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# The Partial Replacement of Lard by *Caryocar brasiliense* Oil in a Western Diet improves Cardiovascular Risk Factors in Rats

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## Abstract

**Background:** Currently, several studies have shown that consuming plant foods high in monounsaturated fatty acids (MUFA) and antioxidants is associated with lower risk for cardiovascular disease (CVD). In this perspective, *Caryocar brasiliense* (*pequi*) oil has a potential, since MUFA represent approximately 60% of its fatty acid content, and it is high in several antioxidant carotenoids.

**Objective:** To evaluate the effects of a partial replacement of lard (high in saturated fatty acid - SFA) by *pequi* oil (high in MUFA and carotenoids), in a Western diet model, on cardiovascular risk factors and *ex vivo* cardiac function of rats.

**Methods:** Animals were assigned into three groups (n = 12): CTRL - AIN93G control diet; HFS – high in SFA (lard) and sucrose diet and HFS-PO – HFS diet with 27% of lard replaced by *pequi* oil. At the end, feces, retroperitoneal and epididymal fat pads, blood and livers were harvested for cardiovascular risk factor assays (systolic blood pressure; heart rate; Lee index; Adiposity index; plasma, hepatic and fecal lipids; plasma glucose). Hearts were used for the *ex vivo* cardiac function.

**Results:** Body weight and Lee index from HFS-PO and HFS animals were equally higher than CTRL ($p<0.05$). Otherwise, HFS-PO diet reduced Adiposity index compared to HFS ($p<0.05$), which was reinforced by a smaller epididymal adipocyte diameter ($p<0.05$) for this group. There was less hepatic triglyceride accumulation for HFS-PO and this diet, improved the *ex vivo* heart contractility and relaxation indexes compared to HFS. There were no differences among other risk factors evaluated, being all equally worsened by HFS and HFS-PO compared to CTRL.

**Conclusions:** The partial replacement of lard by *pequi* oil in a western diet reduced visceral adiposity, hepatic triglyceride deposition and ameliorated cardiac function of rats. Although it did not influence other markers, this can contribute for slowing up cardiovascular disturbances associated to the western diet pattern.

**Keywords:** *Caryocar brasiliense*, *Pequi* oil, Monounsaturated fatty acids, Carotenoids, Adiposity, Cardiac function, Western diet, Cardiovascular disease.

## Introduction

Cardiometabolic disorders include hypertension, insulin resistance, type 2 diabetes, dyslipidemia, hepatic steatosis, and excess body fat, which are individually and collectively, risk factors for cardiovascular disease (CVD) [1]. These diseases are the lead cause of morbidity and mortality, affecting millions of people in developed and developing countries [2]. Although factors such as genetic determination, stress, physical inactivity, and smoking are all known to contribute to CVD, unhealthy dietary patterns is one of the most relevant, such as the "western diet" pattern [3].

The western diet is characterized by higher intakes of red and processed meat, dairy products, processed and artificially sweetened foods, and salt, with minimal intake of fruits, vegetables, fish, legumes, and whole grains. This pattern means a high intake of Trans and saturated fatty acids (SFA), refined

carbohydrates and sodium along with a very low intake of fibers [3-5]. Because of that, western diet can increase plasma triglycerides, VLDL-cholesterol hepatic synthesis as well as reduce HDL-cholesterol. Besides, frequent intake of high glycemic meals can lead to insulin resistance [6].

Thereby, although high intake of fat has been directly associated to CVD risk, recently, attention has been also addressed to the quality of fat, especially amount and type of fatty acids [7,8]. Indeed, in foods, some vegetable oils have instigated interest because their content of MUFA, and, or, PUFA, which have been associated to health benefits [9].

It has been shown that MUFA-rich diets are associated to lower cardiovascular risk [10,11] by favorably improving blood lipids [12], reducing blood pressure [13], and modulating insulin sensitivity and glycemic control [14]. Then, because of the potential benefices from the intake of foods high in unsaturated fatty acids, it is growing the scientific interest in identifying the fatty acid profile of many foods, especially plant foods. Olive, canola e avocado oils, as well as, peanut butter have been highlighted because they are monounsaturated fatty acid-rich.

Although fatty acids, plant foods also have, overall, many bioactive compounds considered cardiovascular protectors. Several antioxidants are being associated to beneficial effects, such as carotenoids [15], which protect against oxidative damage in many tissues and can improve cardiac function [16,17] in animal models.

In this context, *pequi* oil is a possible aid for increasing MUFA and antioxidant content of diets and, in this way, contributing for reducing CVD risk. This oil is extracted from *Caryocar brasiliense* fruit and its major fatty acid is oleic (57%), the main MUFA in the diet [18]. In addition, it has a substantial amount of carotenoids (32 mg.100$^{-1}$g) [18], especially violaxanthin, lutein, zeaxanthin, β-cryptoxanthin, neoxanthin and β-carotene [19,20].

Thus, *pequi* oil could favorably modulate cardiovascular risk factors. Previously in our lab, we observed that *pequi* oil improved cardiac function by increasing Serca2a/PLB ratio and reduced hepatic triglycerides, but it did not promote significant changes in systemic cardiovascular risk factors in healthy rats [18]. However, we thought that systemic cardiovascular risk factors could not have been affected clearly by *pequi* oil, since our model was healthy animals.

Therefore, the aim of this study was to investigate the effects of a long-term intake of *pequi* oil in cardiovascular risk factors and in the *ex vivo* cardiac function of rats feed a western diet (high in SFA and refined carbohydrates), which had its content of lard partially replaced by *pequi* oil.

## Methods

### Rat study

Experimental protocol was performed in accordance with the principles and guidelines adopted by the Brazilian Council of Animal Experimentation Control (CONCEA). It was approved by the Ethics Committee on Animal Use/Federal University of Vales do Jequitinhonha e Mucuri, Diamantina, MG, Brazil (Protocol 03/2013)

Thirty six male *Wistar* rats, 25 days old, were housed in individual stainless steel cages and kept in a room at 22 ± 2 °C and at a 12 h light/dark cycle, with free access to food and water for 6 days previously and during all experimental period (12 weeks). In the first day of the experiment, all animals were randomly assigned into three treatments (n=12): CTRL – control, fed AIN93G diet [21]; HFS – fed a high fat sucrose diet (western diet) and HFS-PO – fed HFS with 27% of lard replaced by *pequi* oil. The composition of experimental diets is presented in Table 1.

**Table 1:** Composition of experimental diets (g.1000$^{-1)}$

| Ingredients | Experimental diets | | |
|---|---|---|---|
| | CTRL | HFS | HFS-PO |
| Protein (casein) | 200.0 | 200.0 | 200.0 |
| Cornstarch | 397.5 | 48.5 | 48.5 |
| Dextrose | 132.0 | 100.0 | 100.0 |
| Sucrose | 100.0 | 341.0 | 341.0 |
| Cellulose (fiber) | 50.0 | 50.0 | 50.0 |
| Soybean oil | 70.0 | 10.0 | 10,0 |
| Mineral Mix | 35.0 | 35,0 | 35.0 |
| Vitamin Mix | 10.0 | 10.0 | 10.0 |
| L-cystine | 3.0 | 3.0 | 3.0 |
| Choline bitartrate | 2.5 | 2.5 | 2.5 |
| Lard | -- | 200.0 | 146.0 |
| *Pequi*oil | -- | -- | 54.0 |

CTRL= AIN93G [21]; HFS = High fat and sucrose; HFS-PO = HFS with 27% of lard replaced by *pequi* oil

CTRL diet had 394.8 kcal.100$^{-1}$, being 63.7% from carbohydrates, 15.9% from lipids and 20.4% from protein. Both diets HFS and HFS-PO had 464.8 kcal.100$^{-1}$, being 42.1% from carbohydrates, 40.7% from lipids and 17.2% from protein. *Pequi* oil was purchased from local market.

During the experiment, body weight and food intake were monitored for Feed Efficiency (FER(g/g) = body gain/food intake) and Energy Efficiency (EER (g/Kcal) = body gain/energy intake) ratios [22]. Feces were collected in the last 72 hours and kept at -80 °C for lipid analysis.

In the last day, overnight fasted animals were anesthetized (quetamin + xilazin/ 50 mg/kg + 10 mg/kg), and their nose-anus length were measured for Lee Index (LI) calculation (LI = [3√body wheight (g) ÷ nose = anus lenght(cm)] ×10) [22]. After that, all animals were euthanized by decapitation for blood and tissue harvesting. All retroperitoneal and epididymal fat pads were removed and weighted in an analytical scale (Shimadzu AX

200) for the Adiposity Index calculation (AdI% = (epididymal pad +retroperitoneal pad)/body weight – (Σepididimal pad + retroperitoneal pad) *100) [23]. Blood was centrifuged in heparinized tubes to obtain plasma, which were aliquoted in eppendorf tubes and kept at -80 °C until analysis.

**Cardiovascular risk factors**

Systolic blood pressure (BP), as well as heart rate (HR), was measured at the last week prior to the end of the experiment by the tail-cuff plethysmography method (MLT1020PPG IR Plethysmograph, PowerLab).

Fasted plasma glucose levels (GLU) were measured by a commercial kit, according the procedures recommended by the manufacturer and using a semi-automatic biochemical analyzer (PIOWAY-3000). Total plasma cholesterol (CHOL), high-density lipoprotein cholesterol (HDL-C) and triglycerides (TG) levels were determined using commercial kits according to the specifications of the manufacturer and a semiautomatic biochemical analyzer (PIOWAY-3000). Liver and feces samples were oven-dried (60 °C ± 2 °C for 72 h), and their lipids were extracted according to Folch et al [24]. CHOL and TG levels were determined using commercial kits, according to specifications of the manufacturer, and using a semi-automatic biochemical analyzer (PIOWAY-3000).

For histological analysis, sections from retroperitoneal adipose tissue (~100 mg) were fixed in buffered-formalin, paraffin embedded, sliced, and stained with hematoxylin-eosin. Results were obtained by means of a digital camera coupled to an optical microscope at 400x. All images were analyzed using the Axion Vision software. The hypertrophy was evaluated by measuring the diameter of 100 adipocytes per animal.

For cardiomyocyte diameter, hearts were fixed in 4% Bouin fixative solution, embedded in paraffin, and sectioned to 5 µm thickness. To determine myocyte cross-sectional area, heart sections were stained with hematoxylin and eosin and examined at 40× magnification. Only myocytes longitudinally cut with the nucleus centrally located in the cell and with cellular limits visible were used. The cross-sectional diameter (um) of the myocytes was traced using ImageJ software (National Institutes of Health), and determined by averaging 50 to 100 individual cardiomyocytes within the ventricular free wall over 5 or 6 sections per animal. A single investigator blinded to the experimental groups performed the analysis.

***Ex vivo* cardiac function**

In the last day of the experiment, animals were anesthetized (quetamin+xilazin / 50mg/kg + 10mg/kg) and decapitated 10–15min after a 400 IU intraperitoneal heparin injection. Hearts were perfused in a Langendorff apparatus (ML785B2, AD Instruments) and left ventricular pressure (± dP/dt) was continuously recorded according to the Langendorff technique, using the Labchart 8 software. Systolic tension, diastolic tension, coronary flow, heart rate, and dP/dT± values were the average of the recorded 30 min. All the dP/dT± measurements were normalized to heart weight. At the end of the cardiac function analysis, wet heart weights were recorded, normalized for the body weight, and expressed as muscle mass index ($mg.g^{-1}$), according to Almeida et al [25].

## Statistics

The statistical analyses were carried out using the Statistica 10.0 software. The experiment was carried out in a completely randomized design with three treatments (diets) and 12 repetitions (animals). Data were analyzed by one way ANOVA and Tukey test at $p<0.05$, using the Statistica 10.0 software. Figures were drawn by means of the SigmaPlot 11.0 software.

## Results

Replacing lard by *pequi* oil in a western diet did not affect body weight, having both groups HFS and HFS-PO, body weights higher than controls ($p<0.05$, Table 2). Food intake was similar between those groups (HFS and HFS-PO) and it was lower than controls ($p<0.05$, Table 2). There were no differences among experimental groups for energy intake (Table 2). However, both feed efficiency and energy efficiency ratios were equally higher for HFS and HFS-PO compared to CTRL ($p<0.05$, Table 2)

We also found no difference between HFS and HFS-PO for Lee Index, plasma cholesterol, triglycerides, HDL-C and hepatic cholesterol having both groups, values higher than CTRL (Table 3). This was also seen for hepatic and fecal cholesterol, fecal triglycerides, systolic blood pressure and heart rate. Otherwise,

**Table 2:** General characteristics of experimental groups after 12 weeks of treatment

| Variables | CTRL | HFS | HFS-PO |
|---|---|---|---|
| Body weight (g) | 400.66±6.48 [b] | 453.6111.91± [a] | 443.8811.86± [a] |
| Food intake (g) | 1853.82±74.44 [a] | 1580.6479.47± [b] | 1520.9134.80± [b] |
| Energy intake (Kcal) | 7318.89293.90± [a] | 7346.79369.37± [a] | 7069.20161.76± [a] |
| Feed efficiency ratio ($g.g^{-1}$) | 0.1900.009± [b] | 0.2500.007± [a] | 0.2400.007± [a] |
| Energy efficiency ratio ($g.\ Kcal^{-1}$) | 0.0200.002± [b] | 0.0500.001± [a] | 0.0500.001± [a] |

CTRL= AIN93G [21[; HFS = High fat and sucrose; HFS-PO = HFS with 27% of lard replaced by *pequi* oil. Feed efficiency ratio ($g.g^{-1}$) = body gain/food intake); Energy Efficiency ratio ($g\ Kcal^{-1}$) = body gain/energy intake. Values are expressed as mean ± standard error. Means followed by different letters (line) are different by One way-ANOVA and Tukey test ($p<0.05$)

**Table 3:** Cardiovascular risk factors of experimental groups after 12 weeks of treatment

| Variables | CTRL | HFS | HFS-PO |
|---|---|---|---|
| Lee index (g/cm$^3$) | 3.18±0.05 $^b$ | 3.47±0.11 $^a$ | 3.32±0.12 $^a$ |
| Adiposity index (%) | 5.67±1.03 $^b$ | 6.97±0.83 $^a$ | 5.54±2.04 $^b$ |
| Plama glucose (mg/dL) | 117.43±13.45 $^c$ | 139.05±17.32 $^a$ | 121.99±8.79 $^b$ |
| Plasma cholesterol (mg/dL) | 71.12±10.53 $^b$ | 84.76±10.74 $^a$ | 81.10±6.42 $^a$ |
| Plasma triglycerides (mg/dL) | 96.75±8.35 $^b$ | 112.88±6.19 $^a$ | 102.08±26.37 $^a$ |
| HDL-C(mg/dL) | 47.06±10.54 $^a$ | 39.35±8.03 $^b$ | 40.33±7.95 $^b$ |
| Hepatic cholesterol (mg.g$^{-1}$) | 21.36±0.53 $^b$ | 25.51±0.29 $^a$ | 25.17±0.57 $^a$ |
| Hepatic triglycerides (mg.g$^{-1}$) | 48.12±13.67 $^b$ | 62.04±8.10 $^a$ | 49.03±8.16 $^b$ |
| Fecal cholesterol (mg.g$^{-1}$) | 18.14±0.38 $^b$ | 19.83±0.55 $^a$ | 19.78±0.38 $^a$ |
| Fecal triglycerides (mg.g$^{-1}$) | 15.47±2.69 $^b$ | 18.18±3.21 $^a$ | 18.24±7.69 $^a$ |
| Systolic blood pressure (mmHg) | 134.40±17.77 $^b$ | 154.32±13.28 $^a$ | 154.40±18.70 $^a$ |
| Heart Rate (bpm) | 406.25±30.68 $^b$ | 446.70±60.41 $^a$ | 433.06±23.78 $^a$ |
| Muscle mass index (mg.g$^{-1}$) | 3.61±0.35 $^b$ | 4.95±0.49 $^a$ | 3.69±0.47 $^b$ |
| Cardiomyocytes diameter (µm) | 11.63±0.64 $^c$ | 15.59±0.54 $^a$ | 13.93±0.89 $^b$ |
| Retroperitoneal adipocyte diameter (µm) | 10835.75±1355.42 $^b$ | 20792.50±5642.29 $^a$ | 9888.1±1188.08 $^b$ |

CTRL= AIN93G [21]; HFS = High fat and sucrose; HFS-PO = HFS with 27% of lard *replaced* by *pequi*oil. Lee index (g/cm$^3$) = [3√body weight (g) ÷ nose = anus length(cm)] ×10); Adiposity index (%) = (epididymal pad +retroperitoneal pad)/body weight – (Σ epididymal pad +retroperitoneal pad) *100); Muscle mass index (mg.g$^{-1}$) = heart weight/body weight. Values are expressed as mean ± standard error. Means followed by different letters (line) are different by One way-ANOVA and Tukey test (p<0.05)

the Adiposity Index, which represents visceral adiposity, as well as retroperitoneal adipocyte diameter, hepatic triglycerides and the muscle mass index, were kept similar to CTRL by HFS-PO diet. In addition, plasma glucose levels and cardiomyocyte diameters were lower by HFS-PO diet compared to HFS, even though they were higher than CTRL (Table 3).

Overall, the western diet have compromised the *ex vivo* cardiac function of the animals, reducing the contractility (dP/dT+) and the relaxation (dP/dT-) efficiency in both groups HFS and HFS-PO, compared to CTRL (Figure). However, the HFS-PO diet attenuated the effects of the HFS diet, since ameliorated both indexes, as well the heart rate (Figure).

## Discussion

*Pequi* oil is a potential functional food due some of its chemical characteristics, such as high content of oleic acid and carotenoids [18-20]. Our laboratory has been investigating metabolic effects of this oil in healthy rats [18]. Therefore, our previous results have stimulated the continuity of the investigations, especially under obesogenic conditions. In this context, the western diet pattern seemed to be an adequate way to going on in further investigations. However, our first challenge was to define how

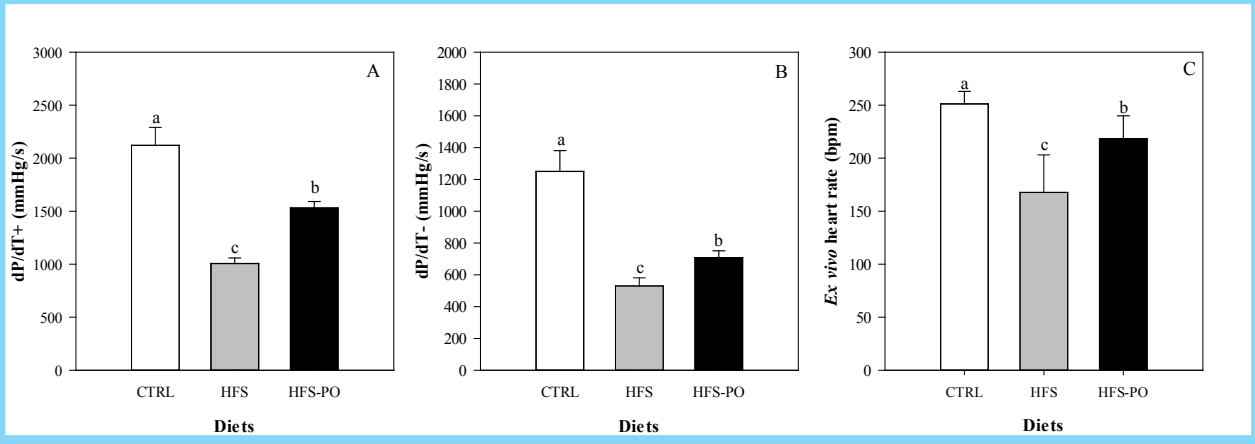


**Figure:** *Ex vivo* cardiac function of rats fed control diet (CTRL, AIN93G diet [21[); High fat sucrose diet (HFS) and HFS diet with 27% of lard replaced by *pequi* oil (HFS-PO). **dP/dT+ = Contractility index (A); dP/dT- = relaxation index (B**) and *ex vivo* heart rate (C). Values are expressed as mean ± standard error. Columns with different letters are different by one way-ANOVA and Tukey test (p<0.05)

much *pequi* oil to test and how to include this food in the experimental diets.

Therefore, we choose to include *pequi* oil in the diet, based on the intake of 02 servings per day (1 tablespoon = 1 serving), considering a 1.800 Kcal daily diet. Therefore, lard was replaced by *pequi* oil in order to modify the SFA/MUFA ratio and to increase the MUFA content, according Lopez-Huertas [26].

Although we did not observe differences in energy intake among groups, both HFS and HFS-PO had higher body weights at lower food intakes. HFS and HFS-PO diets were equally energy dense and higher than CTRL, so maybe, animals from those groups ate food as far as their energy needs. Indeed, according to Patterson & Levin [27], rats trend to intake food to account for their energy needs.

Otherwise, the higher body weights can be explained, at least in part, by the differential composition of the diets. It has been postulated that western diets, high in fats and sucrose, lead in rodents, to metabolic disturbances, higher body weight and fat, similarly to humans [28]. Intermittent intake of high glycemic meals in humans also leads to hormonal changes related to obesity and others CMD [29]. Besides, in rats, high fat intake at long term disrupts energy homeostasis, favoring body weight gain and metabolic disturbances, such as insulin resistance, dyslipidemia and others [30]. In our context, replacing lard by *pequi* oil did not affect differentially the effects of the western diet.

It is known that western diet, due its high content of SFA and high glycemic load, leads to visceral lipid accumulation [31]. It is also known that adipose tissue from this body compartment is metabolically more active, more sensitive to lipolysis and more resistant to insulin action than subcutaneous adipose tissue [32]. Therefore, we can infer that the modification in the fatty acids profile caused by *pequi* oil in the HFS-PO diet, led to a lower fat accumulation in this region. This result was reinforced by the fact that hypertrophy in the retroperitoneal adipocytes was also reduced by *pequi* oil diet.

This is an important result, since visceral fat accumulation is highly associated to metabolic disturbances [33]. Indeed, it has been postulated that fat topography and body distribution impact more than the body weight in CVD development [34]. In the *pequi* diet, these modifications can be related to the higher intake of MUFA. According Krishnan and Cooper [35], high-MUFA diets lead to lower fat deposition, especially in the visceral region because this fatty acid is able to increase its own oxidation rate. It has been postulated that a high MUFA diet could activate catabolic pathways that increases fat oxidation. This may be a result from the degradation of insulin-induced gene-1 protein, and therefore, inactivation of the transcription factor sterol regulatory element binding protein which promotes, among some effects, fat oxidation [36]. These effects are seen also in the liver [35].

Indeed, we also could observe a protector effect from *pequi* oil in the liver fat accumulation, especially in TG. So, we believe that the lower visceral fat accumulation accounted for this effect. Lower visceral fat accumulation implies in a lower lipolysis rate, which can also lower free fatty acids (FFA) in the portal microcirculation and then, lowering TG hepatic synthesis and preventing local accumulation [35].

Indeed, Hussein et al [37] has demonstrated that a high MUFA diet increased fat oxidation rate in several rat tissues, including liver. Recently, our group showed that a *pequi* oil diet reduced hepatic TG accumulation in healthy rat livers [18]. Furthermore, carotenoid supply from *pequi* oil could have weakened stress oxidative caused by the western diet, in the liver fat accumulation, since those compounds are potent exogenous antioxidants [38].

We did not observe any beneficial effect for *pequi* oil in lipid plasma markers. In this case, it seems, visceral and liver effects arising from the *pequi* oil diet, were not sufficient to lower serum cholesterol, LDL-cholesterol and TG, and increase HDL-cholesterol. The western diet provides dietary cholesterol and increases SFA supply. Both nutrients are associated to a reduced activity of LDL-cholesterol receptors in many tissues, increasing its blood levels [39]. Except steroidogenic tissues, cells in general, are unable to metabolize cholesterol, so the non-esterified cholesterol excess is carried by the liver, to the biliary and fecal output. However, this mechanism may become inefficient, and cholesterol accumulates in the hepatocytes [40]. Indeed, HFS and HFS-PO animals had higher hepatic cholesterol accumulation and higher fecal output. Although plasma TG had not been reduced by HFS-PO diet, in absolute values, it was lowered at 10% compared to HFS, which can be biologically important in the development of dyslipidemia.

The HFS diet also led to an increase of plasma glucose levels and the HFS-PO was able to mitigate this effect, but it did not restored normal values, as the CTRL diet. Considering that chronically FFA elevated levels upon western diets can cause disruption in the pancreatic β-cells, affecting insulin secretion and leading to hepatic and peripheral insulin resistance [41], this result in important. Although we could neither measure insulin levels nor evaluate insulin resistance in this study, maybe there was a delay in the glucose homeostasis disruption in the *pequi* oil group, which can contribute to prevent or hold back CVD.

Systolic blood pressure (SBP) was increased by the western diets (HFS and HFS-PO) and *pequi* oil did not bring any differential effect. Although high-MUFA diets have been associated to lower blood pressure in humans and animals [42], it has been also said that these effects can be lost when total dietary fat is high [43]. According to Heinonen et al [44], higher body weight is highly associated with the endothelium relaxation function, so excessive adiposity may lead to a arteries remodeling, turning them harder and thicker, which favor hypertension development [45].

In addition, the higher heart rate can be an adaptive response to

the higher SBP, since it leads to a higher ventricular afterload and, thereafter, heart rate elevation as a compensatory mechanism [46]. Otherwise, heart rate elevation has a direct impact over the arterial wall, causing mechanic stress and, possibly inflammatory effects in the vascular endothelium [47].

The cardiac function was impaired by both the western diets (HFS and HFS-PO) and it could be related to intrinsic myocardium properties. It is known that cardiac muscle has a high glucose and especially fatty acid uptake rate for energy production [48]. However, western diets are able to increase circulating FFA, which can leads to a high fatty acid cardiac uptake [49], and disruption in the activity of calcium transient proteins responsible by cardiac contractility [50]. Therefore, the lower contractility in HFS and HFS-PO animal hearts could be related to reduction in the L-calcium channels from cardiomyocyte cellular membranes and in sarcoplasmic ryanodine receptor activities. In this was calcium release during systole is impaired, reducing cardiac contractility as well [51].

Bradycardia shown by HFS and HFS-PO animals, according to Frank-Starling law, should be followed by an increase in contractility [52], not by a reduction, as seen. This could be explained because western diets suppress cardiac function overall and, in this situation, maybe there is a depression of the sinus node pacemaker activity [53].

However, it is important to consider that, although cardiac function was impaired in the western diet groups, replacing lard by *pequi* oil has attenuated the damage caused by that diet. Upon western diet, lysogenic pathways are stimulated, using as substrates SFA and simple carbohydrates to produce endogenous SFA or MUFA. However, synthesis of desaturases does not follow the fatty acid generation, leading to an imbalance into SFA/MUFA ratio and stimulating the replacement of MUFA by SFA in cardiomyocytes, which is also associated with contractile dysfunction of the cardiac muscle [54]. We believe that the high supply of MUFA in the *pequi* oil diet could have favored their incorporation into the cardiomyocyte membranes, ameliorating the dysfunction cause by SFA/MUFA impairment.

## Conclusion and perspectives

Taken together, our data indicates that replacing lard by *pequi* oil in a western diet reduced visceral and hepatic lipid accumulation, as well as, attenuated deleterious effects from this dietary pattern in the cardiac function. Although it did not influence other markers, this can contribute for slowing up cardiovascular disturbances associated to the western diet pattern.

Therefore, future investigations should be directed to investigate mechanisms that are beyond the visceral and hepatic lipid accumulation *pequi* oil lowering-effect, including molecular pathways controlling lipid metabolism and redox state in those these tissues.

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