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Caryocar brasiliense fruit intake ameliorates hepatic fat deposition and improves intestinal structure of rats

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***Caryocar brasiliense* (*pequi*) is an exotic fruit, high in monounsaturated fat acids (MUFA) and bioactive compounds, which have beneficial effects on cardiometabolic risk factors. However, this fruit is poorly studied in this context. In this study, the effects of *pequi* pulp intake on cardiometabolic risk factors of rats were evaluated. Therefore, 16 male weaned rats were divided into two groups: Control group and *Pequi* group. Control group was feed a standard diet and *pequi* group, the same diet added *pequi* pulp (3.26 g.100^{-1}) for 15 weeks. At the end, plasma lipids, glucose, insulin, Homeostasis Model Assessment of Insulin Resistance index (HOMA-IR), blood pressure, heart rate, hepatic and fecal lipids and intestinal histomorphometric parameters were accessed. Liver and heart samples were harvested for redox status assays. There were no differences between experimental groups for blood pressure, heart rate, glucose, insulin, HOMA-IR, triglycerides, cholesterol, HDL-cholesterol, and liver and heart redox status (p<0.05). *Pequi* group had lowered lipid hepatic deposition and increased fecal output (p<0.05), increased intestinal villus height and crypt depth. Thus, *pequi* pulp intake minimized liver fat deposition by increasing its intestinal output and improved intestinal structure of rats, which can contribute for reducing cardiometabolic risk factors. MUFA, carotenoids and fibres can be associated, at last in part, with these effects.**

Key words: *Caryocar brasiliense*, *pequi*, cardiometabolic risk, lipid metabolism, redox status.

INTRODUCTION

Non communicable diseases (NCD), such as type 2 diabetes and cardiovascular diseases, are major causes of mortality worldwide (up to 38 million by year), and are responsible for 80% of deaths occurring in developing countries (WHO, 2014). Increasing the intake of fruits and vegetables are one of the main recommendations for reducing NCD risk. From this perspective, consumer interest in foods with functional properties is increasing

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(Bumgarner et al., 2012; Jackix et al., 2013; Schreckinger et al., 2010).

In this context, *pequi* (*Caryocar brasiliense*) is an exotic fruit from a Brazilian savannah-like biome. Its pulp is sweet, yellowish and has a high energy density and lipid content (~33%), being monounsaturated fatty acids (MUFAs) (especially oleic, ~54%) its major constituent (Lima et al., 2007). It also has substantial amounts of fibres (18%) (Teixeira et al., 2013) and carotenoids (~42 $mg.100^{-1}g$), especially violaxanthin, lutein, zeaxanthin, β-cryptoxanthin, neoxanthin and β-carotene (Azevedo-Meleiro and Rodriguez-Amaya, 2004).

From *pequi* pulp chemical constituents, MUFAs have been associated with improvements in lipid profile, reduced platelet aggregation, favourable modulation of blood pressure, insulin sensitivity and glycemic control (Hammad et al., 2016). Fibres are related to the regulation of intestinal function, control of body weight and lipid metabolism by decreasing the absorption and increasing excretion of cholesterol and triglycerides, and they have indirect effects on the blood pressure and serum glucose control (Lattimer and Haub, 2010). Carotenoids are powerful antioxidants, and some, such as β-carotene, have vitamin A activity. They are associated with reduced risk for cancer and cardio-vascular diseases, and protection from cell oxidative damage (Gülçin, 2012; Ried and Fakler, 2011).

Thus, the composition of nutrients and bioactive compounds of *pequi* pulp suggests that it could be a food supplement and it can exert effects on metabolism, cardiovascular function and cell redox status as a functional food. However, this fruit has been poorly studied. To the author's knowledge, there are only some research showing healing (Quirino et al., 2009; Bezerra et al., 2015), chemopreventive (Palmeira et al., 2016; Colombo et al, 2015) and anti-inflammatory (Miranda-Vilela et al., 2009) properties of *pequi* oil. Studies regarding functional properties from *pequi* pulp intake are scarce (Teixeira et al., 2013).

Therefore, the aim of this study was to evaluate the effects of *pequi* pulp intake on cardiometabolic risk markers of rats. The *in vitro* antioxidant activity and the chemical composition of *pequi* pulp, were determined previously because some compounds could be related to its potential health benefits.

MATERIALS AND METHODS

Pequi pulp samples

Ripe *pequi* fruits were acquired from the local market of Diamantina city, Minas Gerais State, Brazil. They were washed with tap water and subsequently with distilled water. After drying at room temperature, each fruit was cut in half and the pulp was separated from the almond manually. Afterwards, the pulps were placed on trays and dried at 65°C for 48 h (Teixeira et al., 2013). After drying, the material was grounded, wrapped in a plastic bag, labeled and stored at -18±2°C until the analysis.

Chemical composition and *in vitro* antioxidant activity of *pequi* pulp

Protein, total lipids, dietary fibres (enzymatic–gravimetric method) and total carotenoids were determined as described by The Association of Official Analytical Chemists - AOAC methods (AOAC, 1995). Carbohydrates were calculated by difference, and the total energy value (TEV) was estimated using the Atwater factors (Buchholz & Schoeller 2004). Fatty acids were analyzed by gas chromatography (CGC Agilent 6850 Series GC System) according to The American Oil Chemist´s Society – AOCS (AOCS, 2009).

The *in vitro pequi* pulp antioxidant activity was performed in both 6:4 ethanol: water and 1:1 methanol: acetone extracts. Briefly, dehydrated *pequi* pulp samples were extracted with 40 mL of 1:1 methanol/water solution for 1 h, at room temperature. Afterwards, the mixture was centrifuged (Biosystems, Modelo 80-2B, Curitiba-PR) at 3.000 rpm for 15 min. The supernatant was harvested and the step was repeated, using a 7:3 acetone/water solution (Larrauri et al., 1997). After the solvents evaporation, the mixtures were diluted in 6:4 ethanol : water and 1:1 methanol : acetone solutions. The 2,2-diphenyl-1-picrylhydrazyl free radical scavenging (DPPH) and ferric reducing antioxidant power (FRAP) methods were used according to Rufino et al. (2010).

Rat study

Experimental protocols were performed in accordance with the EU Directive 2010/63/EU for animal experiments. They were approved by the Ethics Committee on Animal Use/Federal University of Vales do Jequitinhonha e Mucuri, Diamantina, MG, Brazil (Protocol 010/2012).

Sixteen male Wistar rats, four weeks aged, were housed in individual stainless steel cages and maintained in a room with controlled temperature (22±2°C) and a 12 h light/dark cycle, with free access to food and water during the experimental period.

A commercial chow (RhosterLab®) was used as a standard diet, and its energy density was 3.28 $kcal.g^{-1}$ (13.77 $kJ\ g^{-1}$). Based on the lipid composition of the *pequi* pulp, the standard diet was added, *pequi* pulp at 3.26.100 g^{-1}, which resulted in a 50% increase in total lipid content, so its energy density turned into 3.39 $kcal\ g^{-1}$ (or 14.2 $kJ.g^{-1}$, a 3.35% increase). The *pequi* pulp supplementation was also added, 1.19 g.100 g^{-1} of oleic acid; 0.63 g.100 g^{-1} of fibres and 0.14 $mg.100\ g^{-1}$ of carotenoids to the standard diet.

All 16 animals were randomly assigned to two treatment groups (n=8): Control group - animals fed the standard diet and *Pequi* pulp group - animals fed the standard diet added *pequi* pulp. The study lasted for 15 weeks. During this period, body weight and food intake were monitored for energy efficiency ratio (EER = weight gain/kcal) and feed efficiency ratio (FER = weight gain/g of diet) calculations. Faeces were collected in the last 72 h of the experiment, dried and kept at -80°C until analysis. The body weight and length (nose–anus length) were measured in all anaesthetized rats (quetamin + xilazin/50 mg/kg + 10 mg/kg) in the previous day to the euthanasia for the Lee index calculation (weight body.$g^{0.33}$ /nose–anus length).

On the last day, overnight fasted animals were anesthetized (quetamin + xilazin/50 mg/kg+10 mg/kg), euthanized by decapitation for blood and tissues (adipose tissues, liver, heart, duodenum) harvesting. Retroperitoneal and epididymal fat pads were used for adiposity index ([retroperitoneal + epididymal pads/body weight – (retroperitoneal + epididymal pads)]*100)) calculation. Blood was centrifuged in heparinized tubes to obtain plasma, and aliquots were transferred to Eppendorf tubes and kept at -80°C until analysis. Liver and heart tissues were processed for redox status analysis. Duodenum fragments of 5 cm were harvested and stored in a 10% formaldehyde solution for complementary histological analyses.

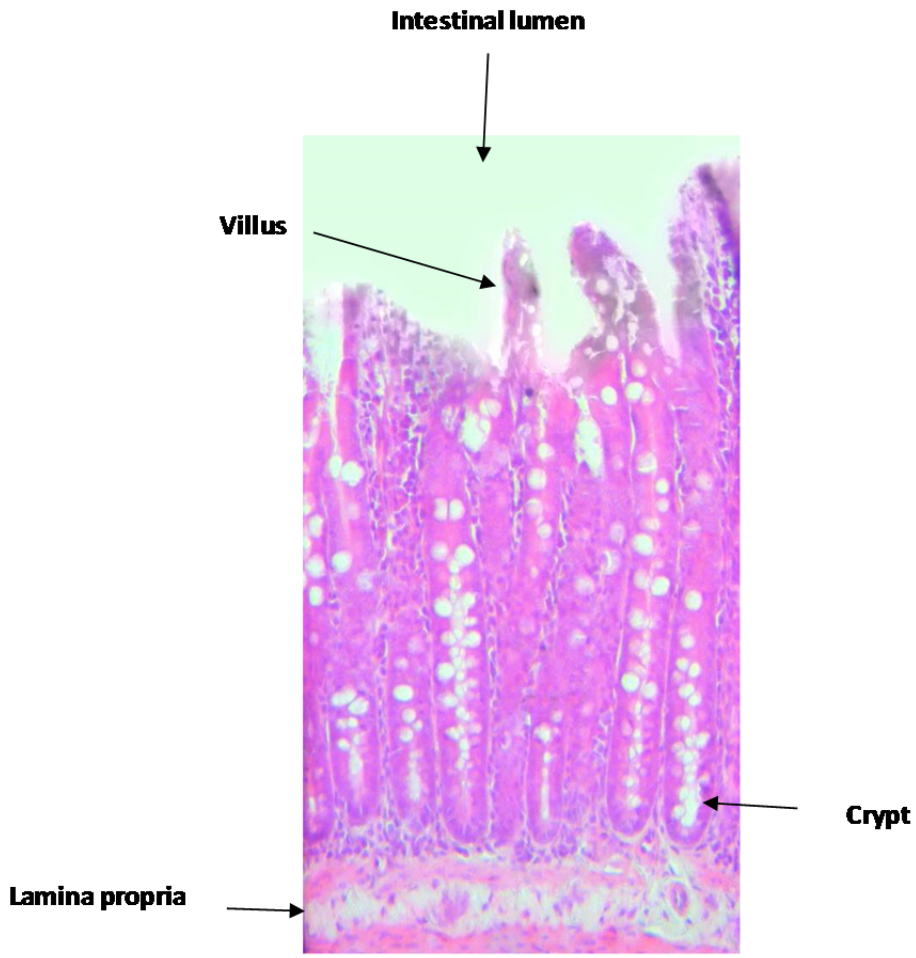

Figure 1. Morphometric analysis of duodenum: villus height and crypt depth. 5 µm cross-section, 100x magnification.

Cardiometabolic risk factors

Tail blood pressure (BP) and heart rate (HR) were measured in the last week of the experimental protocol by the non-invasive tail plethysmography method. The animals were heated to cause vasodilation of the caudal artery. The pulses were recorded by system (AD Instruments Ltd, UK). The BP and HR values were used for the double product calculation (BP x HR). The heart weight and body weight were used for cardiac hypertrophy evaluation, by heart weight/body weight calculation.

Fasted plasma glucose levels (GLU) were measured by means a commercial kit according the procedures recommended by the manufacturer and using a semi-automatic biochemical analyzer (PIOWAY-3000). Fasted plasma insulin (INS) was determined using a commercially available Enzyme-Linked Immunosorbent Assay kit (Linco Research Inc., St. Louis, MO, USA) and a micro-plate reader (Spectra MAX 190, Molecular Devices, USA). Insulin resistance was accessed by the homeostasis model assessment of insulin resistance (HOMA-IR index), from fasted glucose and insulin levels according to Matthews et al. (1985).

Total plasma cholesterol (CHOL), high-density lipoprotein cholesterol (HDL-C) and triglyceride (TG) levels were determined using a semi-automatic biochemical analyzer (PIOWAY-3000) and commercial kits according the procedures recommended by the manufacturer. Liver and faeces samples were oven-dried (60°C ± 2°C for 72 h) after harvesting, grounded, and their lipids were extracted according to Folch et al. (1957). CHOL and TG levels were determined using commercial kits according the procedures recommended by the manufacturer and using a semi-automatic biochemical analyzer (PIOWAY-3000).

Considering the possible influences of some chemicals from *pequi* pulp in the intestinal morphology, which could affect nutrient digestion and absorption, we also preceded histomorphometric assays. For that, fragments of duodenum were removed and fixed in a 4% buffered formaldehyde solution. After dehydration and fixation in paraffin, two 5 µm cross-sections which were stained with haematoxylin/eosin was performed. Results were obtained by means of a digital camera coupled to a microscope. All images were analysed using the Axion Vision software. The villus height (VH) and the crypt depth (CD) were expressed as the arithmetic mean determined from 20 measurements of each sample. The villus height/crypt depth ratio (VH/CD) was also calculated. The villi density per optical field (920764.14 µm²) was taken from five photos from each animal. All measurements were made in µm, at 100x magnification (Figure 1).

For the redox status analysis, liver and heart samples were homogenized in phosphate-buffered saline (PBS) (T 20 basic ULTRA-TURRAX; IKA Labortechnik, China), pH 7.2, and

Table 1. Nutritional composition (g.100 g^{-1}), energy density (kcal.g^{-1}) and total carotenoids (mg.100 g^{-1}) of boiled and dehydrated *pequi* pulp (*Caryocar brasiliense*).

Component	Amount*
Lipids	66.76±1.16
Proteins	4.76±0.38
Fibres	19.19±0.09
Carbohydrates	1.41±3.27
Energy density**	6.25±0.10
Carotenoids	43.3±0.03

*Values expressed in mean ±standard deviation. **26.27± 0.42 kj.g^{-1}.

Table 2. Fatty acid profile from boiled and dehydrated *pequi* pulp oil (*Caryocar brasiliense*).

Nomenclature	g.100g^{-1}
Lauric (C12:0)	0.04±0.01
Myristic (C14:0)	0.11±0.01
Palmitic (C16:0)	40.14± 0.01
Stearic (C18:0)	1.50±0.00
Arachidonic (C20:0)	0.16±0.00
Behenic (C22:0)	0.05±0,01
Lignoceric (C24:0)	0.08±0,00
Total of saturated	42.13±0.01
Palmitoleic (C16:1)	0.89±0.00
Oleic (C18:1)	54.76±0.01
Linoleic (C18:2)	1.53±0.01
α-Linolenic (C18:3)	0.34±0.00
Eicosenoic (C20:1)	0.26±0.00
Total of unsaturated	57.87±0.01

*Values expressed in mean ±standard deviation.

centrifuged for 10 min at 10,000x and 4°C (Jouan BR4i, Thermo Fischer Scientific, USA). The supernatant was harvested and used for the protein determination (Bradford, 1976) and the biochemical assays described below. The total antioxidant capacity was measured using the ferric reducing antioxidant power (FRAP) assay, according to the method of Benzie and Strain (1996). The formation of thiobarbituric acid-reactive substances (TBARS) during a hot acid reaction was used as an index of lipid peroxidation, according to Ohkawa et al. (1979).

Statistical analysis

Results from chemical composition and the *in vitro* antioxidant activity assays were expressed in mean ± standard deviation. Results from rat study were expressed in mean ± standard error. The experiment was performed in a completely randomized design with two treatments (experimental groups) and eight repetitions. Data were analyzed by one way ANOVA at $p<0.05$, using the Statistica 10.0 software. Figures were drawn by means of the SigmaPlot 11.0 software.

Table 3. Antioxidant activity of *pequi* pulp (*Caryocar brasiliense*) extracts by different methods (umol TE/g).

Extract	Method	
	DPPH	FRAP
Ethanol/Water 6:4	0.076±0.001	0.445±0.063
Methanol/Acetone 1:1	0.569*±0.086	1.256*±0.128

*$p<0.05$ by *t-test*. Ethanol/water 6:4 vs Methanol/Acetone 1:1. Values are expressed as mean ± standard deviation. DPPH=2,2-diphenyl-1-picrylhydrazyl free radical scavenging; FRAP = ferric reducing antioxidant power.

RESULTS

Chemical composition of pequi pulp and *in vitro* antioxidant assays

Pequi pulp had expressive amounts of total lipids (Table 1). The main fatty acids from *pequi* lipids were oleic followed by palmitic (Table 2). *Pequi* pulp also had high amounts of fibres, being majorly insoluble, and total carotenoids (Table 1). For the *in vitro* antioxidant activity assays, *pequi* pulp methanol/acetone extract had higher antioxidant capacity, by both FRAP and DPPH methods ($p<0.05$) (Table 3).

Rat study

Similar body weights were found at the beginning and end of the experiment. The food and caloric intake, FER, EER, Lee index and adiposity index did not differ between groups (Table 4). There were no differences for BP, HR, double product, hypertrophy index, plasma markers of glucose and lipids metabolism markers (glucose, insulin, HOMA-IR, triglycerides, cholesterol and HDL-C) (Table 5).

Regarding hepatic and faecal lipids, *pequi* pulp animals had lower hepatic levels of CHOL and TAG when compared with controls ($p<0.05$) (Figure 2A and B). Faecal CHOL did not differ between groups. *Pequi* group had higher faecal TAG levels when compared with C ($p<0.05$) (Figure 2D).

From the duodenum histomorphometric assays, an increase in the villus height (VH), Crypt depth (DC) and number of villous (NV) per optical field for *pequi* pulp group ($p<0.05$) was observed. However, differences were not observed between treatments for VH/DC ratio (Table 6). There were no differences between groups for both liver and heart lipid peroxidation levels and antioxidant capacity (Figure 3). However, for hearts, *pequi* group had a 23% increase in the antioxidant capacity and a 28% decrease in the peroxidation levels as compared to the control.

Table 4. General characteristics of experimental groups.

Variables	Control	*Pequi* pulp
Body weight (g)	286.37 ± 52.26	261.52 ± 25.69
Food Intake (g)	2209.54 ± 255.38	2148.23 ± 243.76
Caloric Intake (Kcal)	7247.29 ± 837.64	7475.84 ± 848.28
FER(g/g)	0.10 ± 0.02	0.10 ± 0.01
EER (g/kcal)	3.18 ± 0.57	2.82 ± 0.20
Lee Index	0.10 ± 0.02	0.10 ± 0.01
Adiposity Index	2.87 ± 0.63	3.01 ± 0.51

Values are expressed as mean ± standard error. FER: feed efficiency ratio; EER energy efficiency ratio.

Table 5. Cardiometabolic risk factors of the experimental groups.

Variables	Control	*Pequi* pulp
BP (mmHg)	152.38 ± 20.38	146.23 ± 26.22
HR (bpm)	392.37 ± 58.17	397.43 ± 33.72
Double product	60451.59 ± 16078.48	58502.86 ± 14663.45
Cardiac hypertrophy (g/g)	0.46 ± 0.06	0.51 ± 0.04
Glucose (mg/dL)	123.75 ± 20.68	116.98 ± 16.02
Insulin (ng/mL)	0.89 ± 0.28	1.17 ± 0.34
HOMA-IR	7.91 ±2.95	9.64 ± 3.59
Triglycerides (mg/dL)	32.56 ± 4.95	34.57 ± 10.46
Cholesterol (mg/dL)	58.11 ±7.91	62.19 ± 10.36
HDL-cholesterol (mg/dL)	22.90 ± 5.50	22.13 ± 4.08

Values are expressed as mean ± standard error. BP: blood pressure; HR: heart rate.

DISCUSSION

Pequi is an exotic fruit with a heavy potential to be a functional food, since it has a peculiar nutritional composition and is high in several bioactive compounds. In this study, the amount of lipids found in the *pequi* pulp samples was in accordance with previous data from the laboratory (Teixeira et al., 2013) and higher than that found by Cardoso et al. (2013) and Lima et al. (2007). Also, *pequi* pulp has a paradoxal composition in fatty acids, since oleic acid, a MUFA, is its major constituent and related to cardiometabolic risk reduction. Otherwise its second higher constituent is palmitic acid, a saturated fatty acid with several cytotoxic effects described (Akazawa et al., 2010; Eitel et al., 2002).

Its amount of fibres and carotenoids were comparable to other commonly consumed foods that are high in those compounds (Rodriguez-Amaya et al., 2008). Otherwise, carotenoids were lower than other samples analysed elsewhere (Cardoso et al., 2013; Lima et al., 2007; Teixeira et al., 2013). These differences may be due the different processing forms of *pequi* pulp samples in those studies. In these samples, boiling and dehydrating may have lowered carotenoid content and also, increased other nutrient concentrations, such as lipids and fibres. The higher antioxidant activity of methanol/acetone *pequi* pulp extract indicated that lipophilic compounds account significantly for the pulp antioxidant power.

Based on these findings, it is clear that this fruit has expressive amounts of certain nutrients and bioactive compounds that have been associated with protection in many biochemical processes that underly the development of cardiometabolic diseases. Therefore, study of some biological effects of these compounds together in a single food which is still poorly studied, is proposed.

Adding *pequi* pulp did not influence body weight gain, food intake, glucose and plasma lipids. In addition, this increase also did not affect BP and HR and it did not cause cardiac overload, since no changes in the double product and cardiac hypertrophy index was observed. There was 50% increase in total lipid content of standard diet by adding *pequi* pulp. Although, there was increase in calories and lipids, the diet did not turned into a high fat, which could cause metabolic disturbance. According to Buettner et al. (2006) and Hariri and Thibault (2010), to have high fat, a diet must have at least 30% of its energy from lipids. The *pequi* group diet had 18.51%.

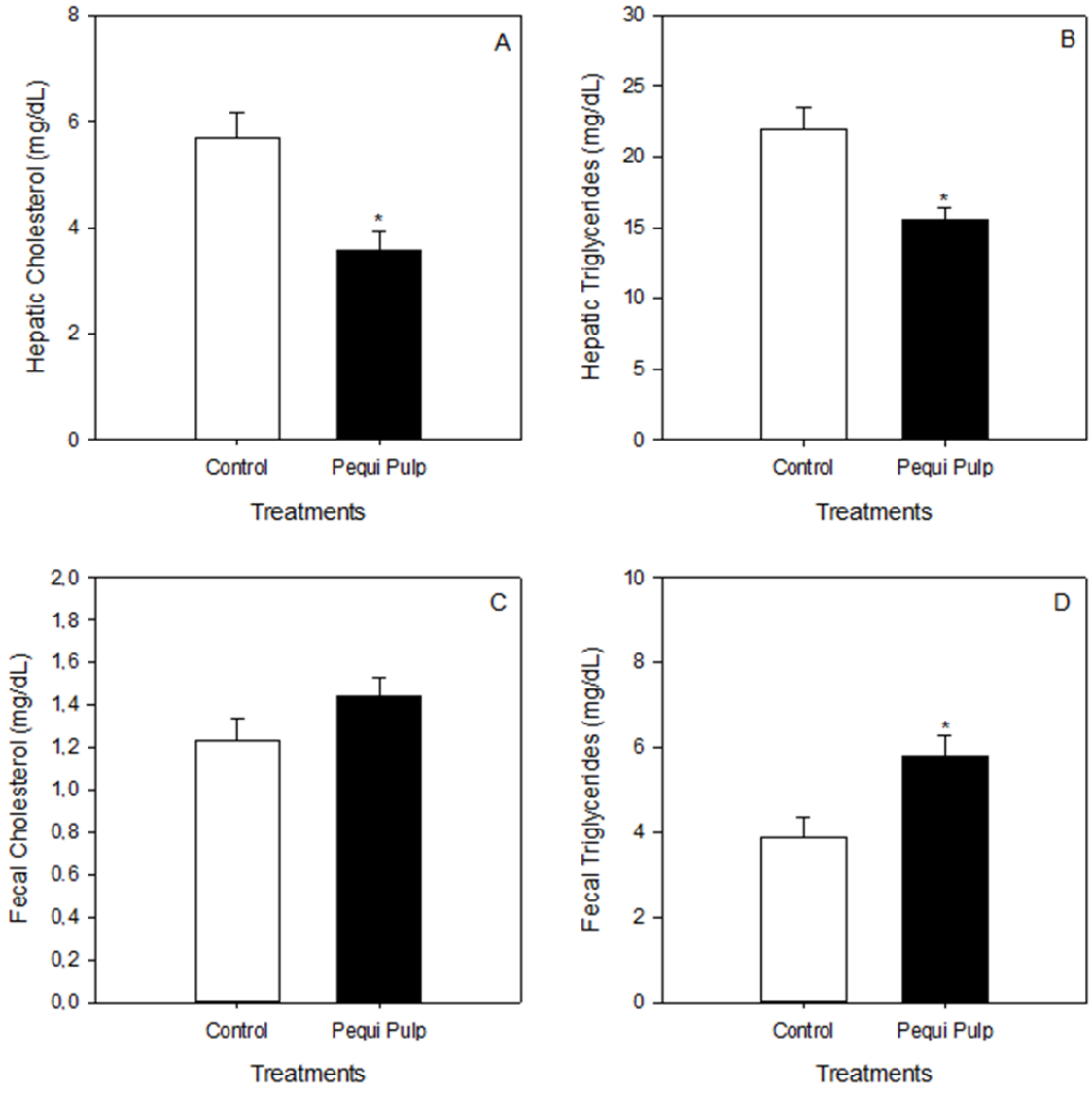

Figure 2. Hepatic and faecal cholesterol and triglycerides levels of experimental groups. Values are expressed as mean ± standard error. * represent statistically significant difference ($p<0.05$) by one way ANOVA.

Table 6. Morphometry of the duodenum: villus height (VH), crypt depth (CD), VH/CD ratio (µm) and villous number (VN) (units per optical field) of the experimental groups.

Variables	Control	*Pequi* pulp
HV (µm)	398.11 ± 42.02	480.14 ± 65.79*
DC (µm)	247.01 ± 9.93	296.63 ± 38.34*
HV/DC Ratio	1.61 ± 0.11	1.62 ± 0.05
VN	19.03 ± 1.57	21.99 ± 2.07*

Values are expressed as mean ± standard error. * represent statistically significant difference ($p<0.05$) by one way ANOVA.

Although, the main chemical constituents from *pequi* pulp (mainly MUFAs and fibre) are related to CHOL, TG and LDL-lowering effects (Ried and Fakler, 2011) as well as having antioxidant properties (carotenoids) (Gülçin,

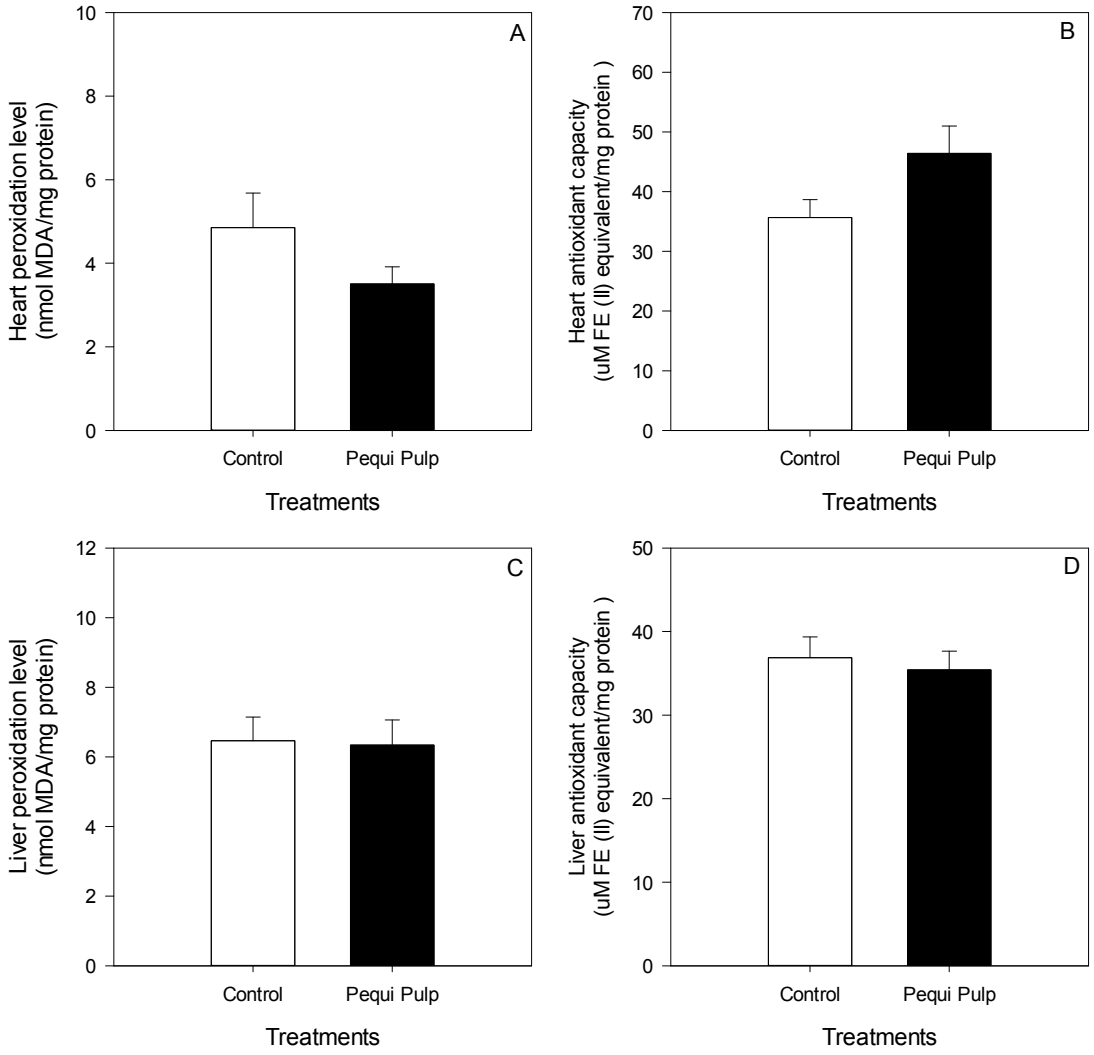

Figure 3. Lipid peroxidation levels and total antioxidant capacity of hearts and livers of experimental groups. Values are expressed as mean ± standard error.

2012), we did not observe these effects on plasma lipids. Otherwise, there were pronounced effects from *pequi* pulp supplementation in the hepatic and faecal lipids. The lower hepatic levels of TRI and CHOL and higher TRI faecal output in the *pequi* group may be associated with the higher *pequi* pulp fibre content.

Fibres, especially soluble, can increase the lumen viscosity, bind to cholesterol, triglycerides, bile acids and other lipids, impairing their digestion/absorption and increasing their faecal output (Lattimer and Haub, 2010). They can also be fermented by microflora and generate products, as acetate, propionate and butyrate, which affect the endogenous synthesis of theses lipids (Ngoc et al., 2012). Insoluble fibres, in turn, regulate intestinal transit-time, contributing to the lower absorption of those nutrients (Lattimer and Haub, 2010). Furthermore, insoluble fibres are related to a higher expression of hepatic genes that increase fatty acid oxidation (Isken et al., 2010).

Therefore, the *pequi* pulp could have modulated the function of the gastrointestinal tract to increase the lipids excretion. Histological data corroborated these findings. *Pequi* group showed higher villous height and crypt depth, implying this food exerted a positive effect in the mucosa integrity. Conversely, the DC increase in this group indicated a high rate of cell differentiation in crypts. In addition, the increase in VH indicated cell migration and renovation to the villus (Rosa et al., 2010).

Some compounds of *pequi* pulp can be related to that. Fibres were associated with VH increase (Ashraf et al., 2013) and oleic acid was associated with a better gut development and a DC increase (Rosa et al., 2010). In addition, carotenoids, as vitamin A precursors can act on intestinal cell growth and differentiation (Allen et al.,

2002). As antioxidants, they can decrease damage caused by oxidative agents and therefore, contribute to cell preservation (Turan et al., 2009). Then, it can be inferred that *pequi* pulp may have increased the duodenal absorption surface area and cell renovation, helping the maintenance of the mucosa integrity.

Regarding redox status, statistical differences were not observed between groups for livers and hearts. According to several authors (Feillet-Coudray et al., 2009; Sour et al., 2015), changes on redox status parameters are easily detectable when dietary lipid and caloric overload occurs, or when there are some physiological disturbance, such as inflammation, obesity and dyslipidaemia. Adding *pequi* pulp did not increase significantly lipid content of the diet. In addition, the lipid lower liver accumulation and increased faecal output upon *pequi* pulp intake may also be related to these results, since it can have contributed to a less generation of reactive oxygen and nitrogen species with subsequent lower peroxidation of membrane lipids.

However, in the heart, it is important to consider that *pequi* pulp led to a trend in increasing antioxidant capacity and decreasing lipid peroxidation levels. It seems that the heart was the more sensitive organ upon *pequi* pulp intake. Carotenoids are natural antioxidants, have lipophilic characteristic and may be incorporated into mitochondrial membranes, which are the main site for free radical production during the electron flow (Vega et al., 2009). Furthermore, this *in vitro* assay showed that methanol and ethanol extracts of *pequi* pulp had a high antioxidant capacity.

Conclusion

Taken together, the results indicate that *pequi* pulp intake minimized liver fat deposition by increasing its faecal output and improved intestinal structure, which could account for reduction of cardiometabolic risk in rats. Fibres, MUFA and carotenoids from this fruit may be responsible, at last in part, for these effects.

Conflicts of interests

The authors have not declared any conflict of interest.

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