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Original/*Otros*

The consumption of acai pulp changes the concentrations of plasminogen activator inhibitor-1 and epidermal growth factor (EGF) in apparently healthy women

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Abstract

Introduction: obesity, characterized by adiposity excess, is associated with endothelial dysfunction and possible inflammatory state with release of cytokines that determine endothelial function and can trigger chronic diseases. The dietary pattern are associated with the synthesis these cytokines. Fruits as the acai, which is rich in flavonoids, have a direct and beneficial effect on the control of this inflammatory process through the exercised antioxidant capacity.

Objective: to evaluate the effect of acai pulp consumption on the inflammatory markers, anthropometric measurements, body composition, biochemical and dietary parameters in healthy women.

Methods: forty women, were divided in 25 eutrophic and 15 with overweight. They intaked 200 g of acai pulp during 4 weeks. Anthropometric measurements, body composition, inflammatory markers, biochemical data, dietary intake and dietary antioxidants capacity were evaluated before and after the intervention.

Results and discussion: after the intervention, there was significant increase of EGF (p=0.021) and PAI-1(p=0.011) in overweight women. Moreover, there was increase in body weight (p=0.031), body mass index (p=0.028), percentage of truncal fat (p=0.003) and triceps skinfold thickness (p=0.046) in eutrophic women.

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EL CONSUMO DE PULPA ACAI CAMBIA LAS CONCENTRACIONES DE ACTIVADOR DEL PLASMINÓGENO INHIBIDOR 1 Y FACTOR DE CRECIMIENTO EPIDÉRMICO (EGF) EN MUJERES APARENTEMENTE SANAS

Resumen

Introducción: la obesidad, que se caracteriza por el exceso de adiposidad, se asocia con disfunción endotelial y posible estado inflamatorio con liberación de citoquinas que determinan la función endotelial y pueden desencadenar enfermedades crónicas. El patrón de dieta está asociado con la síntesis de estas citoquinas. Los frutos de el acai, que es rico en flavonoides, tienen un efecto directo y positivo en el control de este proceso inflamatorio a través de los ejercicios de la capacidad antioxidante.

Objetivo: evaluar el efecto del consumo de pulpa de acai en los marcadores inflamatorios, las medidas antropométricas, la composición corporal y los parámetros bioquímicos y dietéticos en mujeres sanas.

Métodos: cuarenta mujeres fueron divididas en 25 eutróficas y 15 con sobrepeso. Se las adeministró 200 g de pulpa de acai durante 4 semanas. Antes y después de la intervención se evaluaron: medidas antropométricas, composición corporal, marcadores inflamatorios, datos bioquímicos, ingesta dietética y antioxidantes en la dieta.

Resultados y discusión: después de la intervención, hubo un aumento significativo de EGF (p=0,021) y PAI-1 (p=0,011) en las mujeres con sobrepeso. Por otra parte, en las mujeres eutróficas hubo aumento del peso corporal (p=0,031), el índice de masa corporal (p=0,028), el porcentaje de grasa del tronco (p=0,003) y el espesor del pliegue cutáneo del tríceps (p=0,046). Sin embargo, el espesor del pliegue cutáneo (p=0,018) y la grasa corporal total (p=0,016) se redujeron en las mujeres con sobre-

However, the skinfold thickness (p = 0.018) and total body fat (p = 0.016) decreased in overweight women. There was reduction of total protein (p = 0.049) due to the globulin reduction (p = 0.005), but the nutritional status was maintained in eutrophic group.

Conclusion: **the intake of 200g acai pulp, modulated the EGF and PAI-1 expression, possibly by modulation of acai on the parameters of body composition, dietary, clinical, biochemical and inflammatory, led to a redistribution and resizing of body fat of the trunk area, and presumably increased visceral fat.**

peso. Hubo una reducción de la proteína total (p = 0,049) debida a la disminución de globulina (p = 0,005), pero el estado nutricional se mantuvo en el grupo eutrófico.

Conclusión: **la ingesta de 200 g de pulpa de acai modula el EGF y PAI-1 de expresión, posiblemente por la modulación del acai en los parámetros de la composición corporal, la dieta, clínicos, bioquímicos e inflamatorios, lo que dio lugar a una redistribución y modificación del tamaño de la grasa corporal de la zona del tronco, y, presumiblemente, un aumento de la grasa visceral.**

Abbreviations

%BF: Body fat percentage
%TBF: Total body fat percentage
%TF: Truncal fat percentage
aMED: Score Alternative Mediterranean Diet Score
AC: Abdominal circumference
AFMI: Arm fat-muscle index
BI: Biceps Skinfold
BIA: Tetrapolar electrical bioimpedance
BMI: Body Mass Index
CD: Chronic diseases
EGF: Epidermal Growth Factor
ELISA: Enzyme linked immunosorbent assay
FFQ: Food Frequency Questionnaire
FRAP: Ferric Reducing Antioxidant Power
GLUT-4: Insulin-sensitive glucose transporter
HC: Hip circumference
HDL–c: High Density Lipoprotein cholesterol
HEI: Healthy Eating Index
HOMA-IR: Homeostatic model assessment of insulin resistance
IL-1: Interleukin-1
IL-8: Interleukin-8
IQD-I: International Quality Diet Index
LDL–c: Low Density Lipoprotein cholesterol
MAPK: Mitogen activated protein kinase
METs: Metabolic equivalent units
NF-kB: Nuclear Transcription Factor Kappa Beta
DQI: Original Diet Quality
PAI-1: Plasminogen activator inhibitor-1
PDGF–AA: Platelet Derived Growth Factor
PI3-k: Phosphatidylinositol 3-kinase
PKB/AKT: Protein kinase –B
QUICKI: Quantitative insulin sensitivity check index
REE: Resting Energy expenditure
RFS: Recommended food score
SUB: Subscapular Skinfold
SUPR: Supra-iliac Skinfold
TEAC: Trolox equivalent antioxidant capacity

TFEQ: Three Factor Eating Questionnaire
TNF-α: Tumor Necrosis Factor-α
TRI: Triceps Skinfold
TUA: Total upper arm area
UAC: Upper arm circumference
UAMC: Upper arm muscle circumference
UFA: Upper arm fat area
UMA: Upper arm muscle area
UMAc: Corrected upper arm muscle area
VEGF: Vascular Endothelial Growth Factor
WC: Waist circumference
WHR: Waist-hip ratio

Introduction

The obesity is considered a complex disease of multifactorial and endemic etiology. Its clinical feature is associated with increased morbidity and mortality by chronic diseases (CD), such as: diabetes type 2, hypertension and cardiovascular disease. The assumption that the metabolic changes lead to adiposity excess is essential to extensively studied hypothesis that the distribution of body fat, as well as truncal and or visceral fat increase, seems to be related to the CD, and the main link involves oxidative and inflammatory state[1,2].

As a result, there are evidences that a higher level of oxidative stress in obese is an important factor which relates to the increase of the inflammatory process and the modification of endothelial function, leading to a state of low-grade chronic inflammation[2], accompanied by the expression of markers of coagulation cascade and growth factors; quoting the plasminogen activator inhibitor-1 (PAI-1), which in situations of larger adiposity is released by adipocytes performing the function of physiological inhibitor of fibrinolysis, being able to lead the formation of thrombi[3], the fibrinogen, as an acute phase reactant protein, in response to inflammation, may accelerate the formation of these clots/thrombus[4].

Together, the growth factors can participate of this inflammatory process. The vascular endothelial growth factor (VEGF) induced by endothelial cells and inflammatory cytokines, interleukin-1 (IL-1), interleukin-8 (IL-8) and tumor necrosis factor (TNF-α), is a major regulator of angiogenesis and repair of the lesions generated by the inflammation[5].

As the platelet derived growth factor (PDGF-AA), the transforming growth factor-alpha (TGF-α) and epidermal growth factor (EGF) released by inflammatory cells and the endothelium exercise chemoattractant functions for the local endothelium and consequent repair of this process. However, the growth factors determine endothelial functions and data in the literature of how they protect the endothelium or promote CD are still insufficient[6].

Studies reported that high levels of PAI-1 in plasma are influenced by age, gender, obesity, hypertension, smoking, hypercholesterolemia, dietary patterns, and genetic polymorphisms, and suggested that the visceral adipose tissue, this marker is differentiated to provide more information about the fibrinolytic state[7,8]. The plasma fibrinogen has been identified as a cardiovascular risk factor, their concentrations have been associated with body mass index (BMI), waist circumference (WC), hip circumference (HC), fasting glucose, blood insulin, HDL -cholesterol, systolic and diastolic blood pressure[8,9]. Few studies have reported that higher plasma VEGF concentration in obese patients, and its relation to the distribution of fat remains unclear. However, their concentrations are associated with the increased BMI, progression of atherosclerosis, low HDL-c, hypertriglyceridemia, systolic pressure and hyperglycemia[10].

Current researches point the EGF and its inverse correlation with concentrations of fasting glucose, insulin and HOMA-IR index. Its concentration is also proportional to the increase in adiposity; however there is not relationship between EGF with BMI[11].

The modulation of the mechanisms involved in inflammation, by the diet, is highlighted by the protective effect to CD[2]. Several effects of interventions with fruits, rich in flavonoids, have been found to inflammatory biomarkers. It was observed a reduction in PAI-1, fibrinogen and VEGF in patients with overweight or comorbidities[12,13,14]. In healthy subjects, other studies point the reduction of EGF, PAI-1[13] and fibrinogen[15].

The beneficial effect of fruits and dietary pattern in the improvement of the inflammatory status is already well understood[13]. In fact, fruits increase the antioxidant capacity in plasma. Among the eleven most consumed fruits in Brazil, recent studies suggest the acai as the main responsible for the increase in the daily intake of phenolic compounds, flavonoids and total antioxidant capacity[13,16]. Mertens-Talcott and colleagues (2008) demonstrated that consumption of acai pulp tripled the plasma antioxidant capacity[16].

In view of its potential of nutritional utilization and due to its relevance in Brazilian dietary consumption, in this study, it was given importance to Acai berry due to its antioxidant properties and modulation of endothelial homeostasis. No studies in the literature presenting its action on markers of endothelial dysfunction[17].

Due to the paucity of studies in the literature, this study will allow us to verify by the first time in humans, the effect of acai on the endothelial injury markers and their relationship with anthropometric, body composition, dietetic and inflammatory parameters, since it was known that the obesity leads to endothelial dysfunction. So, the acai can modulate the inflammatory profile, protecting the endothelial damage and reducing the risk of cardiovascular disease through its antioxidant properties. In this view, the objective of this study was to investigate if the consumption of 200g acai pulp has an effect on inflammatory markers of fibrinolytic cascade (PAI-1 and fibrinogen) and growth factors (EGF, TGF-α and PDGF-AA and VEGF), and also, on the anthropometric, body composition, clinical and biochemical parameters in healthy women.

Methods

Study sample

This is a nutritional intervention, self-controlled involving women aged between 18 and 35 and BMI determined between 27 - 35 kg/m² for the group 2 (overweight and obesity class I and II) and 18.5 - 25 kg/m² for group 1.

It were excluded: women who could not read or write, who had more than 10% of body weight change before the beginning of the study, as well as, blood pressure > 130/85 mmHg, fasting glucose > 100 mg/dl, dyslipidemia history, total cholesterol > 200mg/dl or triglycerides > 150 mg/dl, allergies, eating disorders and/or acai intolerance. It were also excluded women who had special diets (vegetarian diet, Atkins diet, etc.), who were taking nutritional supplements (vitamin complex , minerals) or chronic medication (except contraceptives), women who were top athletes or had acute chronic diseases, infectious, inflammatory, as well as, pregnant or breastfeeding, smokers and were classified at the Three Factor Eating Questionnaire - TFEQ[18] which assess the restriction, disinhibition and hunger with low scores: 0-5; 0-9; 0-4; and high: ≥ 10; ≥ 13; ≥ 8. The omitted cases were discussed in group with the study coordinator (ACPV).

Experimental design

The study was conducted during 4 weeks and divided in 3 steps (Figure 1).

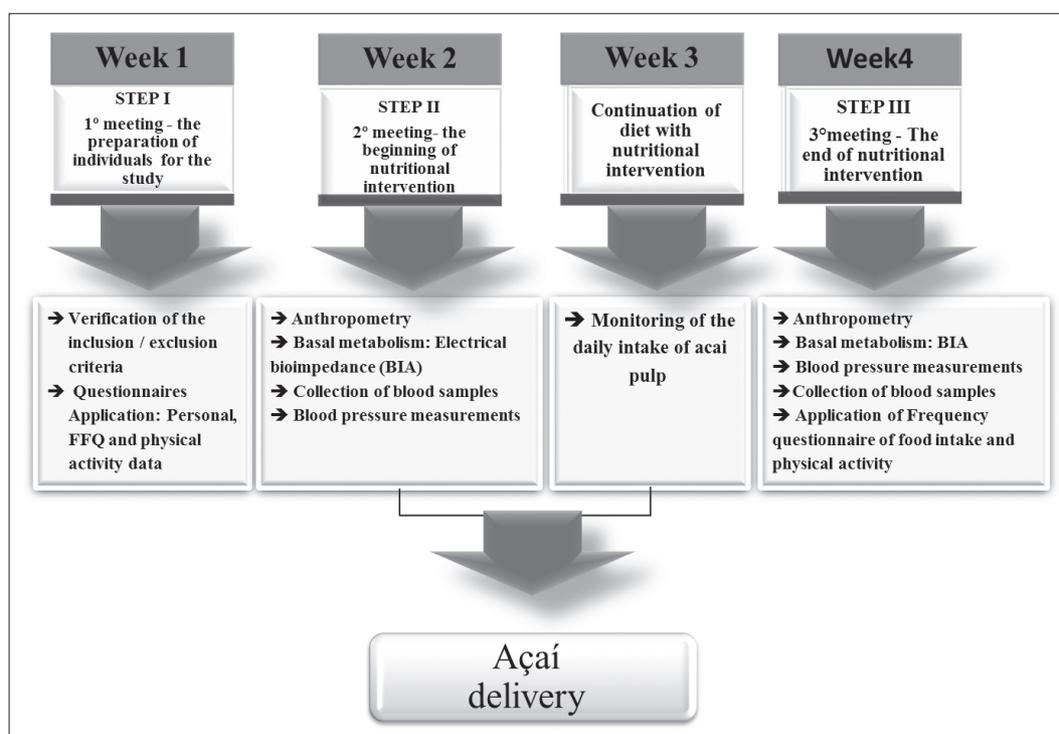| Week 1	Week 2	Week 3	Week4
STEP I 1° meeting - the preparation of individuals for the study	**STEP II** 2° meeting- the beginning of nutritional intervention	**Continuation of diet with nutritional intervention**	**STEP III** 3°meeting - The end of nutritional intervention
→ Verification of the inclusion / exclusion criteria → Questionnaires Application: Personal, FFQ and physical activity data	→ Anthropometry → Basal metabolism: Electrical bioimpedance (BIA) → Collection of blood samples → Blood pressure measurements	→ Monitoring of the daily intake of acai pulp	→ Anthropometry → Basal metabolism: BIA → Blood pressure measurements → Collection of blood samples → Application of Frequency questionnaire of food intake and physical activity

Açaí delivery

Fig. 1.—Study design.

Anthropometric and body composition parameters

The body weight was measured using a Welmy® digital scale (model W200-A); the height was evaluated by a vertical stadiometer coupled to the scale, with 2.00 m long, divided into centimeters and subdivided into millimeters (1 mm of precision)[19,20]. The BMI was calculated by means of weight and height in order to group the women into normal weight and overweight group. It were measured the waist circumference (WC), hip circumference (HC), abdominal circumference (AC) and upper arm circumference (UAC) with a flexible and inelastic metric tape, divided into centimeters and subdivided into millimeters (accuracy of 1 mm)[21,22].

The thickness of the subcutaneous tissue was analyzed by measuring the following skinfolds: triceps (TRI), biceps (BI), subscapular (SUB), supra-iliac (SUPR), and the sum of them was used to calculate the percentage of total body fat (%TBF)[23]. All skinfolds and circumferences were performed in triplicate and the mean of three measurements was considered.

The sum of skinfolds; the total upper arm area (TUA); upper arm muscle area corrected for women (UMAc); upper arm muscle circumference (UAMC); upper arm fat area (UFA); truncal fat percentage (%TF); were calculated using the circumference and skinfold measures. Body composition represented by body fat variables (kg), fat-free mass (kg), body fat percentage (%BF) and the resting energy expenditure (REE; Kcal) were determined by horizontal tetrapolar bioelectrical impedance (Biodynamics, model 310e)[22,23].

Clinical and biochemical parameters

The systolic and diastolic blood pressure were performed in triplicate following a 5 minute interval between measurements by means of a Omron® pressure apparatus (model HEM-705CP) positioned on the right arm of the women volunteer which were sitting at rest[24].

In order to quantify the physical activity value, it was used an activities of different intensities scale, in 24-hour period. After, they were converted into equivalent metabolic units (METs)[25].

The extraction of blood samples was performed after 12 hours of fasting by a trained pharmaceutical. One blood sample was extract of each volunteer: two serum tubes (5 mL each) and four tubes containing EDTA for plasma (4 mL). One of the 5 mL tubes and a serum sample of 1 mL of EDTA to plasma were sent for biochemical analysis (fasting glucose, insulin, total cholesterol and lipoproteins, triacylglycerols and total proteins). The fasting glucose, total cholesterol and high density lipoprotein cholesterol (HDL-c) were determined by enzymatic colorimetric method (Metrolab® spectrophotometer, model 2800). Low density lipoprotein cholesterol (LDL-c) concentrations were calculated according to the Friedewald et al. (1972) equation: LDL-c = total cholesterol - HDL-c - (Triacylglycerols/5), for samples which showed result of triglycerides < 400 mg/dL. Total proteins were determined by Biuret-colorimetric method and albumin by green- colorimetric of bromocreso (Metrolab® spectrophotometer, model 2800) with commercially available specific kits (Bioclin, Quibasa). The dosage of globulin was determined by subtracting the amount of to-
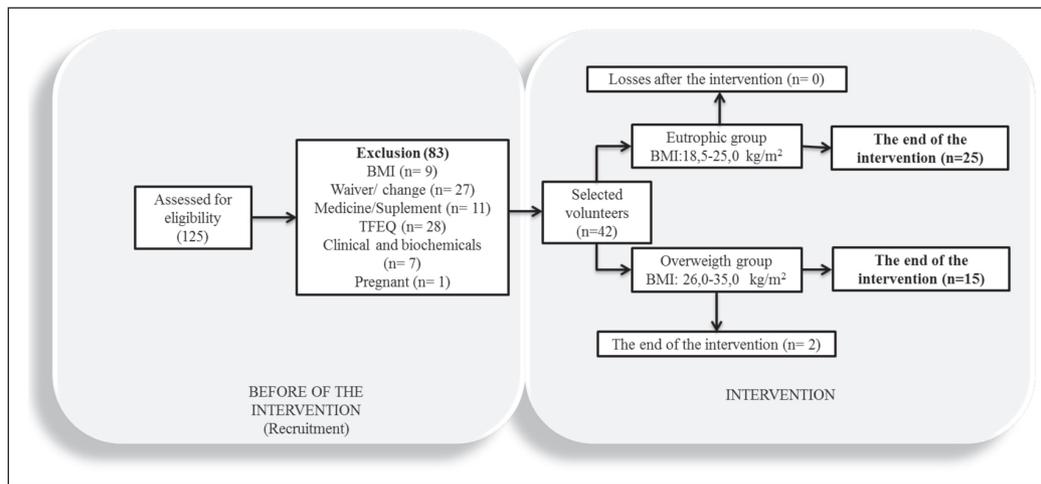
Fig. 2.—Selection scheme of volunteers.

tal protein and albumin - the albumin/globulin ratio. The dosage of total proteins was performed in order to monitoring the nutritional status of the volunteers. To dose fasting insulin, it was used the Access Ultrasensitive Insulin test (Acess® Immunoassay System), determined by chemiluminescence immunoassay. In chemiluminescent immunoassay, it was used the protocol provided by the manufacturer. The kit detection sensitivity was 0.3 μIU/mL and precision of $<10\%$ variation coefficient (VC). The results were expressed as μIU/mL.

To determine the insulin sensitivity, the HOMA-IR (model assessment of insulin sensitivity homeostasis) was determined by the following formula: HOMA-IR$=$[Insulin (μU/L) X glucose (mmol/L)/22.5] . To determine the functional capacity of the pancreatic beta cells, the HOMA beta formula was used: [20 X insulin (μU/L)/(glucose (mmol/L) -3.5)][26].

Serum concentrations of PAI-1, fibrinogen, VEGF, EGF, TGF-α and PDGF-AA were determined by Enzyme linked immunosorbent assay (ELISA), using MILLIPLEX® MAP specific commercial kits (Millipore Corporation, Billerica, MA, USA) with a sensitivity 2.8 pg/mL to EGF; 0.8 pg/mL to TGF-α; 0.4 pg/mL to PDGF-AA; 26.3 pg/mL to VEGF; 4.8 pg/mL to PAI-1 and 0,004ng/mL to fibrinogen. The PAI-1/body fat (kg) index were calculated through the PAI-1 marker and body fat (kg).

Dietary parameters

Calories, protein, carbohydrates, lipids, fatty acids: satured, monounsaturated, polyunsaturated, trans, omega-3, omega-6, omega-9, (omega-3/omega-6) ratio, total fiber (g) and cholesterol (mg) were calculated. The acai pulp has been added to the FFQ, adopting the nutritional composition of the label of acai pulp. Acai pulp (100g) contains: 70 calories, 3g of carbohydrates, 2 g of protein, 5 g of total fat, 1 g of saturated fat and 3g of dietary fiber.

To evaluate the diet quality, the amount of the food registered as household measures, in Food Frequency Questionnaire (FFQ), was converted in grams of food/day and, later, in portions to calculate the Original Diet Quality Index (DQI)[27]; the International Quality Diet Index (IDQ-I)[28]; the Healthy Eating Index (HEI)[29]; the alternative Mediterranean Diet Score (aMED Score)[30] and the Recommended Food Score (RFS)[31]. The dietary antioxidant capacity was also calculated by means of comparing tables containing values of Ferric Reducing Antioxidant Power (FRAP), the antioxidant content of each food present in the FFQ. The table information referred a portion of 100 g of food represented by two parameters to assess the antioxidant capacity: values of Ferric Reducing Antioxidant Power (FRAP). Through the portion of each food consumed by volunteers before and after acai consumption, were obtained as the proportion of the FRAP values representing its antioxidant content.

Data analysis

The data were presented as mean±standard deviation for those variables that fit in a normal distribution, or median and interquartile range for the variables that did not fit in a normal distribution. We used the Kolmogorof-Smirnov normality test to check these settings. In addition, the Student t test and Wilcoxon, both paired, were performed to evaluate the intervention effect by BMI. Comparisons between the baseline mean and median of groups were made by Student t-test (parametric) and U-Mann-Whitney (non-parametric). For all statistical tests, we adopted a significance level of 5%. Statistical analysis were performed using SPSS version 17.0 Statistics software.

Results and discussion

The average age of the eutrophic group was 23.58±0.66 and of overweight group was 24.27±0.98. In the basal state, the anthropometric variables in the overweight group were increased compared to the eu-

Table I

Anthropometric and body composition characteristics before and after the consumption of acai pulp

	TOTAL (n=40)				EUTROPHICS (n=24)				OVERWEIGT (n=15)				
	T^0	T^1	Δ	p^a	T^0	T^1	Δ	p^a	T^0	T^1	Δ	p^a	p^b
Body weigt kg	61.55 (55.12-77.00)	61.45 (56.00-77.70)	-0.10	0.076	57.86±5.54	58.26±5.50	0.40	**0.031**	80.32±11.53	80.40±11.12	0.08	0.866	**<0.001**
BMI, kg/m²	22.71 (20.91-27.43)	23.17 (21.07-27.51)	0.46	0.065	21.52±1.56	21.68±1.59	0.16	**0.028**	29.36±2.75	29.38±2.70	0.02	0.890	**<0.001**
Upper arm circumference, cm	28.33 (26.53-31.90)	28.33 (26.17-31.75)	0	**0.025**	27.00±1.56	26.80±1.95	-0.20	0.157	32.50±2.27	32.03±2.29	-0.47	0.136	**<0.001**
Waist circumference, cm	73.00 (68.85-81.45)	72.50 (68.25-79.53)	-0.50	0.147	69,87±3,30	69,78±1,95	-0,09	0.769	84.81±6.96	84.10±6.88	-0.71	0.129	**<0.001**
Abdominal circumference, cm	84.99±10.09	84.02±9.76	-0.97	0.045	70.07±5.27	78.2±4.89	8.13	0.130	90.80 (88.53-104.00)	91.76 (88.00-95.66)	0.96	0.256	**<0.001**
Hip circumference, cm	98.70 (94.00-110.45)	100.00 (95.65-109.09)	1.30	0.767	97.24±5.29	97.36±4.38	0.12	0.870	113.33±8.86	113.25±8.29	-0.08	0.884	**<0.001**
Triceps skinfold, mm	22.50±6.25	21.49±5.73	-1.01	**0.013**	19.52±4.22	18.80±4.14	-0.72	0.181	27.47±6.01	25.98±5.24	-1.49	**0.018**	**<0.001**
Biceps skinfold, mm	11.44 (8.44-16.78)	10.70 (8.40-13.89)	-0.74	**0.005**	10.1 (7.56-11.70)	9,06 (7.65-11.21)	-1.04	0.253	17.20±4.63	16.21±5.56	-0.99	0.045	**<0.001**
Subscapular skinfold, mm	19.99±7.18	19.89±6.62	-0.10	0.823	16.15±4.41	16.28±4.51	0.13	0.812	26.39±6.33	25.92±5.00	-0.46	0.553	**<0.001**
Supra-iliac skinfold, mm	22.34±7.97	22.52±6.90	0.18	0.694	17.70±4.85	18.86±5.42	1.16	**0.046**	30.08±5.83	28.69±4.123	-1.39	0.054	**<0.001**
Skinfolds, mm	77.78±23.09	75.99±20.98	-1.79	0.103	63.76±12.76	63.49±13.13	-0.27	0.846	101.15±16.42	96.82±13.71	-4.33	**0.018**	**<0.001**

Izabelle de Sousa Pereira et al.

Table I (cont.)
Anthropometric and body composition characteristics before and after the consumption of acai pulp

	TOTAL (n=40)				EUTROPHICS (n=24)				OVERWEIGT (n=15)				
	T^0	T^1	Δ	p^a	T^0	T^1	Δ	p^a	T^0	T^1	Δ	p^a	p^b
Upper arm muscle	20.78±23.09	20.99±20.98	0.21	0.917	20.86±1.34	20.89±1.38	0.03	0.866	23.87±1.73	23.86±1.71	-0.01	0.978	**<0.001**
Upper arm total area, cm^2	63.91 (56.02-81.02)	63.88 (54.52-80.21)	-0.03	**0.024**	57.58 (54.34-60.76)	55.88 (52.08-63.88)	-1.70	0.072	84.47±11.91	82.03 ±12.03	-2.44	0.153	**<0.001**
Corrected upper arm muscle area, cm^2	32.33± 7.46	32.39± 7.48	0.06	0.919	28.28±4.36	28.39±4.57	0.11	0.857	39.08 ±6.66	39.04 ±6.64	-0.04	0.974	**<0.001**
Upper arm fat area, cm^2	35.75± 10.56	34.28± 9.69	-1.47	**0.008**	29.97±5.91	29.07±5.85	-0.90	0.145	45.38± 9.57	42.98 ±8.56	-2.40	**0.023**	**<0.001**
Truncal fat, %	54.80 (51.65-58.60)	57.34 (50.89-59.32)	2.54	**0.001**	53.02 (50.0-58.2)	55.34 (50.78-59.19)	2.32	**0.003**	55.59 (53.04-59.106)	57.44 (54.69-59.410)	1.85	0.191	0.119
Body fat (formula*)%	34.15±0.70	33.94±0.67	-0.21	0.371	31.54±3.17	31.58±3.31	0.04	0.911	38.49 ±2.47	37.88 ±2.17	-0.61	**0.016**	**<0.001**
Body fat (BIA), kg	18.60 (15.62-26.17)	19.30 (16.32-25.17)	0.70	0.190	16.73±3.17	16.70±2.99	-0.03	0.029	30.20 (24.80-35.40)	28.50 (24.20-35.50)	-1.70	0.589	**<0.001**
Fat-free mass (BIA), kg	44.67±6.96	44.61±6.78	-0.06	0.687	39.90 (38.45-44.05)	40.00 (38.90-43.65)	0.10	0.951	50.68 ±6.98	50.50 ±6.80	-0.18	0.474	**<0.001**
Body fat (BIA), %	31.79±5.26	32.17±5.12	0.38	0.105	28.82±3.62	29.38±3.24	0.56	0.092	36.74±3.568	36.82±4.23	0.08	0.804	**<0.001**
REE, kcal	1331.50 (1180.75-1518.75)	1323.00 (1202.75-1508.25)	-8.0	0.706	1212,00 (1169,50-1342.00)	1217,00 (1183.00-1327.00)	5.0	0.951	1542.33 ±210.44	1535.60 ±206.32	-6.73	0.389	**<0.001**

T^0 : Baseline characteristics (before the intervention). T^1 : Final characteristics (after the intervention). Δ: Delta, difference between groups. T^1 : Final characteristics (after the intervention). Δ: Delta, difference between final and initial period (Δ= T^1 –T^0). REE: Resting Energy expenditure.
Presented data: mean± standard deviation or median (Q1-Q3) as parametria. Normality test: Kolmogorov- Smirnov, p<0.05.
*Body Fat as calculated from sum of 4 skinfolds (tríceps, bíceps, supra-iliac and subscapular).
*ª Paired Student t Test or Paired Wilcoxon Test.
*ᵇ Student t Test or U-Mann- Whitney, for comparison between groups before the acai intake.

trophic group. It was expected, since BMI quantifies the level of body fat considering the body weight of the individual. However, the truncal fat was indifferent between the groups, which could be expected, because BMI is related to fat-free mass, which impede the distinction of the local where the fat is located, either the muscle amount of the individual. So eutrophic individuals can present similar or greater change in truncal fat than overweight individuals[19,33].

The anthropometric assessment enabled the detection of changes in nutritional status and body composition mediated by acai consumption. In all volunteers there was a reduction of AC,TRI, BI, TUA, UFA and a considerable increase in truncal fat, however, without changing in food pattern, REE or METs. These results suggest a redefinition of body fat in all volunteers with accumulation in the truncal region and decrease in peripheral areas of the upper limbs, with a possible reduction of subcutaneous fat and considerable increase in visceral fat. In this study there was no measurement of body fat for invasive methods of dual energy absorption of the conventional X-ray (DEXA) which would allow better evaluation of these parameters. However, it is known that truncal fat reflects an accumulation of fat in the intra-abdominal or visceral cavity, including subcutaneous routes and intermuscular fat throughout the fat deposits region of the trunk, in addition to epicardial and pelvic deposits[34].

In eutrophic group, the acai consumption focused on weight gain, BMI, supra-iliac skinfold, truncal fat percentage and reduction in body fat (kg). These results possibly show a fat redistribution with increased visceral subcutaneous or intramyocellular due their high relationship with truncal fat alteration[35,36]. The fat redistribution is directly related to inflammatory markers concentration. Raji et al. (2001) showed that individuals with greater accumulation of abdominal fat had greater relationship between PAI-1 concentrations and deposition of abdominal fat: abdominal (r=0.7), visceral (r=0.62), subcutaneous (0, 46) (p<0.01)[37].

In the overweight group, there was a significant reduction in triceps skinfold, biceps skinfold, sum of skinfolds, upper arm fat area and total body fat. The changes of anthropometric measurements and skinfolds represented a reduction in subcutaneous fat. Nevertheless, visceral adipose tissue was not sensitive to modification. Moreover, the reduction in skinfold measures and the upper arm fat area are closely related to the amount of adipose tissue in the body, thus the decrease of these anthropometric parameters presumably is directly related to the reduction of total fat percentage[36,38]. We evaluated the muscle mass of the volunteers by means of the adequacy of UAC and TRI measures. Eutrophic and overweight volunteers were classified as obese. However, after adjustment of the UAMC, the two groups were classified as normal. These results demonstrate that the muscle mass was preserved in both groups.

The biochemical and clinical data, before the intervention with acai, were within the normal range. These results were expected, because this normality was interposed the inclusion criteria of the study. However for those with overweight it was observed a higher HOMA-IR, SBP and DBP when compared to eutrophic group. The decrease in blood pressure is, possibly, correlated with a decrease in body fat and consequent reduction in the risk of developing cardiovascular disease. Lavie et al (2003) suggested that the high percentage of body fat, estimated by skinfold measurements, can be an independent predictor of cardiovascular death by increased pressure[39].

As noted, the overweight group presented reduction of fat percentage, skinfolds and remained with a lipid, glicidic and insulin homeostasis even if at baseline had higher HOMA-IR, and the maintenance has been sustained after acai consumption. This homeostasis preservation was due to the good overall clinical status of the participants and, possibly, by the positive impact of acai pulp consumption, since the volunteers kept their eating pattern, the total antioxidant capacity of the diet and level of physical activity.

Udani et al. (2011) in its pilot study, performed with overweight individuals in homeostasis, who consumed, for 4 weeks, 200g of acai pulp, it was observed significant reduction in total cholesterol, fasting glucose and insulin, in addition to the blood pressure maintenance, however such volunteers received a brochure with orientation to avoid foods that contained nitrates (eg. bacon and hot dogs), which could have contributed to these results[40].

Other intervention works with fruits rich in anthocyanins reinforce our results. Basu et al (2010) showed that the use of blueberry reduced the systolic and diastolic blood pressure by about 6% and 4%, respectively, in individuals with metabolic syndrome[41]. In healthy human or with any cardiovascular risk factor there was a reduction in systolic and diastolic blood pressure after supplementation with blueberries[42]. Another study conducted with acai extract (injections of 10 to 100 mg extract) in rats showed that acai had a endothelium dependent vasodilator effect due to the effects on the nitric oxide production[17].

We found that the total protein - an important marker of nutritional status - were reduced after the intervention with acai, as verified for the total and eutrophic group, and the reduction of globulin in greater magnitude caused the increase of the albumin/globulin ratio, but the values remained within the reference values and without affecting the muscle reserves. So, all the volunteers kept their nutritional condition. At the same time, albumin is an indication of the diet protein content, and it refers to inflammatory processes, therefore there is a reverse correlation between the concentrations of albumin and globulin as a compensatory mechanism, in order to maintain the total protein level and blood oncotic pressure[43].

In relation to eutrophic voluntary, the isolated increase in serum albumin supposedly correlates with its carrier function of free fatty acids. The increase in trun-
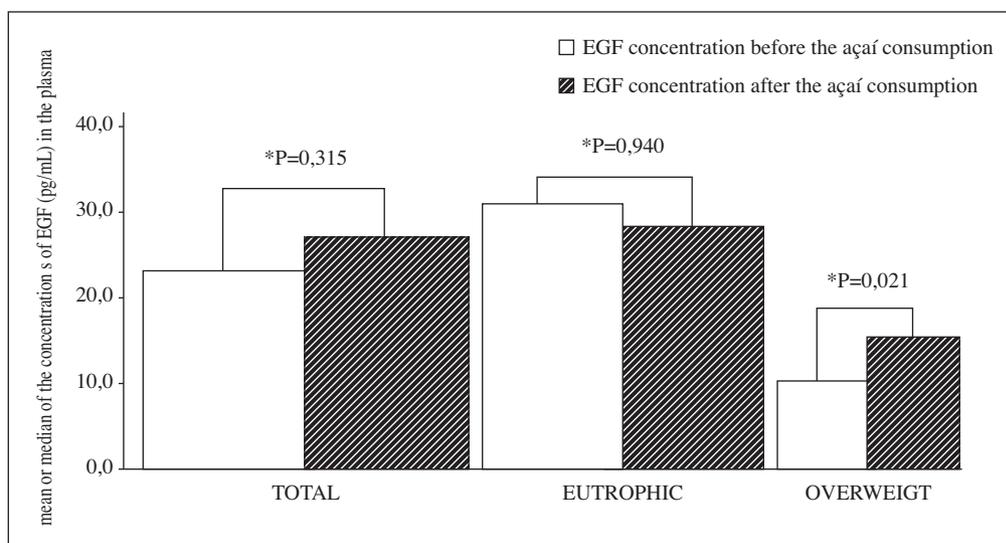
*Fig. 3.—Representation of the concentration of EGF marker in plasma. Total (n=30); eutrophic (n=21) and overweight (n=9). Teste t-Student (mean) or *Wilcoxon paired (median), (p<0,05).*

cal fat percentage of this group is directly related to the visceral fat increase, and the literature addresses the visceral adipose tissue (VAT), the most active, sensitive to lipolysis and more resistant to insulin action and allows a higher concentration of free fatty acids (FFA), which are carried by albumin directly to peripheral tissues for storage or as an energy source. Thus a possible likely explanation for this isolated increase can be related to its transport function[35,36].

In view of the inflammatory markers production PAI-1 and fibrinogen and growth factors (EGF, TGF-α, PD-GF-AA and VEGF) dependent of the oxidative stress increase and endothelial inflammation mainly, the inflammatory profile before the acai consumption was not different between eutrophic and overweight volunteers.

The effect of the intervention (consumption of 200g of acai) in free-living context, during four weeks, showed just alteration on the PAI-1 increase in all volunteers and EGF and PAI-1 alteration in the overweight group. The isolated increase of PAI-1 can be inter-related to PAI-1 changes in the overweight group, concomitant with this fact, another supposed explanation is related to the increase in truncal fat evidenced by the total group. Studies indicated that an increase a truncal fat is associated with PAI-1 increase. Together with the truncal fat increase, there is an increase mobilization of non-esterified fatty acids which stimulates the adipocyte secretion of PAI-1[44].

Likewise, the PAI-1/BF ratio, in all volunteers (0.039) and in the overweight group (0.019) supports the hypothesis of PAI-1isolated increased, since its increase was independent of body fat in total or overweight group[44]. The PAI-1 change can be influenced by several factors of anthropometry, clinical, biochemical
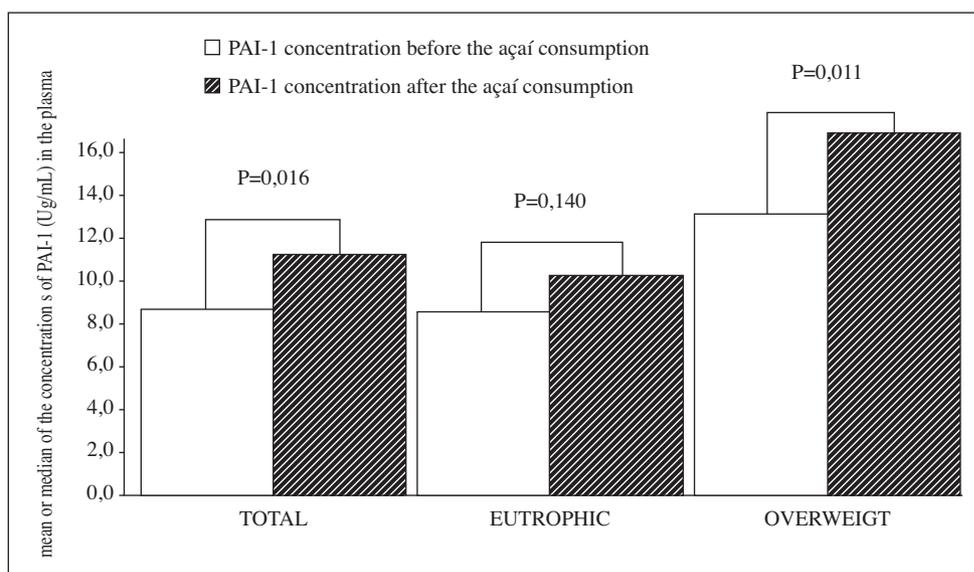

*Fig. 4.—Representation of the concentration of PAI-1 marker in plasma. Total (n=37); eutrophic (n=23) and overweight (n=14). Teste t-Student (mean) or *Wilcoxon paired (median), (p<0,05).*

Table II
Biochemical and clinical characteristics before and after the consumption of acai pulp

	TOTAL (n=40)				EUTROPHIC (n=25)				OVERWEIGT (n=15)				
	T^0	T^1	Δ	Pa	T^0	T^1	Δ	Pa	T^0	T^1	Δ	p^a	p^b
Glucose, mg/dl	77.50 (75.00-83.50)	78.00 (75.00-84.00)	0.50	0.771	79.28±6.44	78.08±5.04	-1.20	0.250	77.00 (75.00-80.00)	83.00 (76.00-90.00)	6.00	0.421	0.911
Insulin**	5.68 (4.40-7.56)	6.17 (4.05-8.02)	0.49	0.868	5.34 (4.40-6.40)	6.00 (4.05-8.49)	0.66	0.737	7.12±2.45	6.67±3.16	-0.45	0.613	0.144
HOMA-IR**	1.23 (0.88-1.61)	1.15 (0.73-1.64)	-0.08	0.412	1.05 (0.87-1.28)	1.17 (0.77-1.64)	0.12	0.798	1.48 ±0.56	1.25 ±0.65	-0.23	0.230	*0.044*
HOMA-β**	133.19 (108.33-189.38)	125,12 (86.31-189.80)	-8.07	0.989	128.00 (102.27-169.13)	146.80 (97.71-206.36)	18.80	0.088	157.68 (115.78-199.80)	94.75 (73.05-161.15)	-62.93	0.125	0.214
QUICK	0.37 (0.35-0.39)	0.37 (0.35-0.40)	0	0.085	0.38 (0.36-0.39)	0.37 (0.35-0.41)	-0.01	0.427	0.36 ±0.02	0.38 ±0.03	0.02	0.063	0.234
Total proteins, g/dl	7.05 (6.62-7.67)	7.00 (6.25-7.30)	-0.05	*0.048*	7.27±0.55	7.06±0.46	-0.21	*0.049*	6.96 ±0.58	6.72 ±0.80	-0.24	0.295	0.101
Albumin, g /dl	3.75±0.30	3.84±0.45	0.09	0.263	3.73±0.29	3.89±0.32	0.16	*0.008*	3.78 ±0.31	3.75 ±0.61	-0.03	0.888	0.660
Globulin, g/dl	3.60 (2.82-3.80)	3.15 (2.42-3.70)	-0.45	*0.007*	3.54±0.57	3.17±0.65	-0.37	*0.005*	3.40 (2.40-3.70)	2.90 (2.30-3.60)	-0.50	0.244	0.130
Albumin/globulin ratio	1.03 (0.94-1.37)	1.20 (0.90-1.59)	0.17	*0.006*	1.08±0.23	1.29±0.37	0.21	*0.001*	1.08 (0.97-1.62)	1.14 (0.94-1.81)	0.06	0.349	0.150
Cholesterol, mg/dl	188.46 ±34.00	188.27 ±38.29	-0.19	0.964	184.56±29.05	184.72±32.99	0.16	0.977	194.96 ±41.26	194.20 ±46.45	-0.76	0.897	0.356
Triaciglicerol, mg/dl	79.94±35.85	81.32±35.44	1.38	0.760	70.00 (56.00-84.00)	71.00 (50.50-88.00)	1.00	0.737	89.06 ±41.94	95.40 ±43.57	6.34	0.512	0.252
LDL, mg/dl	106.79±30.59	105.64±33.94	-1.15	0.753	102.30±27.82	101.12±31.13	-1.18	0.810	114.26 ±34.40	113.18 ±38.08	-1.08	0.845	0.236
HDL, mg/dl	65.00 (55.22-77.50)	66.00 (56.00-76.00)	1.00	0.723	67.20±14.41	69.12±14.41	1.92	0.232	63.06±12.03	61.66±12.38	-1.40	0.604	0.357
Systolic blood pressure, mmHg	106.16 (95.58-115.00)	103.99 (93.41-112.91)	-2.17	0.083	100.93±9.41	100.59±10.54	-0.34	0.856	118.00 (107.00-124.00)	107.66 (97.33-118.33)	-10.34	*0.013*	*<0.001*
Diastolic blood pressure, mmHg	74.16 (64.41-80.16)	71.16 (64.41-78.50)	-3.00	0.202	70.33 (63.16-77.00)	67.00 (62.67-76.33)	-3.33	0.786	76.66 (73.30-88.00)	75.00 (68.67-81.00)	-1.66	0.096	*0.009*
Metabolic equivalent units/ day	39.35 (34.86-48.35)	38.57 (35.03-45.45)	-0.78	0.886	39.50 (34.15-46.25)	38.00 (35.13-45.30)	-1.50	0.904	39.20 (34.75-48.40)	40.27 (33.62-47.05)	1.07	0.875	0.342

T^0 : Baseline characteristics (before the intervention). T^1 : Final characteristics (after the intervention). Δ: Delta, difference between final and initial period (Δ=T^1 –T^0).
HOMA-IR: Homeostatic model assessment of insulin resistance; HOMA-beta INDEX; QUICKI: Quantitative insulin sensitivity check index; LDLc: LDL Cholesterol; HDLc: HDL Cholesterol.
Presented data: mean± standard deviation or median (Q1-Q3) as parametria. Normality test: Kolmogorov- Smirnov. p<0.05.
*a Paired Student t Test or Paired Wilcoxon Test. *b Student t Test or U-Mann- Whitney, for comparison between groups before the acai intake. ** n= 37: 26 eutrophic and 11 overweight.

or even inflammatory changes, which acted jointly or individually. Among them, the body composition is a very sensitive determinant to the inflammatory markers changes.

Therefore, another possible modulation of PAI-1 could be justified by an increase in baseline HOMA-IR in overweight group associated with an increase insulin resistance; however, they are lower concentrations to supposed values for developing diabetes type 2. As reported in the literature, in states of bigger insulin resistance and inflammation, the plasma membrane of adipocytes express EGF receptors, which by means of intracellular signaling increase the binding of EGF binding. These phosphorylated sites give the activation of different proteins, such as: Grb2, Shc, Src family tyrosine kinases, PI3 kinase, and phospholipase. Even as the insulin, some studies indicate that its impaired signaling, in cells with increased resistance to insulin, EGF increases the tyrosine phosphorylation and activation of the IRS-1 and IRS-2, which enhances translocation induced by EGF and GLUT[4] in the plasma membranes and possible stimulation of glucose uptake in target tissues[45]. At the same time, the increase of EGF is likely to change that mediates the PAI-1 increase in the overweight group. By means of the binding of EGF to its receptor (EGFR) with high affinity in cell surface, triggering the dimerization and phosphorylation each other through their tyrosine kinase activity. Phosphorylated receptors can initiate signal transduction, including MAPK, Akt and JNK,AP-1[45]. Thus, in the vasculature, the AP-1 transduction, mediates not only the cell growth, but also the gene expression of prothrombotic factor, pro-fibrotic, PAI-1 in response to various stimuli[3]. Therefore, under certain concentrations, the growth factor can be acting on behalf of the process by improving the effectiveness of insulin, at the same time, interceding in gene production of PAI-1 by activating the same signaling pathways that lead to their expression. However, the literature that addresses these mechanisms does not report the magnitude of change of the growth factor concentrations, which leading to activation of this process. All these tests were conducted only *in vitro*.

Thus, the acai effect was possibly performed by the action of its cellular protection properties. The acai is rich in anthocyanins which are a class of flavonoids derived from polyphenols and according to Noratto et al. (2011) the polyphenols action on the cellular protection is indirect, by act on the inhibition of protein kinase C (PKC) and Mitogen activated protein kinase (MAPK)[46]. The inhibition of these enzymes reduces the binding ability of transcription factors, such as: NF-kB or activator protein-1 (AP-1) to DNA and PAI-1 expression rate is controlled. Thereby, the acai consumption in this study, supposedly held its attenuation effect, controlling the increase in gene expression of PAI-1 to lower concentrations that could lead to their pro-thrombotic action. However, we know that studies that confirm the PAI-1 increase relates them to the formation of thrombi in endothelial wall, also increasing the risk of cardiovascular events and due to the relatively low increase and very below of the reference limits presented in the literature (5.0 to 40 ug/ml), was not considered malefic. It was just a consequence of the EGF increase that by means of cellular mechanisms may have effected an improvement in glucose uptake in the overweight voluntary[46,47,48]. So far, it was not found in the literature intervention studies with acai in humans, in free-living situation, which assess the effect of this fruit on PAI-1, EGF, neither, on our other evaluated markers. The studies which approach this subject are often performed in vitro or with other types of fruit.

In contradiction to our studies, in an intervention study with cherry, oriented individuals for limiting other fruits rich in polyphenols (tea, wine, *berries*) besides the cherry itself (except those supplied by the study) during 28 days, led to EGF and PAI-1 reduction. In this study, these decreases were related to the cardiovascular disease, cancer and inflammation reduction. However, in the weeks after the cherry consumption, there was a minimal increase of PAI-1, and the authors attributed this change to the residual effects left after the cherry consumption, which attenuated its increase. In this study, several other markers reduction in the intervening period, did not demonstrate the possibility of EGF to modulate PAI-1, since in inflammation, a number of mediators are associated in cytokines cascade promoting or reducing their secretions[13].

In other intervention study with following diets: low-fat, high-fat diet and high fat supplemented with 20% of lingonberry, blackcurrant, blueberry, raspberry, acai, cranberry, plum or blackberry, for 13 weeks, there was an increase in PAI-1 concentration, in rats that received acai compared to other groups who received other fruits. So, it must evaluate and consider the mechanisms underlying of these effects, in view of the optimal dose quantity, administration time and even the reproducibility in humans[49].

It is not possible to evaluate if the diet or some specific compound of acai exerts direct action on the markers changes or if the changes caused by diet on the anthropometric, biochemical, clinical and inflammatory variables contributed to change these markers. However this cause-effect study, has been the recognition that dietary characteristics may have a decisive influence on the inflammatory status and the dietary patterns, that assess the quality of diet, may be associated with deficiencies or to chronic diseases.

In relation to the total dietary antioxidant capacity, there was no change after acai consumption, because, possibly before the acai consumption, the volunteers consumed foods (eg, fruits, juices and vegetables) rich in other antioxidants as beta-carotene, lycopene, retinol, ascorbic acid, tocopherols, zinc, selenium and copper among others, replacing of the antioxidant effect of acai anthocyanins.

Table III

Diet characteristics before and after the consumption of açaí pulp

	TOTAL (n=40)				EUTRÓPHIC (n=25)				OVERWEIGT (n=15)				
	T⁰	T¹	Δ	p[a]	T⁰	T¹	Δ	p[a]	T⁰	T¹	Δ	p[a]	p[b]
Kcal	1980.43 (1673.67-2357.78)	1783.78 (1535.75-2606.98)	-196.64	0.989	1881.62 (1680.34-2395.10)	1729.65 (1503.66-2354.96)	-151.97	0.657	2040.60 (1470.90-2234.61)	2062.53 (1585.08-3324.53)	21.93	0.334	0.834
Protein, g	83.73 (67.22-114.06)	80.36 (68.92-110.97)	-3.36	0.468	83.43 (69.96-111.06)	76.18 (67.85-96.92)	-7.25	0.276	94.73±34.20	108.61±48.32	13.87	0.380	0.944
Lipids, g	65.62 (48.35-81.71)	62.01 (47.23-81.35)	-3.61	1.000	68.69±22.37	63.85±19.97	-4.84	0.236	67.40±28.55	79.75±49.32	12.35	0.297	0.874
Cholesterol, mg	270.18 (201.30-416.30)	268.14 (196.39-394.05)	-2.03	0.648	265.38 (202.37-410.86)	243.09 (192.09-301.38)	-22.29	0.158	328.61±167.94	396.77±228.47	68.15	0.408	0.542
Carboydrate, g	340.60 (235.35-564.30)	362.12 (258.93-689.49)	21.52	0.554	374.00 (264.24-589.77)	341.18 (237.63-558.44)	-32.82	0.253	291.42 (231.48-346.48)	517.23 (288.95-937.79)	225.81	*0.036*	0.548
Omega-6	11.08 (7.36-14.22)	10.55 (6.87-14.38)	-0.52	0.635	11.39 (7.52-13.44)	11.29 (7.41-13.48)	-0.10	0.946	11.81±5.76	10.72±5.18	-1.09	0.368	0.791
Omega-3	1.186 (0.77-1.47)	1.10 (0.74-1.31)	-0.08	0.219	1.17 (0.78-1.37)	1.12 (0.76-1.34)	9.95	0.778	1.18±0.48	1.04±0.33	-0.14	0.206	0.727
w-6/w3 ratio	9.69 (9.09-10.51)	9.64 (8.67-10.65)	-0.05	0.933	9.62 (9.18-10.49)	9.67 (8.96-10.56)	0.05	0.345	9.67±1.77	9.95±2.33	0.28	0.386	0.461
RFS	10.00 (6.25-10.75)	9.00 (6.00-10.75)	-1.00	0.118	8.68±2.32	8.24±2.50	-0.44	0.177	10.00 (6.00-11.00)	9.00 (5.00-11.00)	-1.00	0.301	0.600
aMED score	4.50 (4.00-5.00)	5.00 (4.00-5.00)	0.50	0.839	3.00 (2.00-3.50)	2.00 (1.00-3.50)	-1.00	0.163	5.00 (4.00-5.00)	4.00 (3.00-5.00)	-1.00	0.793	0.503
HEI	3.00 (2.00-4.00)	2.00 (2.00-4.00)	-1.00	0.594	74.10±10.55	74.58±7.62	0.48	0.803	3.00 (3.00-4.00)	2.00 (2.00-4.00)	-1.00	0.681	0.539
DQI	75.37 (69.318-81.79)	78.74 (70.25-81.92)	3.37	0.519	6.68±2.26	6.52±1.80	-0.16	0.701	77.69 (69.71-82.18)	81.33 (78.30-83.66)	5.31	0.233	0.525
DQI-I	55.00 (45.00-60.00)	55.00 (45.00-63.75)	0.00	0.331	59.72±8.30	59.28±10.72	-0.44	0.835	57.33±9.23	61.33±11.56	4.00	0.344	0.312
Total oxidant capacity of diet (mmol/100g)	7,72 (5,523-10,84)	7,76 (3,72-9,68)	0,04	0,204	7,73 (4,94-12,25)	7,76 (3,55-10,30)	0,03	0,137	7,28,55±3,39	7,26±3,52	-0,02	0,374	0,986

T⁰: Baseline characteristics (before the intervention). T¹: Final characteristics (after the intervention). Δ: Delta, difference between final and initial period (Δ=T¹ –T⁰). KCAL: Total calories of food frequency questionnaire; w6/w3 ratio: omega 6/omega 3;RFS: Recommended food score; HEI: Healthy Eating Index; aMED score: Score Alternative Mediterranean Diet Score; DQI :Original Diet Quality index; DQI-I: International Quality Diet Index.

Presented data: mean±standard deviation or median (Q1-Q3) as parametria. Normality test: Kolmogorov- Smirnov, p<0.05.

*[a] Paired Student t Test or Paired Wilcoxon Test . *[b] Student t Test or U-Mann- Whitney, for comparison between groups before the açaí intake

Furthermore, it was shown that overweight volunteers at baseline had a similar pattern food of eutrophic, despite differences in body composition. The evaluation of the diet quality, by dietary indexes, before the acai consumption, showed that all volunteers had a similar dietary pattern. In addition, the scores of HEI, RFC, aMED, IDQ, DQI-I demonstrate the necessity of improving the dietary patterns for both groups. Followed by the stratification by group, after the acai consumption, there was an increase only for the carbohydrates consumption in the overweight group without change the food pattern.

This interventional study about the beneficial effects of acai in humans is the greatest executed so far. So, our results provide interesting insights underlying the acai intake consequence on the inflammatory status, food intake, body composition and hormonal apparently healthy women. Our work did not include a control of acai intake, first because we understand that there is no placebo for a fruit, consumed in a free-life context and second because the volunteers were controls of themselves in the intervention, which allowed an intra-subject statistical analysis, enabling for the accuracy of the results. As much as the monitoring of acai intake was accomplished by means of food records before, during and after the consumption, the intake of acai pulp, in a free-living context, was not monitored, since the project does not aimed to interfere in the food standard in order to not induce any result.

In this cause-effect study, we cannot affirm that the variables changes were due to a direct or indirect effect of the acai pulp intake, it is important to emphasize that the clinical experiment was carefully designed, especially for the precision in the duration of the intervention and the amount of acai pulp offered to the volunteers. Thus, we consider this work relevant, since it will provide the basis for further studies.

Conclusion

After the evaluation of inflammatory biomarkers, food intake and body composition in eutrophic and overweight women who consumed acai pulp during four weeks, it was observed an increase in the pro-thrombotic marker PAI-1 and in the growth factor EGF. These biomarkers are considered risk factors to different diseases and are affected by oxidative stress and inflammation, however, our study showed a possible modulation of them on anthropometric, biochemical, inflammatory and dietary parameters by means of the beneficial action of acai pulp intake. It improved the insulin sensitivity and reduced the PAI-1 on in overweight women. In eutrophic group, it was observed a body fat redistribution, mainly to truncal region with possible increase in intramuscular and visceral fat, besides a subcutaneous body fat reduction. However in the overweight group, there was a reduction in body fat, and the blood pressure, consequently, decreased.

Our findings have shown that dietary pattern may reflect the modulation of inflammation or a set of anthropometric, biochemical, clinical and inflammatory changes inducing an imbalance of inflammatory mediators which act by inhibiting or promoting DC.

Competing interests

The authors declare they have no competing interests.

Author contributions

ISP: conducted field work, data collection, laboratory processing of samples, statistical analysis, wrote and edited the manuscript; TCMCMP, RALV, GAFF: conducted field work, data collection, laboratory processing of samples and statistical analysis; FLPO: guided the statistical analysis and edited the manuscript; FCS, JFA: guided the field work and laboratory processing as well as edited the manuscript; RNF: project co-lider, guided the field work and laboratory processing as well as edited the manuscript; ACPV: project leader and general coordinator, performed the financial management, designed the study, guided the field work and laboratory processing, besides edited the manuscript. All authors read and approved the final manuscript.

Acknowledgments

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