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Effects of Baru Almond and Brazil Nut Against Hyperlipidemia and Oxidative Stress *In Vivo*

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Abstract

This study evaluated the effect of baru (*Dipteryx alata* Vog.) almond, an edible seed native from Brazilian Savanna, and Brazil nut (*Bertholletia excelsa* H. B. K.) on serum lipid profile and hepatic lipid peroxidation in rats fed high-fat diets. Four groups of eight young adult male Wistar rats were treated for nine weeks with one of the following diets: high-fat diets - 0.1% colic acid + 1% cholesterol + 5% lard + 15% of lipid from lard, baru almond or Brazil nut - and reference diet (7% soybean oil). Groups fed with baru almond and Brazil nut showed lower serum contents of total cholesterol and triacylglycerols than those of lard group. Baru almond group also showed higher HDL-c concentration than those of Brazil nut and lard groups, similar to that of reference group. Lipid peroxidation (through total malondialdehyde) was lower and vitamin E content was higher in the livers of the animals treated with baru almond and Brazil nut than those of lard group. These results indicate that the Brazilian native oilseeds, especially baru almond, have great potential for dietary use in dyslipidemia prevention and control.

Keywords: *Bertholletia*, *Dipteryx*, high-fat diet, lipid peroxidation, monounsaturated fat, rat

1. Introduction

The consumption of edible seeds and nuts has been associated with a reduction in cardiovascular diseases risk (Lee, Lavie, O'Keefe, & Milani, 2011). Studies using experimental models of dyslipidemia have showed evidence in an improvement of serum lipid profile and antioxidant status in animals fed oilseeds, like peanut (Emekli-Alturfan, Kasikci, & Yarat, 2007; Stephens, Dean, Davis, Osborne, & Sanders, 2010), flaxseed (Cintra et al., 2006) and pistachio (Alturfan, Emekli-Alturfan, & Uslu, 2009). Their beneficial effects on cardiovascular health are attributed to their high contents of unsaturated fatty acids, particularly monounsaturated acids. In addition, oilseeds are rich in antioxidant nutrients and other bioactive compounds, such as phenolics, which probably contribute to the decrease of cardiovascular diseases risk (Segura, Javierre, Lizarraga, & Ros, 2006; Lee et al., 2011).

Even though edible seeds and nuts have high contents of unsaturated fat and bioactive compounds, there are remarkable differences among their chemical composition (Freitas & Naves, 2010; Venkatachalam & Sathe, 2006), which might generate different health effects. The baru almond (*Dipteryx alata* Vog) is an edible seed from Brazilian Savanna, and it is locally consumed in roasted form and as an ingredient of traditional and exotic dishes, but it has a relatively high cost, mainly due to its handmade conditions of harvesting and processing. Baru almond contains high amounts of monounsaturated fatty acids (MUFA), zinc and phenolic compounds (Marin, Siqueira, & Arruda, 2009; Freitas & Naves, 2010; Sousa, Fernandes, Alves, Freitas, & Naves, 2011). On the other hand, Brazil nut (*Bertholletia excelsa* H.B.K.) is a true nut, which is produced in large scale and is consumed worldwide; consequently, it is cheaper than baru almond. It contains high levels of polyunsaturated fatty acids (PUFA) and selenium (Segura et al., 2006; Venkatachalam & Sathe, 2006; Freitas & Naves, 2010).

The effect of baru almond and Brazil nut consumption on serum lipid profile and oxidative status has not been studied in animal model of dyslipidemia. Therefore, the aim of this study was to investigate the effect of these native Brazilian oilseeds on serum lipid profile and hepatic lipid peroxidation in animals treated with high-fat diets.

2. Method

2.1 Fruit Collection and Sample Preparation Procedures

Baru seed and Brazil nut were purchased locally in the city of Goiânia, GO, Brazil. Raw baru seeds were roasted at 140 °C for 30 min in an electric oven (Fernandes, Freitas, Czeder, & Naves, 2010); then, they were peeled. Brazil nuts were purchased peeled and raw, because this nut is usually consumed *in natura*. The samples were ground in a multiprocessor and then used for chemical analyses and for formulation of the experimental diets.

2.2 Chemical Composition of the Samples

The following analyses were performed: moisture (Association of Official Analytical Chemists [AOAC], 2012); total nitrogen, according to the micro-Kjeldahl method and converted into protein using the factor 6.25 (AOAC, 2012); total lipids, extracted using the technique of Bligh and Dyer (1959) and subsequently estimated by gravimetry; ash, by burning in an oven at 550 °C, and total dietary fiber (AOAC, 2012). Total carbohydrates were estimated by difference, subtracting the values obtained for moisture, protein, fat, ash and fiber from one hundred. The energy value of the samples was estimated based on the proximate composition data, considering the Atwater conversion factors, which are 4, 4, and 9 kcal/g for protein, carbohydrate, and lipid, respectively. The analyses were performed in triplicate.

2.3 Antioxidant Nutrient Analysis

Zinc was characterized and quantified, in triplicate, by atomic absorption spectrophotometry (Perkin Elmer Analyst-200 spectrometer). Samples were incinerated and then dissolved with concentrated hydrochloric acid (analytical grade). Specific instrumental parameters (lamp wavelength, lamp current and slit width) were used for the mineral analysis. Selenium was analyzed by atomic absorption spectrophotometry (HITACHI, model Z-5000, Tokyo) with generation of hydrides in a quartz tube (AOAC, 2012).

Vitamin E was estimated in the samples by α-tocopherol analysis, according to Klejdus et al. (2004). Samples of baru almond and Brazil nut (approximately 2.0 g) were weighed in conical centrifuge tubes. Then, 8 mL of absolute ethanol were added and it was stirred for approximately 30 s in a mixer, next 10 mL of distilled water were added and again stirred on vortex for 30 s. Thereafter, 8 mL of hexane were added and stirred for 30 s on vortex. Afterwards, the samples were centrifuged at 3.500 rpm for 5 min. After centrifugation, the upper layer was taken and filtered (filter 0.45 mM). The standard curve was prepared from a solution with α-tocopherol dissolved in methanol. The samples were injected in duplicate on HPLC apparatus (Gilson, Wisconsin, USA), in column ODS2 type, with 10 cm × 4.6 mm (ES Industries, Berlin, Germany). The flow rate was 0.7 mL/min and auto sampler injection was 3 μL.

2.4 Fatty Acid Composition of the Samples

To determine the fatty acid composition, the samples were extracted as described by Folch, Less and Stanley (1957) and esterified according to Hartman and Lago (1973). Fatty acids were separated by Finnigan Focus GC gas chromatography (Thermo, Austin, TX, USA) with a flame ionization detector and capillary column (RESTEK, Bellefonte, PA, USA - polyethylene glycol crossbond - 30 m × 0.25 mm) of fused silica. The running conditions were: carrier gas – hydrogen (2 mL/min); make up gas – nitrogen (28 mL/min); hydrogen (30 mL/min), and synthetic air (300 mL/min) in order to maintain the detector flame. The injection volume was 1 μL and split ratio of 2:98. Retention time, peak area, and area relative values were obtained using the Chrom Quest 4.1 software. Fatty acids were identified and quantified by reference to the calibration curve prepared with standard methyl esters of fatty acids (Sigma Aldrich). The analyses were performed in triplicate.

2.5 Biological Assay

The experiment was carried out with young adult male Wistar rats purchased from Bioagri Laboratories Experimental Animal Center (Federal District, Brazil). In the experiment, 32 rats were randomly divided into 4 groups (n= 8/group), whose averages of body weight (218-239 g) were not significantly different ($p < 0.05$). The rats were kept in individual cages for 10 weeks (one week acclimation to the experimental facility and 9 weeks of assay), in a temperature-controlled room at 22 ± 2 °C and under a 12:12 h of light: dark photoperiod. All procedures with animals were conducted according to the guide for the care and use of laboratory animals (National Research Council, 2011), and were approved by the Ethics Committee on Animal Use of the Federal

University of Goiás (Protocol nº 153/08).

Three high-fat diets with similar contents of protein, lipid and energy, and a reference diet (7% soybean oil) were prepared (Table 1), according to AIN-93G (Reeves, Nielsen, & Fahey Junior, 1993). High-fat diets were formulated with 5% lard (Sadia), 1% cholesterol (Sigma Aldrich) and 0.1% colic acid (Rhoster) (Cintra et al., 2006; Riediger et al., 2008), and 15% lipid from lard, baru almond or Brazil nut. The chemical analysis of the diets was performed following the methods described in item 2.2. The total lipid concentration of the high-fat diets was around 20% (Table 1), reaching approximately three times the regular content of reference diet's lipid (Reeves et al., 1993).

At the end of nine weeks of experimentation, overnight-fasted animals were euthanized with a mixture of ketamine and xylazine (0.2 mL per 100 g of body weight); blood samples were collected by heart puncture, and serum was taken after centrifugation at 2400 g for 15 min. Liver tissue samples were collected and immediately stored at -80 °C for further analyses.

Table 1. Ingredients and chemical composition of the experimental diets

Component (g/100 g)	High-fat diet			Reference diet
	Lard	Baru almond	Brazil nut	
Ingredient				
Casein	21.10	12.35	17.48	21.10
Baru almond	-	35.38	-	-
Brazil nut	-	-	23.03	-
L-cystine	0.32	0.19	0.26	0.32
Soybean oil	-	-	-	6.68
Lard	20.00	5.00	5.00	-
Cholesterol	1.00	1.00	1.00	-
Cholic acid	0.10	0.10	0.10	-
Cellulose	5.00	0.90	3.18	5.00
Mineral mix	3.50	3.50	3.50	3.50
Vitamin mix	1.00	1.00	1.00	1.00
Choline birtatrate	0.25	0.25	0.25	0.25
Corn starch	47.73	40.33	45.20	62.15
Chemical composition				
Protein	17.62	18.29	18.61	17.00
Lipids	19.82	20.29	20.17	6.88
Energy (kcal/100g)	479	481	481	413

Baru almond and Brazil nut were added to diets to supply 15 g of lipids/100 g of diet.

2.6 Blood Lipids

Total serum cholesterol (TC), triacylglycerol (TG) and high-density lipoprotein cholesterol (HDL-c) concentrations were analyzed using diagnostic kits (Labtest Diagnostics SA, Minas Gerais, Brazil).

2.7 Hepatic Analyses

Lipid peroxidation was estimated in the liver by the analysis of malondialdehyde (MDA), according to the method proposed by Gerard-Monnier et al. (1998). For that, 100 mg of liver tissue were weighed and homogenized in 1 mL of 1% KCl solution. Thereafter, 650 µL of 10 mM solution of 1-methyl-2-phenylindole in acetonitrile and methanol (2:1, v/v) and 150 µL of pure HCl (37%) were added to the homogenate. The vials were stirred on vortex and incubated in a water bath at 45 °C for 40 min, centrifuged at 4000 rpm for 15 min, and the supernatant absorbance was read at a wavelength of 586 nm in a SpectraMax M-5 (Molecular Advices,

California, USA). MDA concentration was calculated by constructing a hydrolyzed 1,1,3,3-tetramethoxypropane (TMP) curve.

Hepatic reduced glutathione (GSH) concentration was evaluated according to the method described by Sedlak and Lindsay (1968). Hepatic tissue protein concentration was determined by the biuret method, in order to correct the MDA and GSH results. Protein content was analyzed by a commercial kit (Labtest Diagnostics S.A., Minas Gerais, Brazil). Copper ions of biuret reagents react with peptide bonds of proteins resulting in a purple solution, which was read at 545 nm in a SpectraMax M-5.

The analysis of hepatic vitamin E was performed as described by Arnaud, Fortis, Blachier, Kia and Favier (1991). A sample of 200 mg of liver tissue was weighted and 500 μL of an ethanol, NaOH and BHT (butylated hydroxyl toluene) mixture were added. Then, samples were incubated in water bath at 50 °C for 40 min, with subsequent stirring for 1 min, and were centrifuged (5 min at 3500 rpm). Samples were dried in liquid nitrogen and frozen at -80 °C until the time of injection in HPLC (column C-18 type, Shimpack CLC-ODS 4.6 × 25 cm and flow rate of 2.0 mL/min). Concentration was calculated using internal and external standards of α-tocopherol.

2.8 Statistical Analysis

Data were expressed as mean ± standard deviation. Student's t test was used for comparisons between baru almond and Brazil nut's composition. ANOVA followed by Tukey test was used for comparisons between experimental data. Differences were considered significant when $p < 0.05$. Data were analyzed using Statistica software (Stat Soft Inc, version 7, Tulsa, USA).

3. Results

Baru almond showed high content of vitamin E and low amounts of selenium, and Brazil nut showed high selenium concentration. Both baru almond and Brazil nut have high contents of zinc. Regarding the lipid profiles of the samples, baru almond has more MUFA (mainly oleic acid) than Brazil nut, which showed higher contents of saturated fatty acids (SFA) and PUFA (linoleic acid) than baru almond (Table 2).

After nine weeks on high-fat diets, body weights were similar between groups, except for the baru almond group, which showed the lowest final body weight compared to high-fat diet groups. However, it was similar to that of reference group (Table 3).

Animals fed high-fat diets containing baru almond or Brazil nut showed lower TC and TG serum concentrations than those of lard group ($p < 0.05$). Compared to baru almond and reference groups, lard group's animals presented high TC and TG serum concentrations, as well as low HDL-c serum concentration, thus endorsing the dyslipidemia model (Table 3).

Brazil nut group showed a lower lipid peroxidation level than that of lard group; however, baru almond group had the lowest lipid peroxidation level among groups fed high-fat diet. The higher MDA value found in lard group, compared to the other groups, confirm the oxidative effect of dyslipidemic diets on liver tissue. Hepatic GSH concentrations were not different among high-fat diet groups, but hepatic vitamin E contents of the rats treated with baru almond and Brazil nut were higher than that of lard group (Table 3).

4. Discussion

Experimental studies have evaluated the effects of edible seeds and nuts on serum lipid profile (Cintra et al., 2006; Emekli-Alturfan et al., 2007; Alturfan et al., 2009). Nevertheless, this is the first study that investigated the effects of baru almond and Brazil nut on serum lipid profile and lipid peroxidation in animal model of high-fat diet. Brazil nut and baru almond consumption prevented hyperlipidemia and lipid peroxidation on liver tissue of rats fed high-fat diets, suggesting a protective role of these foods against the high intake of saturated fat and cholesterol.

The effect against hyperlipidemia observed in this study is probably related to the fatty acid profile of the Brazilian oilseeds, as they are rich in unsaturated fatty acids (baru almond = 70 g/100 g; Brazil nut = 77 g/100 g total lipids). The content of unsaturated fatty acid in foods is inversely related to the serum cholesterol concentrations, as observed in the study of Mohamed, Hussein, Bhathena and Hafez (2002). In other study, hamsters treated for 24 weeks with a high-fat diet supplemented with peanuts, which is an edible seed rich in unsaturated fatty acid (82 g/100 g total lipids (Freitas & Naves, 2010), exhibited lowered concentrations of CT, LDL-c and VLDL-c, and lowered risk of developing atherosclerosis (Stephens et al., 2010).

Table 2. Baru almond and Brazil nut's composition

Content	Baru almond	Brazil nut
Chemical composition (g/100 g)		
Moisture	3.17 ± 0.11[a]	2.35 ± 0.04[b]
Protein	28.94 ± 0.30[a]	13.36 ± 0.03[b]
Total lipids	42.40 ± 0.65[b]	65.13 ± 0.98[a]
Carbohydrates	10.79	6.05
Total dietary fiber	11.70 ± 0.20[a]	9.53 ± 0.06[b]
Soluble fiber	2.40 ± 0.10[a]	1.50 ± 0.00[b]
Insoluble fiber	9.30 ± 0.10[a]	8.03 ± 0.06[b]
Ash	3.01 ± 0.04[b]	3.58 ± 0.02[a]
Energy (kcal/100 g)	574.01 ± 6.15[b]	663.81 ± 4.98[a]
Antioxidant nutrient (mg/100g)		
Vitamin E	21.40 ± 1.40[a]	5.96 ± 0.11[b]
Zinc	6.74 ± 0.04[b]	7.52 ± 0.20[a]
Selenium	0.26 ± 0.03[b]	100.81 ± 11.07[a]
Fatty acid (g/100 g total lipids)		
C12:0	0.12 ± 0.01	not detected
C14:0	0.18 ± 0.03[a]	0.04 ± 0.00[b]
C16:0	5.94 ± 0.48[b]	14.15 ± 0.03[a]
C17:0	0.04 ± 0.00	0.06 ± 0.00
C18:0	5.42 ± 0.26[b]	7.99 ± 0.34[a]
C20:0	1.27 ± 0.03[a]	0.09 ± 0.00[b]
C24:0	not detected	0.09 ± 0.00
Total SFA	**12.98 ± 0.76[b]**	**22.43 ± 0.37[a]**
C17:1	0.05 ± 0.01	not detected
C18:1	41.41 ± 2.08[a]	33.66 ± 0.33[b]
C20:1	0.13 ± 0.00	not detected
C24:1	3.90 ± 0.08[a]	0.07 ± 0.01[b]
Total MUFA	**45.49 ± 2.01[a]**	**33.74 ± 0.32[b]**
C18:2	24.40 ± 1.31[b]	43.77 ± 0.04[a]
C18:3	not detected	not detected
C20:2	0.10 ± 0.02	not detected
C22:6	0.29 ± 0.02	not detected
Total PUFA	**24.79 ± 1.31[b]**	**43.77 ± 0.04[a]**

Data are mean ± standard deviation of 3 replicates. [a,b]Different letters in the same row indicate significant difference ($p<0.05$) by Student's t test. SFA: saturated fatty acids; MUFA: monounsaturated fatty acids; PUFA: polyunsaturated fatty acids.

Beyond the protective effect of the two oilseeds tested in the present study, baru almond showed better effects on serum lipid profile and lipid peroxidation than Brazil nut, since the animals fed with baru almond had higher serum HDL-c concentration, similar to that of the reference group, and lower hepatic MDA content than those of Brazil nut (Table 3). These differences might be related to the fatty acid profiles of these foods, especially by the high MUFA contents of baru almond (Table 2). The consumption of peanuts, which is also an edible seed with

high MUFA contents (Freitas & Naves, 2010), improved HDL-c concentrations in rats treated for 12 weeks with a hyperlipidemic diet (Emekli-Alturfan et al., 2007). MUFA, especially oleic acid, have an important role in HDL-c increase, and the enrichment of lipoproteins with MUFA increases their resistance to oxidation, as MUFA is less prone to oxidation than PUFA (Cintra et al., 2006; Freitas & Naves, 2010; Lee et al., 2011). Furthermore, PUFA, especially linoleic acid, are associated with increased synthesis of pro-inflammatory molecules, such as IL-10, which can reduce HDL-c concentrations (Kris-Etherton & Yu, 1997; Moraitis et al., 2015), as observed in this study for Brazil nut. The baru almond fatty acid profile is being reported for the first time in our study, and the Brazil nut fatty acid profile described in this paper is consistent with the literature (Venkatachalam & Sathe, 2006; Freitas & Naves, 2010).

Table 3. Wistar rat's body weight, diet and lipid intakes, serum lipid profile and hepatic parameters after nine weeks of treatment with high-fat diets containing baru almond or brazil nut in comparison to lard and reference diets

	High-fat diet			Reference diet
	Lard	Baru almond	Brazil nut	
Body weight (g)				
final	367.38 ± 26.33^{a}	300.03 ± 22.55^{b}	361.92 ± 34.90^{a}	$336.93 \pm 32.97^{a,b}$
gain	128.12 ± 24.85^{a}	68.50 ± 8.80^{b}	126.88 ± 28.35^{a}	119.00 ± 7.26^{a}
Total intake (g)				
diet	$1003.40 \pm 65.75^{a,b}$	908.37 ± 57.89^{b}	1051.65 ± 70.40^{a}	1169.80 ± 94.65^{a}
lipid	198.87 ± 13.03^{a}	184.31 ± 11.75^{a}	212.12 ± 14.20^{a}	76.24 ± 8.22^{b}
Serum lipid (mg/dL)				
TC	217.50 ± 5.62^{a}	126.50 ± 11.21^{b}	119.33 ± 11.50^{b}	57.67 ± 8.08^{c}
TG	223.38 ± 3.00^{a}	60.67 ± 1.53^{c}	57.00 ± 2.31^{c}	71.00 ± 1.73^{b}
HDL-c	24.25 ± 0.96^{b}	36.33 ± 1.43^{a}	23.00 ± 3.16^{b}	38.50 ± 3.78^{a}
Hepatic parameter				
MDA (mmol/g protein)	345.69 ± 7.99^{a}	92.31 ± 7.80^{c}	153.65 ± 15.32^{b}	71.20 ± 2.17^{d}
GSH (µmol/g protein)	1.32 ± 0.16^{b}	1.78 ± 0.18^{b}	1.58 ± 0.45^{b}	5.06 ± 0.33^{a}
Vitamin E (µmol/g tissue)	7.22 ± 0.01^{c}	10.81 ± 1.31^{b}	11.62 ± 0.62^{b}	15.88 ± 0.61^{a}

Data are mean ± standard deviation. [a-d] Different letters in the same row indicate significant difference ($p<0.05$ by Tukey's test). TC: total cholesterol; TG: triacylglycerol; HDL-c: High Density Lipoprotein; MDA: malondialdehyde; GSH: hepatic reduced glutathione.

The higher contents of hepatic vitamin E observed in the animals' livers of baru almond and Brazil nut groups than that of lard group are probably associated to their lowered hepatic lipid peroxidation, estimated by MDA (Table 3). Vitamin E, especially as α-tocopherol, may block the initiation and inhibit the propagation of lipid peroxidation, as an effective *in vivo* antioxidant (Kurtoglu et al., 2008; Niki, 2014). The role of vitamin E in the reduction of lipid peroxidation has been confirmed in the literature, since the supplementation with α-tocopherol (100 IU/kg) in animals fed high-fat diet for 20 weeks was associated to significant reduction in serum and hepatic MDA concentrations (Kim et al., 2013). In addition to its antioxidant role, α-tocopherol may act in the regulation of genes related to lipids uptake (Azzi et al., 2004), as well as in transcription factors involved in fat metabolism, as the peroxisome proliferator-activated receptors (PPAR) (Yu et al., 2003). On the other hand, hepatic GSH concentrations were not different between the high-fat diet groups (Table 3). One possible explanation for the lower GSH levels in animals treated with baru almond and Brazil nut would be related to the GSH utilization in vitamin E recovery. Glutathione has important role on neutralizing peroxides and on the protection of cells against oxidative stress (Niki, 2014).

Although it is known that PUFA are more prone to oxidation than MUFA, edible seeds and nuts are rich in antioxidants that may protect those fatty acids from oxidative damage (Lee et al., 2011). This effect has been observed in experimental studies, since peanut and pistachio consumption improved the oxidative balance in rats

fed high-fat diets (Emekli-Alturfan et al., 2007; Alturfan et al., 2009). Baru almond is rich in vitamin E and Brazil nut is the richest dietary source of selenium (Table 2). Therefore, the present study's data may be explained by the synergic effect of the fatty acid profiles, antioxidant micronutrients, and probably other compounds present in these foods, like phenolics (Venkatachalam & Sathe, 2006; Marin, Siqueira, & Arruda, 2009; Lee et al., 2011; Sousa et al., 2011). A study using baru almond in animal model reported that the consumption of baru seed protected the animals' tissues against iron supplementation-induced oxidative stress, and the authors suggest that the baru almond's phytic acid content was partially responsible for this antioxidant effect (Siqueira et al., 2012).

Even though oilseeds are high-energy foods, their consumption is not associated with weight gain (Martínez-González & Bes-Rastrollo, 2011). According to Mattes and Dreher (2010), intake of edible seeds and nuts promotes satiety, especially due to their significant content of proteins and fibers, coupled with the need for intensive chewing upon consumption. Some authors also suggest that the intake of those foods is associated with an increase in energy expenditure or thermogenesis (Casas-Agustench et al., 2011). Therefore, the lower food consumption and body weight gain of baru almond group (Table 3) might be related to baru almond's higher contents of protein and fiber than those of Brazil nut (Table 2), and to the higher concentration of baru almond (35 g/100 g) than Brazil nut (23 g/100 g) in the experimental diets (Table 1). The daily energy consumption of baru almond group (69 kcal) was lower than that of Brazil nut group (80 kcal), and the energy intake of lard group was similar to that of reference group (76 kcal/d), which justify the similar body weight gain of these two last groups (Table 3).

In previous studies with humans, the supplementation with Brazil nut did not increase the serum HDL-c concentration of normolipidemic subjects (Strunz, Oliveira, Vinagre, Lima, & Cozzolino, 2008), but this effect was observed in obese women with dyslipidemia (Cominetti, Bortoli, Garrido Jr., & Cozzolino, 2012). These data suggest that the effects of Brazil nut against dyslipidemia may be more evident in pathologic conditions. Concerning baru almond, a daily consumption of 20 g for 6 weeks decreased serum total cholesterol, LDL-c and non-HDL-c in mildly hypercholesterolemic individuals (Bento et al., 2014). Data of the present study and of the literature justify further investigations in order to clarify the role of these Brazilian oilseeds on prevention and treatment of dyslipidemia.

Overall, our data suggest that the consumption of baru almond and Brazil nut has a potential protective role against hyperlipidemia and lipid peroxidation. Nevertheless, the protective effect of baru almond was the most evident, since its consumption improved HDL-c concentrations and greatly decreased lipid peroxidation. Based on our results, these foods, especially baru almond, have a potential use in healthy diets, aiming at reducing hyperlipidemia and preventing related chronic diseases.

5. Conclusions

Baru almond and Brazil nut improve serum lipid profile and protect against oxidative stress in an animal model of dyslipidemia. These beneficial effects are more evident for baru almond than for Brazil nut. The high contents of monounsaturated fatty acid of baru almond may be related to the antidyslipidemic effect observed in this study. Therefore, baru almond has great potential for dietary use in dyslipidemia prevention and control.

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