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WEIGHT GAIN AND METABOLIC CHANGES IN OBESE MICE FED WITH LYOPHILISEDPITAIA (HYLOCEREUS UNDATUS)

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ABSTRACT

The objective of this study was to investigate the influence of white pitaya (Hylocereus undatus) lyophilised in mice fed with high-fat diet to induce obesity. The animals were divided in groups (n=10), fed with control diet (C1), high-fat diet (C2), high-fat diet with lyophilised white pitaya at 2% (P2) and 4% (P4) during 12 weeks. It was calculated weight gain, food consumption and Feed Efficiency Ration (FER). The glucose tolerance test was performed. The histology of the liver and epididymal adipose tissue were analyzed. Fasting glycemia, lipid profile, and volume density of hepatic steatosis were measured. No influence of lyophilized white pitaya on weight gain and glucose tolerance in mice were observed. However, the lyophilised pitaya induced a greater satiety and showed improvement in the lipid profile, especially on values of HDL (increase of 72, 27% - P4) and triglycerides (decrease of 43,68% - P2). In addition, it presented a small improvement in liver impairment caused by excess saturated fat and simple carbohydrates. The consumption of pitaya did not contribute to reduce weight gain, however, it reduced the food consumption and improved the lipid metabolism even in the face of an unbalanced diet, rich in saturated fats and simple carbohydrates.

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INTRODUCTION

Obesity is a public health issue that encompasses populations around the world. According to data from the World Health Organization (WHO), in 2014 more than 1.9 billion adults (18 years or more) were overweight, with 600 million classified as obese (WHO, 2016). According to the publication made by the Brazilian Association for the Study of Obesity and Metabolic Syndrome (ABESO) (DBO/ABESO, 2016), obesity is equivalent to the excessive accumulation of adipose tissue increasing the risk for cardiovascular diseases; in addition, it is a multifactorial disease that involves genetic, environmental, emotional and, especially, behavioral factors. In the behavioral, we emphasize the sedentarism and the bad eating habits.

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Not just obesity, but the consequences of obesity cause a series of metabolic alterations detrimental to the body. In addition to the change in the lipid profile, with elevated total cholesterol and triglycerides, accumulation of visceral fat and excess free circulating fatty acids, a chronic subclinical inflammation and insulin resistance may develop, reducing the uptake and alteration in the glucose metabolism leads to type 2 diabetes (Grundy, 2015). The dietary treatment is done in the form of reeducation of the alimentary habit. The effectiveness of nutritional reeducation occurs as food planning is incorporated into the routine of the individual, affecting their food choices that also reflect caloric amounts. On this occasion, fruits and vegetables are included in quantities already recommended by the Food Guides, generally have low calorie, high fiber content and nutrients that can act as antioxidants. Being of extreme importance in the treatment and prevention of obesity (DBO/ABESO, 2016). Some fruits have been investigated for contributing to the treatment of obesity and its complications

(Kowalska and Olejnik, 2016). Studies reveal the presence of bioactive components and seek to clarify their effects on the human body. A physical-chemical analysis (Sato *et al*., 2014)showed the low caloric density of the fruit, together with a high content of insoluble fibers, besides micronutrients like vitamin B1, B2 and B3, calcium, zinc, phosphorus, potassium and magnesium. Found the presence of high levels of linoleic acid in the pitaya seed (*Hylocereus undatus*),also possessing palmitic, stearic, oleic and linolenic acid. This set of characteristics has benefits in the treatment of obesity and oxidative stress. (Nunes *et al*., 2014). In addition, the antioxidant capacity of pitaya has attracted the attention of researchers (Ariffin *et al*., 2009; Mello *et al*., 2015)for its bioactive components such as betalain, a purplish pigment with high free radical scavenging ability, for its structure containing aromatic rings with hydroxyl groups (Mello *et al*., 2015). Thus, the aim of this study was to evaluate the influence of white pitaya (*Hylocereus undatus*) lyophilised in mice fed with high-fat diet to induce obesity.

MATERIALS AND METHODS

Animals and diets: All procedures were approved by the Ethics Committee on the Use of Animals and improved of Federal University of Mato Grosso do Sul (UFMS) nº 824/2015, in accordance with the National Council for the Control of Animal Experimentation (CONCEA), and with the current legislation and other provisions of animal research ethics. Forty male mice strain C57BL/6, purchased from the vivarium of UFMS, 8 weeks old. Before starting the experiments, the animals were acclimated to the test environment for 5 days and placed in polypropylene cages each with 4 animals, lined with wood shavings. The cages were kept in an environment with controlled light, humidity and temperature. The temperature was maintained at around 22 ± 2ºC, with a 12-12 h light-dark cycle, except for group 1, which had a 10-14 h light-dark cycle, maintained at 24 ± 2ºC. All groups were given food and water *ad libitum.* The animals were divided into four groups, with 10 animals each. The groups received differentiated diets for each group, as specified in Table 1, for 12 weeks. The percentages of 2 and 4% were determined from the study of (Lenquiste *et al*., 2012). Control of feed and water intake was done every 3 days, and control of weight gain was recorded weekly. From these data the weekly weight gain, food consumption and the Feed Efficiency Ration (FER) were calculated, using equation (Aguila *et al*., 2003) (Table 2).

Oral Glucose Tolerance Test (OGTT): An oral glucose tolerance test (OGTT) were performed on food-deprived (12h). After 12 weeks of tratament. Blood glucose levels were measured with an Accu-chek handheld glucometer using appropriate test strips. For the OGTT, a solution of 50% D-glucose (2 g/kg body weight) was administered oraly. Blood samples were collected from the tail vein at 0, 15, 30, and 120 min for determination of glucose concentrations (Santos *et al*., 2008). The area under the curve of glucose was calculated.

Euthanasia and colletion of material: After tree days of Oral Glucose Tolerance Test (OGTT), the animals were anesthetized with ketamine and xylazine (15 mg/kg: 75 mg/kg) and euthanized. The blood samples was collected via retro-orbital micro-tube containing gel separator, which later were centrifuged at 4000 rpm for 5 min, and the serum was separated and stored for further analysis. The animal was placed in a gas chamber containing CO_2 for confirmation of death. After confirmation, the liver and five sites of adipose tissue were collected, of which epididimal, omental, perirenal, retroperitoneal and mesenteric. All tissues collected were weighed in a semi-analytical balance (Bel®) and annotated in table.

Biochemical analysis: The colleted serum was processed with kit Labtest® according to manufacturer's instructions. For the biochemical dosages the formulas described in Table 2 were applied to obtain the values triglycerides, total cholesterol, glucose, HDL (high density lipoprotein), VLDL (very low density lipoprotein) cholesterol and LDL (low density lipoprotein) (Florence *et al*., 2014).

Histological analysis: For histological analysis, the liver tissue and the epididymal adipose tissue were placed in a solution of formaldehyde 10%, were fixed and stained with hematoxylin-eosin. Slide images of liver sections were captured with Leica Application Suite, version 4.0.0 [Build: 877], at 200x magnification. The analyzed for the presence of hepatic steatosis by a test system consisting of 36 points (Pt), by software STEP*anizer*© stereology tool, version 1.0, which is used to estimate the volume density (Vv) of non-alcoholic hepatic steatosis, from the number of points that (Pp) the fat droplets in hepatocytes. The points counted were placed in the formula described in Table 2 (Catta-Pieta *et al*., 2011).For the analysis of adipocytes, the areas of 100 adipocytes were measured with the aid of *software* LEICA *Application Suite* versão4.0 (Leica Microsystems, Wetzlar, Germany,). The result represents of the mean area of the 100 adipocytes.

Statistical analysis: The results were expressed as mean ± standard deviation, when the numerical variables had normal distribution. Analysis of variance (ANOVA) was used to compare the groups. When a statistically significant difference was revealed, the analysis was complemented by the Tukey test, calculated with the use of the Jandel-Sigma Stat program, to reject the null hypothesis at the 5% level.

RESULTS

Among the groups in the first, second and third month, there was no significant difference for the weight gain. However, analyzing the total weight gain, the P2 and P4 groups presented greater weight gain when compared to the C1 group (Table 3).Food intake was lower in groups P2 and P4, when compared to C1 and C2, from the second month of experiment.This result was also significant in total food intake ($p < 0.001$). Analyzing the FER, in the second month of study, the P4 group presented higher food efficiency in relation to the C1 group ($p = 0.018$). For the total FER, groups P2 and P4 presented higher values in relation to group C1 and C2 ($p < 0.001$). The groups that received a hypercaloric diet (C2, P2 and P4) showed an increase in the weight of the epididimal and retroperitoneal tissues compared to the C1 group, as shown in Table 3.In addition, the P4 group had numerically lower values than the P2 group for all tissues. the relative weight of the liver, only the P4 group had a lower significant value in relation to the C1 and C2 groups.In the evaluation of the adipocyte area the C1 group was smaller than the P2 group. Through the method of counting the points for the diagnosis of hepatic steatosis, there was no difference between the groups ($p = 0.086$); however, the groups containing the pitaya had the lowest percentage of the group compared to the C2 group.

Table 1. Percentage composition of the diet offered to the groups

Feed composition				
Ingredient	Control 1	Control 2	Pitaya 2%	Pitaya 4%
Comercial ration Nuvital®	100%	51.1%	49.1%	47.1%
Fat	0	25.9%	25.9%	25.9%
Currant	0	23%	23%	23%
liophylized Pitaya	0	0	2%	4%
Total	100%	100%	100%	100%

Table 2. Formulas for calculation of the Feed Efficiency Ration (FER), biochemical parametersand calculation of Volume Density of hepatic steatosis

Parameter	Formula
FER	weight gain (g) ÷ food consumption (g)
Triglycerides	Absorbance of the test ÷ standard absorbance x 200
Total Cholesterol	Absorbance of the test ÷ standard absorbance x 200
Glucose	Absorbance of the test ÷ standard absorbance x 100
HDL	Absorbance of the test ÷ standard absorbance x 40
VLDL	Triglycerides ÷ 5
LDL	Total Cholesterol - (HDL + VLDL)
Volume density of steatosis (Vv)	Overlapping points (Pp) ÷ Total points (Pt)

Table 3. Monthly weight gain, total weight gain, monthly and total Feed Efficiency Ration(FER) of control and treated animals with pitaya

Parameter	Groups				
	Control Group 1 (n = 9)	Control Group 2 (n = 10)	Pitaya Group 2% (n = 9)	Pitaya Group 4% (n = 10)	p
Weight gain month 1 (g)	1,89 ± 0,42	1,30 ± 0,42	2,55 ± 0,58	2,00 ± 0,49	.0,348
Weight gain month 2 (g)	1,33 ± 0,33	1,90 ± 0,48	2,89 ± 0,65	3,00 ± 0,36	0,052
Weight gain month 3 (g)	1,22 ± 0,32	1,80 ± 0,66	2,44 ± 0,53	3,00 ± 0,82	.0,230
Weight gain month (g)	$4,44 \pm 0,58^{a}$	$5,00 \pm 0,68^{a,b}$	$7,89 \pm 0,67^{b}$	$8,00 \pm 1,04^{b}$	0,003
Food consumption month 1	$104,66 \pm 4,40^{a}$	$99,20 \pm 3,09^{a,b}$	$90,89 \pm 2,56^{b}$	$88,70 \pm 2,42^{b}$	0,004
Food consumption month 2	$104,55 \pm 3,36^{a}$	$102,24 \pm 3,04^{a}$	$88,22 \pm 2,85^{b}$	$95,00 \pm 1,96^{a,b}$	0,001
Food consumption month 3	$103,33 \pm 1,32^{a}$	$103,40 \pm 4,57^{a}$	$91,44 \pm 1,02^{b}$	$85,00 \pm 1,39^{b}$	<0,001
Total Food consumption	$312,55 \pm 6,00^{a}$	$304,83 \pm 9,68^{a}$	$270,55 \pm 3,80^{b}$	$268,69 \pm 4,57^{b}$	< 0,001
FER month 1	0,02 ± 0,003	0,01 ± 0,004	0,03 ± 0,006	0,02 ± 0,005	.0,199
FER month 2	$0,01 \pm 0,003^{a}$	$0,02 \pm 0,005^{a,b}$	$0,03 \pm 0,007^{a,b}$	$0,03 \pm 0,004^{b}$	.0,018
FER month 3	0,01 ± 0,003	0,02 ± 0,007	0,03 ± 0,005	0,03 ± 0,009	.0,115
Total FER	$0,01 \pm 0,002^{a}$	$0,02 \pm 0,002^{a}$	$0,03 \pm 0,002^{b}$	$0,03 \pm 0,003^{b}$	< 0,001

Values represent mean ± standard error of mean. ANOVA, followed by Tukey's post-test. Different letters on the same line represent significant differences between groups.
Group C1 fed with commercial diet, Group C2 fed a high-fat diet, group P2 fed a high-fat diet with 2% pitaya and group P4 fed a high-fat diet with 4% pitaya.

Table 4. Effects of pitaya lyophilized on liver weight, five sites of adipose tissue (TA), total site weight and adipocyte area of epididymal adipose tissue and steatosis volume density

Parâmetro	Groups				
	Control Group 1 (n = 9)	Control Group 2 (n = 10)	Pitaya Group 2% (n = 9)	Pitaya Group 4% (n = 10)	p
Liver (g)	$1,13 \pm 0,03^{a}$	$1,13 \pm 0,047^{a}$	$1,01 \pm 0,02^{a,c}$	$0,98 \pm 0,03^{b,c}$	0,006
TA Epididimal (g)	$0,33 \pm 0,02^{a}$	$0,99 \pm 0,12^{b}$	$1,12 \pm 0,12^{b}$	$1,07 \pm 0,19^{b}$	< 0,001
TA Omental (g)	0,06 ± 0,01	0,07 ± 0,01	0,07 ± 0,01	0,05 ± 0,01	0,560
TA Retroperitoneal (g)	$0,10 \pm 0,01^{a}$	$0,53 \pm 0,06^{b}$	$0,55 \pm 0,06^{b}$	$0,48 \pm 0,09^{b}$	< 0,001
TA Mesenteric (g)	$0,21 \pm 0,02^{a,c}$	$0,39 \pm 0,04^{b,c}$	$0,45 \pm 0,06^{b}$	$0,41 \pm 0,07^{b,c}$	0,049
TA Perirenal (g)	$0,05 \pm 0,01^{a,c}$	$0,12 \pm 0,01^{b}$	$0,12 \pm 0,02^{b}$	$0,12 \pm 0,02^{b,c}$	0,039
Total fat (g)	$0,75 \pm 0,05^{a}$	$2,10 \pm 0,20^{b}$	$2,32 \pm 0,24^{b}$	$2,12 \pm 0,37^{b}$	< 0,001
Area of TA Epididimal (μm^2)	$3518,97 \pm 1519,67^{a}$	$7222,11 \pm 636,04^{a,b}$	$8320,60 \pm 572,09^{b}$	$7379,42 \pm 1143,73^{a,b}$	0,033
Steatosis volume density (%)	59,25 ± 16,41	73,00 ± 3,43	55,20 ± 8,23	70,20 ± 11,44	0,307

Values represent mean ± standard error of mean. ANOVA, followed by Tukey's post-test. Different letters on the same line represent significant differences between groups. TA: adipose tissue. Group C1 fed with commercial diet, Group C2 fed a high-fat diet, group P2 fed a high-fat diet with 2% pitaya and group P4 fed a high-fat diet with 4% pitaya.

The group P2, with a 24.3% difference, as shown in Table 4. Analyzing the lipid profile, all groups obtained a significant increase in total cholesterol when compared to C1. For HDL cholesterol there was no significant difference in the comparison between the groups. However, the groups that consumed PIA had an increase in HDL cholesterol, with a difference of 47.19% for P2 and 72.27% for the P4 group.For the triglycerides, the groups that received pitaya presented values without significant difference in relation to C1. However, the group P2 which had a more expressive reduction in relation to group C2 (43.68% less). For LDL and VLDL quantifications there was no significant difference (Table 5). Regarding glycemia, in the OGTT, there was no significant difference between the different times of analysis of the glycemic level as recorded in Table 6. In the fasting glycemia (T0), the P2 and P4 groups had smaller means than the C2 group.

Table 5. Fasting glycemia and serum lipids of control and treated mice with pitaya

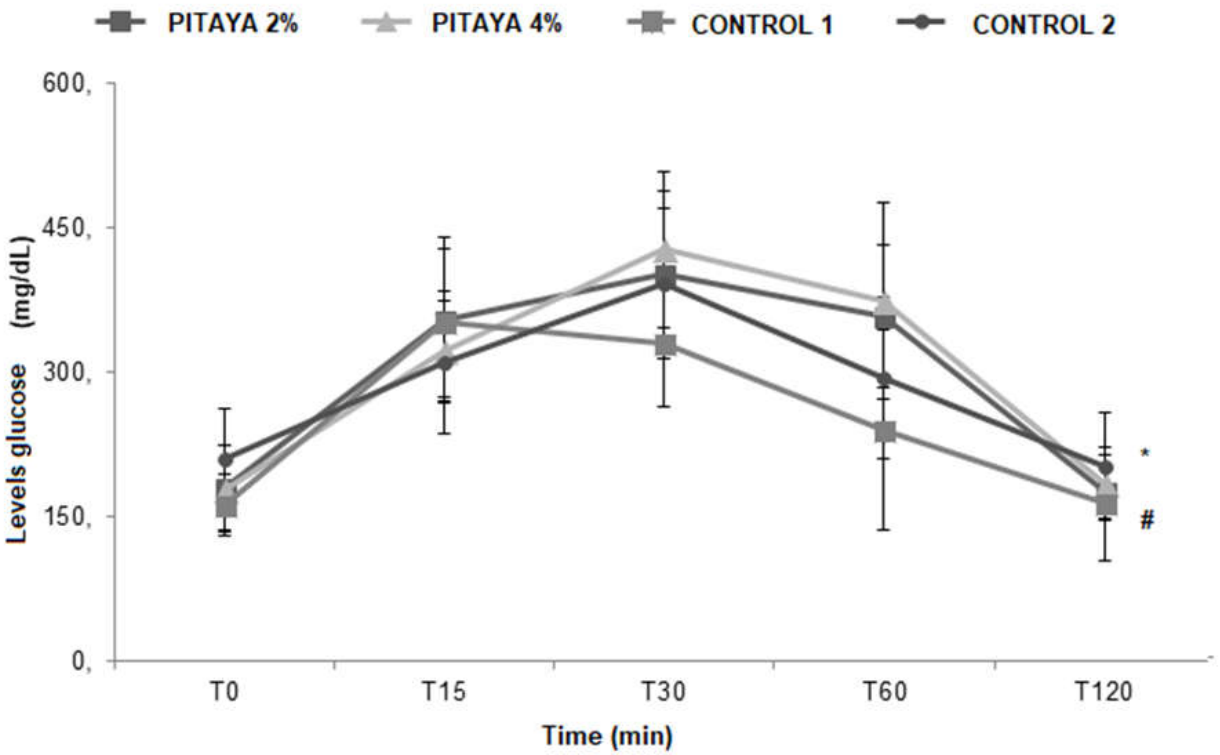
Parameter	Groups				
	Control Group 1 (n = 9)	Control Group 2 (n = 10)	Pitaya Group 2% (n = 9)	Pitaya Group 4% (n = 10)	p
Fasting glycemia (mg/dL)	87,38 ± 10,15	107,24 ± 17,05	108,82 ± 9,21	121,07 ± 12,59	0,287
Triglycerides (mg/dL)	84,32 ± 3,36[a,c]	158,50 ± 25,86[b]	89,26 ± 8,14[b,c]	126,17 ± 26,79[b,c]	0,030
Total Cholesterol (mg/dL)	120,21 ± 6,65 [a]	199,51 ± 7,84 [b]	213,98 ± 28,24 [b]	202,09 ± 13,16 [b]	0,002
HDL (mg/dL)	41,87± 6,47	43,46 ± 8,03	63,97 ± 12,86	74,94 ± 14,23	0,140
LDL (mg/dL)	68,00 ± 8,81	103,82 ± 18,21	132,15 ± 19,43	94,25 ± 30,20	0,236
VLDL (mg/dL)	14,99 ±1,96	17,85 ± 1,62	25,36 ± 5,87	20,19 ± 5,40	0,367

Values represent mean ± standard error of mean. ANOVA, followed by Tukey's post-test. Different letters on the same line represent significant differences between groups. Group C1 fed with commercial diet, Group C2 fed a high-fat diet, group P2 fed a high-fat diet with 2% pitaya and group P4 fed a high-fat diet with 4% pitaya.

Table 6. Glycemic level of mice submitted to administration of four different treatments, according to the time of capillary blood analysis

Colletion time	Groups				
	Control Group 1 (n=9)	Control Group 2 (n=10)	Pitaya Group 2% (n=9)	Pitaya Group 4% (n=10)	p
T0	161,89±10,57	210,80±16,63	180,88±14,75	179,10±14,39	0,131
T15	351,63±27,34	310,20±23,28	354,77±28,79	322,10±16,49	0,479
T30	329,88±23,18	392,50±24,77	402,33±29,02	428,20±25,65	0,085
T60	239,78±34,74[a,c]	294,44±27,88[b,c]	358,22±24,78[b]	375,00±32,09[b]	0,013
T120	163,89±19,57	201,90±17,77	175,55±8,78	184,10±9,68	0,329

Values represent mean ± standard error of mean. ANOVA, followed by Tukey's post-test. Different letters on the same line represent significant differences between groups. Group C1 fed with commercial diet, Group C2 fed a high-fat diet, group P2 fed a high-fat diet with 2% pitaya and group P4 fed a high-fat diet with 4% pitaya.



* Refers to the statistical difference between the groups C2 and P2 (p = 0,008)
\# Refers to the statistical difference between the groups C2 and P4 (p = 0,001)

Figure 1.Glycemic levels of mice with different treatments, according to the time of blood analysis. Values represent mean ± standard error of mean

In 60 minutes the glycemic level of the C1 group was significantly lower than that of the P2group (p = 0.008), as well as the P4 group (p = 0.001) (Figure 1). Table 6 shows that after glucose administration, there was an increase in glycemic levels in all study groups, with a subsequent reduction (p = 0.042). The area on the curve showed no difference between the study groups (p = 0.093) (Figure 2).

DISCUSSION

Obesity is characterized by the accumulation of adipose tissue increasing the risk for cardiovascular diseases. the cardiovascular diseases that are the main responsible for death in the world, being considered the target of research aimed at prevention and treatment are the focus of current research.

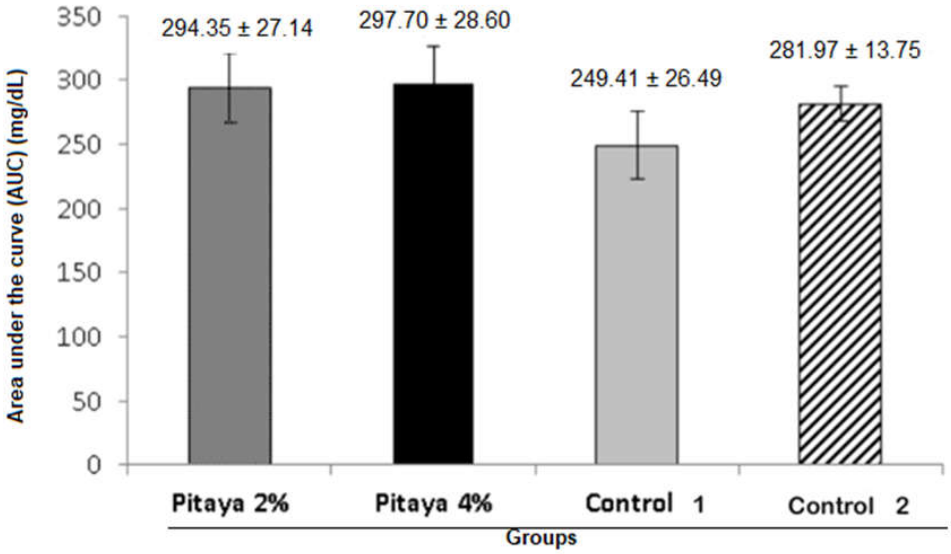


Figure 2. Area under the curve (AUC) obtained of Oral Glucose Tolerance Test (OGTT). Values represent mean ± standard error of mean

For this, animal models are developed with the aim of improving the understanding of the etiology of obesity(Write *et al*., 2013). Parallel to the development of models, is the investigation of possible treatments related to nutrition, with the main focus on foods with high antioxidant potential (Kowalska and Olijnik, 2016; Lenquiste *et al*., 2012). In this work, the mice strain C57BL/6, which presented an adequate response as an obesity model for diets rich in carbohydrates and fats (West *et al*., 1992; Winzell and Ahrén, 2004). In addition, this strain is also prone to developing diabetes along with obesity in response to the same diet. (Alexander *et al*., 2006). The high fat diet was used for low cost and also for good induction to obesity, being more indicated by researchers for the lipid composition of saturated fats (Write *et al*., 2013; Buettner *et al*., 2006). Currants were added to the diet to improve the palatability and acceptance of the diet of groups C2, P1 and P2, in addition to adding carbohydrates(White *et al*., 2013). Our results show that the diets used for induction were efficient in the development of obesity, evidenced by the higher food efficiency when compared to the commercial ration. The mice fed with high-fat diet ate less and had greater weight gain, according to the data in Table 3. The lower dietary intake observed in the treatment groups (P2 and P4) can be attributed to their high total dietary fiber content (Abreu *et al*., 2012), and the content of essential fatty acids such as linoleic acid contained in its seeds (Ariffin *et al*., 2009). These components may contribute to increased satiety and consequently cause a reduction in food intake. After the 12 weeks of treatment, the groups that received pitaya in the feed composition presented greater weight gain when compared to C1 gruop. This counterpoint indicates that the content of sugars and fats of the pitahias contributed to the increase of weight (Song *et al*., 2016). In addition to weight gain and alteration in the lipid profile, a high-fat diet can cause insulin resistance and other consequences such as type 2 diabetes, atherosclerosis, and non-alcoholic liver diseases (Lenquiste *et al*., 2012). Diabetes is one of the consequences of obesity, as it is the cause of many cases of renal failure, blindness and amputations, in addition to raising the risk for cardiovascular and cerebrovascular diseases (Bugianesi *et al*., 2005). At the beginning of the 21st century, as the fifth leading cause of death in the world (5.2% of deaths); attributing this index to the bad habits of life as a sedentary lifestyle and unbalanced eating habits (Roglic *et al*., 2005; DSBD, 2016).

In the present study, although there was no difference in the area below the glycemic curve, as expected, there was a reduction in fasting glycemia in the treatment groups (P2 and P4) when compared to the group that ate a high calorie diet, as presented in previous studies (Ramli *et al*., 2014; Song *et al*., 2016). The development of obesity is responsible for the increase of oxidative stress, capable of deregulating serum levels of total cholesterol, LDL, VLDL, HDL and triglycerides. The pigment present in the bark of white pitahaya, betalain, as well as phenolic compounds and ascorbic acid, inactivate the action of free radicals responsible for metabolic stress (Lenquiste *et al*., 2012; Song *et al*., 2016). In this context, the treatment groups (P2 and P4) obtained higher HDL values compared to C1 and C2.Together with the increase in HDL, triglyceride levels showed a good reduction in the P2 and P4 groups, compared to the C2 group, especially in the P4 group (43.68% reduction).It was found that the pitaya in larger doses would be responsible for having a more expressive effect (Song *et al*., 2016). Non-alcoholic liver disease is present in 57-74% of people classified as obese.And this response is associated with the high consumption of saturated fats, simple carbohydrates and insufficient consumption of fiber, vitamins and minerals(Wang *et al*., 2006; Crispim *et al*., 2016). Oxidative stress resulting from the high intake of saturated fats promotes the development of insulin resistance and hepatic dysfunction. By this aspect, the pitaya may have exerted some effect under the action of the hyperlipidic diet. Although not significant ($p = 0.307$), the groups that ingested lyophilized pitaya had lower percentages of hepatic impairment when compared to the group with a high-fat diet. The group P2 presented 24.3% less steatosis volume density than the C2 group. This result can be correlated with the reduction in triglyceride values in the groups treated with pitaya, in which the response for the P2 group presented a reduction of 20.39%. These findings are in agreement with the results as presented in previous studies in which the pitaya has an antioxidant effect due to the oxidative stress caused by the diet rich in saturated fats.

Conclusion

The white pitaya (*Hylocereus undatus*) lyophilised not able to control of weight gain and glucose tolerance induced by a high-fat diet, but it did reduce food intake.The treatments

containing 2 and 4% of pitaya were able to improve lipid profile, with increase of HDL and reduction of triglycerides. In addition, in the diet containing 2% of pitaya, there was a small difference in the conservation of liver function compared to gruop high-fat diet without tratament. In view of these results, further studies should be carried out to clarify the efficiency of the Pitaya in different concentrations.

REFERENCES

Abreu, W.C., Lopes, C.O., Pinto, K.M., Oliveira, L.A., Carvalho, G.B.M., Barcelo, M.F.P. 2012. Características físico-químicas e atividade antioxidantes total de pitaias vermelha e branca. Rev Inst Adolfo Lutz, 71(4), 656-661.

Aguila, M.B., Pinheiro, A.R., Parente, L.B., Mandarim-de-Lacerda, C.A. 2003. Dietary effect of different high-fat diet on rat liver stereo logy. Liver Int, 23, 363-370.

Alexander, J., Chang, G.Q., Dourmashkin, J.T., Leibowitz, S.F. 2006. Distinct phenotypes of obesity-prone AKR/J, DBA2 and C57BL/6J mice compared to controle strains. Int J Obes, 30, 50-59.

Ariffin, A.A., Bakar, J., Tan, C.P., Rahman, R.A., Karim, R., Loi, C.C. 2009. Essential fatty acids of pitaya (dragon fruit) seed oil. Food Chem, 114, 561-564.

Buettner, R., Parhofer, K.G., Woenckhaus, M., Wrede, C.E., Kunz-Schughart, L.A., Schoölmerich, J., Bollheimer, L.C. 2006. Defining high-fat-diet rat models: metabolic and molecular effects of different fat types. J Mol Endocrinol, 36, 485-501.

Bugianesi, E., Gastaldelli, A., Vanni, E., Gambino, R., Cassader, M., Baldi, S., Ponti, V., Pagano, G., Ferrannini, E., Rizzetto, M. 2005. Insulin resistance in non-diabetic patients with non-alcoholic fatty liver disease: sites and mechanisms. Diabetologia, 48, 634-642.

Catta-Preta, M., Mendonça, L.S., Fraulob-Aquino, J., Aguila, M.B., Mandarim-de-Lacerda, C.A. 2011. A critical analysis of three quantitative methods of assessment of hepatic steatosis inlayer biopsies. Virchow Arch, 459, 477-485.

Crispim, F.G.S., Elias, M.C., Parise, E.R. 2016. Consumo alimentar dos portadores de Doença Hepática Gordurosa Não Alcoólica: comparação entre presença e ausência de Esteatoepatite Não Alcoólica e Síndrome Metabólica. Rev de Nutri, 29(4), 495-505.

Diretrizes brasileiras de obesidade - Associação Brasileira para o Estudo da Obesidade e da Síndrome Metabólica (DBO/ABESO). 2016, São Paulo, 4.ed.

Diretrizes da Sociedade Brasileira de Diabetes (DSBD) (2015-2016) / Adolfo Milech. 2016 Organização José Egidio Paulo de Oliveira, Sérgio Vencio - São Paulo: A.C. Farmacêutica.

Florence, N.T., Benoit, M.Z., Jonas, K., Alexandra, T., Désiré, D.D.P., Pierre, K., Théophile, D. 2014. Antidiabetic and antioxidant effects of Annona muricata (Annonaceae), aqueous extract on streptozotocin-induced diabetic rats. *J Ethnopharmacol,* 151(2), 784–790.

Grundy, S.M. 2015. Adipose tissue and metabolic syndrome: too much, too little or neither. *J Clin. Invest,* 45(11), 1209-1217.

Kowalska, K., Olejnik, A. 2016. Beneficial effects of cranberry in the prevention of obesity and related complications: Metabolic syndrome and diabetes. *J Funct Foods,* 20, 171-181.

Lenquiste, S.A., Batista, A.G., Marineli, R.S., Dragano, N.R.V., Marostica Jr, M.R. 2012. Freeze-dried jabuticaba peel added to high-fat diet increases HDL-cholesterol and improves insulin resistance in obese rats. Food Res Internat, 49, 153-160.

Mello, F.R., Bernardo, C., Dias, C.O., Gonzaga, L., Amante, E.R., Fett, R., Candido, L.M.B. 2015. Antioxidant properties, quantification and stability of betalains from pitaya (Hylocereus undatus) peel. Ciência Rural, 45(45), 323-328.

Nunes, E.N., Sousa, A.S.B., Lucena, C.M., Silva, S.M., Lucena, R.F.P., Alves, C.A.B., Alves, R.E. 2014. Pitaia (Hylocereus sp.): Uma revisão para o Brasil. Gaia Sci, 8, 90-98.

Ramli, N.S., Brown, L., Ismail, P., Rahmat, A. 2014. Effects of red pitaya juice supplementation on cardiovascular and hepatic changes in high-carbohydrate, high-fat diet-induced metabolic syndrome rats. BMC Complementary and Alternative Medicine, 14, 189.

Roglic, G., Unwin, N., Bennett, P.H., Mathers, C., Tuomilehto, J., Nag, S., Connolly, V., King, H. 2005. The burden of mortality attributable to diabetes: realistic estimates for the year 2000. Diabetes Care, 28(9), 2130-2135.

Santos, S.H.S., Fernandes, L.R., Mario, E.G., Ferreira, A.V.M., Pôrto, L.C.J., Alvarez-Leite, J.I., Botion, L.M., Bader, M., Alenina, N., Santos, R.A.S. 2008. Mas deficiency in FVB/N mice produces marked changes in lipid and glycemic metabolism. Diabetes, 57(2), 340-347.

Sato, S.T.A., Ribeiro, S.C.A., Sato, M.K., Souza, J.N.S de. 2014. Caracterização física e físico-química de pitayas vermelhas (Hylocereus costaricensis) produzidas em três municípios paraense. J Bioen and Food Sci, 1, 46-56.

Song, H., Zheng, Z., Wu, J., Lai, J., Chu, Q., Zheng, X. 2016. White Pitaya (Hylocereus undatus) Juice Attenuates Insulin Resistance and Hepatic Steatosis in Diet-Induced Obese Mice. PLoS ONE, 11(2), 1-14.

Swarup, K.R.A., Sattar, M.A., Abdullah, N.A., Abdulla, M.H., Salman, I.M., Rathore, H.A., Johns, E.J. 2010. Effect of dragon fruit extract on oxidative stress and aortic stiffness in streptozotocin-induced diabetes in rats. Pharmac res, 2(1), 31-35.

Wang, D., Wei, Y., Pagliassotti, M.J. 2006. Saturated fatty acids promote endoplasmic reticulum stress and liver injury in rats with hepatic steatosis. *Endocrinol,* 147(2), 943-951.

West, D.B., Boozer, C.N., Moody, D.L., Atkinson, R.L. 1992. Dietary obesity in nine inbred mouse strains. AJP – Regu Phyisiol, 262, 1025-1032.

White, P.A.S., Cercato, L.M., Araújo, J.M.D., Souza, L.A., Soares, A.F., Barbosa, A.P.O., Neto, J.M.R., Marçal, A.C., Machado, U.F., Camargo, E.A., Santos, M.R.V., Brito, L.C. 2013. Modelo de obesidade induzida por dieta hiperlipídica e associada à resistencia à ação da insulina e intolerância à glicose. Arq Bras Endocrine Metab, 57(5), 339-345.

Winzell, M.S., Ahrén, B. 2004. *The High-Fat Diet–Fed Mouse. Diabetes, 53(3), 215-219.*

World Health Organization (WHO). Obesity and overweight. 2016. Acessed: 2017 Fev 13; Available: http://www.who.int/mediacentre/factsheets/fs311/en/.

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