 U / L 2014). [31], it appears that all groups had serum AST values within normal range, except for the group of animals with increased calorie diet coconut oil, where it is registered values above the normal range. Thus, it is believed that organs other than the liver of animals belonging to the treated group increased calorie diet of coconut oil may have suffered some injury, which demand new studies to confirm this suspicion. When considering as ALT values for reference levels Wistar rats 38-82 U / L Lima *et al*., (2014) [31] there is none of the experimental groups used in our study showed normal values different. However, considering as reference value the findings described by Melo *et al* (2012) [32] for defining aminotransferase AST aspartate from adult male Wistar rats levels of 81-180 U / L.; 2012 and ALT aminotransferase aalanina the amount would be 36-58 U / L, indicate different results, it would point to high levels of AST in rats consuming high calorie diet plus coconut oil and those who ate commercial food with coconut oil. Would also change the results for ALT, would occur high among animals fed commercial feed. Thus, justifies the need to determine the specific reference values for each animal house. [33]

Conclusion

Our results suggest that coconut oil did not show hepatoprotective effect, however, other studies need to be developed to better elucidate this relationship. Although not have conclusive results on action of coconut oil in animals at high calorie diet, our findings point to the effect of high fat diet on liver function, although no significant changes in transaminases, show histopathological changes consistent with hepatoesteatose.

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