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Original / *Obesidad*

Short term low-calorie diet improves insulin sensitivity and metabolic parameters in obese women

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Abstract

Obesity and insulin resistance are associated with an increase of cardiovascular risk factors, including adipocytokines. The aim of this study was to investigate the effect of low-calorie diet on serum lipids, adipokines, insulin resistance and body composition in obese women. It was a clinical trial with class I obese women aged 30-45 years submitted to hypocaloric diet for 90 days. Dietary intake, anthropometric parameters, body composition, serum lipids, glucose, insulin, leptin, adiponectin, HOMA-IR and QUICKI indexes were evaluated at the baseline, 30, 60 and 90 days. There was 30% significant decrease in energy intake, and also decrease in body weight, body mass index and waist circumference ($p < 0.01$) throughout the treatment period. Despite the amount of lean body mass (kg) reduced in average, it was observed that lean body mass (%) had increased ($p < 0.01$) and that the amount of fat body mass (kg) had decreased significantly in the third month ($p < 0.05$). Systolic blood pressure reduced up to -5mmHg ($p < 0.05$) after 90 days. Was observed a decrease ($p < 0.05$) on serum insulin and HOMA-IR until the 60th day, while the serum adiponectin increased ($p < 0.01$) during treatment. Corroborating with the reduction of fat body mass and weight, serum leptin also reduced ($p < 0.01$). These results suggest that the short-term low-calorie diet reduces total body fat, mainly found in the abdominal region, and efficiently improve insulin sensitivity decreasing cardiovascular risk in obese women.

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Key words: *Obesity. Hypocaloric diet. Insulin. Adiponectin. Body weight.*

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LA DIETA BAJA EN CALORÍAS A CORTO PLAZO MEJORA LA SENSIBILIDAD A LA INSULINA Y LOS PARÁMETROS METABÓLICOS EN LAS MUJERES OBESAS

Resumen

La obesidad y la resistencia a la insulina se asocian con un aumento de los factores de riesgo cardiovascular, incluyendo las adipocitocinas. El propósito de este estudio fue investigar el efecto de una dieta baja en calorías sobre los lípidos séricos, las adipocinas, la resistencia a la insulina y la composición corporal en mujeres obesas. Se trataba de un estudio clínico en mujeres con obesidad de clase I, con edades entre 30-45 años, sometidas a una dieta hipocalórica durante 90 días. Se evaluaron basalmente y a los 30, 60 y 90 días la ingesta dietética, los parámetros antropométricos, la composición corporal, los lípidos séricos, la glucosa, la insulina, la leptina, la adiponectina y los índices HOMA-IR QUICKI. Hubo un descenso significativo del 30 % en el consumo de energía y un descenso del peso corporal, el índice de masa corporal y la circunferencia de la cintura ($p < 0,01$) durante todo el periodo de tratamiento. A pesar de que se redujo en promedio la masa magra corporal (kg), se observó que la masa magra corporal (%) se incrementó ($p < 0,01$) y que la cantidad de masa corporal grasa (Kg) disminuyó significativamente al tercer mes ($p < 0,05$). La presión sanguínea sistólica se redujo en 5 mmHg ($p < 0,05$) a los 90 días. Observamos un descenso de la insulina sérica y del HOMA-IR en el día 60 ($p < 0,05$), mientras que la adiponectina sérica aumentó ($p < 0,01$) durante el tratamiento. Como corroboración de la reducción de la masa corporal grasa y del peso, la leptina sérica también se redujo ($p < 0,01$). Estos resultados sugieren que la dieta hipocalórica a corto plazo reduce la grasa corporal total, fundamentalmente en la región abdominal y mejora de forma eficiente la sensibilidad a la insulina disminuyendo el riesgo cardiovascular en mujeres obesas.

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Palabras clave: *Obesidad. Dieta hipocalórica. Insulina. Adiponectina. Peso corporal.*

List of abbreviations

BMI: Body mass index.
CVD: Cardiovascular disease.
DBP: Diastolic blood pressure.
HDL: High-density lipoprotein.
FBM: Fat body mass.
HOMA-IR: Homeostatic Model Assessment-Insulin Resistance.
95% CI: 95% confidence interval.
IR: Insulin resistance.
LBM: Lean body mass.
LDL: Low-density lipoprotein.
OR: Odds ratio.
SBP: Systolic blood pressure.
VLDL: Very-low-density lipoprotein.
WC: Waist circumference.

Introduction

Obesity is defined by an accumulation of adipocytes throughout the body and is also associated with a variety of metabolic diseases[1]. To improve the adverse metabolic condition it is necessary to create a negative balance in energy intake, thereby leading to weight loss[2]. Obesity and insulin resistance are associated with an increase of cardiovascular risk factors, including inflammatory markers and adipocytokines[3].

The incidence of obesity and associated comorbidities is clearly increasing worldwide. In the past few years, this epidemiologic evidence has led to increased interest in adipose tissue as an active participant of physiologic and pathological processes[4,5]. Adipose tissue, then, can be considered as an active secretory organ able to send and respond to signals that modulate appetite, insulin sensitivity, energy expenditure, and inflammation. Regarding obesity and type 2 diabetes, it is well established that the association between visceral adipose tissue and insulin resistance is associated with increased cardiovascular risk[6].

Adiponectin, an adipose-specific plasma protein, possesses insulin-sensitizing and anti-atherogenic properties[7]. It has been well documented that plasma adiponectin is lower in obese subjects than in lean subjects and is negatively correlated with body weight, visceral fat mass and resting level of insulin[8]. Leptin is produced by adipocytes and its action occurs through the receptor in the hypothalamic nucleus. Leptin concentration is proportional to the total body fat[9].

Energy restriction followed by weight loss is important for reducing the risk of type 2 diabetes in individuals with obesity and impaired glucose homeostasis as well as for improving dyslipidemia and reducing blood pressure[2]. The purpose of this study was to evaluate the effect of short term low calorie diet on serum lipids, adipokines, insulin resistance and body composition in obese women.

Methods

Study participants

Thirty volunteers obese women; aged 30 to 45 years with a body mass index (BMI) of 30 to 34.9 kg/m^2 participated in the study at the Josué de Castro Institute of Nutrition, University of Rio de Janeiro (Rio de Janeiro, Brazil). Exclusion criteria were: diabetes mellitus; non-treated thyroid disease; treatment with lipid-lowering drugs and glucocorticoids; and weight loss treatment within the previous three months. All volunteers gave their written informed consent and this study was approved by the Ethics Committee of the State University of Rio de Janeiro (Rio de Janeiro, Brazil) (Protocol number 007.3.2008).

Energy requirement and composition of diets

The daily energy requirement was estimated by Dietary Reference Intakes[10]. The subjects were then prescribed a daily energy intake of 500 kcal lower than the estimated energy requirement. The dietary target for fat content was 25 to 30% of energy (E%), 15 to 20 E% from protein and 50 to 60 E% from carbohydrates[11]. The subjects were given dietary instructions consisting of isoenergetic interchangeable servings. Instructions were given to minimize differences in the amount of fruit and vegetables eaten, type of fat, amount and type of fibers, type of carbohydrates and frequency of meals.

Study design

A 3-month clinical trial was performed. All recruited women underwent a screening visit for selection before enrolling in the study. A dietitian supervised weight loss and diet compliance every month, until the third month. At the beginning and on 30, 60 and 90 days were evaluated meal records, anthropometric measures and laboratory assessment.

Dietary intake

All enrolled subjects received instructions to record their dietary intake for three days including a weekend day of each month completed during the intervention. Records were reviewed by a dietician and analyzed with a computerized dietary assessment program (Food Processor ESHA Research, Salem, Oregon, 1998). USDA composition food table were used as a reference[12].

Anthropometric measurements

Body weight, BMI and waist circumference (WC) were measured. Tetrapolar body electrical bioimpe-

dance was used to determine body composition[13]. An electric current of 0.8 mA and 50 kHz was produced by a calibrated signal generator (model 410e, Biodynamics, Seattle, WA, USA) and applied to the skin using adhesive electrodes placed on the right hand and foot. Fat body mass (FBM), and lean body mass (LBM) were estimated by body electrical bioimpedance. Blood pressure was measured twice in the right arm by the trained investigator, with a mercury sphygmomanometer and stethoscope after subjects had rested for a minimum of 10 minutes[14].

Laboratory assessment

Blood samples were obtained after 12 hour overnight fasting and were collected into tubes (Vacutainer, Becton Dickinson, USA) to obtain serum samples, respectively. Aliquots of serum were separated by centrifugation at 4,000 rpm for 15 minutes at room temperature (Excelsa Baby I, model 206, FANEM®, São Paulo, Brazil) and were individually kept at –20°C until assaying.

Serum concentrations of total cholesterol, triglycerides, high-density lipoprotein (HDL) cholesterol, glucose and uric acid were determined by enzymatic colorimetric assay (commercial kit BioSystems S.A, Barcelona, Spain), using an A15 Automatic Analyzer (BioSystems S.A., Barcelona, Spain). Low-density lipoprotein (LDL) was calculated[15]. Serum glucose and uric acid were measured by spectrophotometry using the glucose oxidase/peroxidase and uricase/peroxidase methods, respectively (A15 Automatic Analyzer, BioSystems S.A., Barcelona, Spain)[16]. Serum insulin was measured by a radioimmunoassay (RIA) kit (ImmuChem™ Coated Tube, MP Biomedicals®, LLC, USA)[17], with an assay sensitivity of 4.6 µU/mL and 12.2% of intra-assay coefficients of variation. Serum leptin was measured using a commercially available RIA assay kit (LINCO Research®, Missouri, USA), with 0.5 ng/mL assay sensitivity and < 8.3% intra-assay coefficient of variation. Finally, serum adiponectin was also measured by available a RIA assay kit (MILLIPORE®, Missouri, USA), with 2 ng/mL assay sensitivity and < 3.59% intra-assay coefficient of variation. To count samples of RIA assays a 2470 WIZARD² Automatic Gamma Counter (Perkin-Elmer Inc.) was used.

Homeostasis Model Assessment for insulin resistance (HOMA-IR) and Quantitative Insulin-Sensitivity Check Index (QUICKI) were calculated[18,19].

Statistical analysis

Data were presented as mean ± standard error mean. To check the distribution of continuous variables of interest, Kolmogorov-Smirnov adhesion test was performed. The comparisons of mean values over time were made using the paired Student's *t*-test and One-way ANOVA for comparison of fatty acids intake at baseline. Statistical analyzes were conducted, using the statistical package SPSS Statistics 17.0 and GraphPad Prism 5.0. Differences were considered significant at $p < 0.05$.

Results

Table I shows the general characteristics of the women studied. The mean age was 39.1 ± 0.8 years, and BMI and WC were 32.7±0.4 kg/m² and 99.2 ± 1.4 cm, respectively. Basal assessment of nutritional intake showed high variability, as it can be seen in table II.

Table I
Baseline characteristics of obese women (n = 30)

Measurements	*Mean*	*Minimum*	*Maximum*
Age (years)	39.1	30	45
Body weight (kg)	86.0	67.6	103.0
BMI (kg/m²)	32.7	29	37
WC (cm)	99.2	89	116
LBM (kg)	53.2	44.6	62.8
LBM (%)	62.0	56.1	67.3
FBM (kg)	32.1	19.0	44.2
FBM (%)	37.2	21.7	43.1
SBP (mmHg)	120.3	110	140
SBP (mmHg)	80.5	60	100
Total cholesterol (mg/dL)	182.4	124	239
HDL-c (mg/dL)	53.9	32	80
LDL-c (mg/dL)	106.2	51.6	161.4
Triglycerides (mg/dL)	110.9	45	272
Uric acid (mg/dL)	4.2	69	99
Fasting glucose (mg/dL)	83.8	2.9	7.8
Insulin (µU/mL)	34.0	12.4	92.0
Leptin (ng/mL)	25.4	15.0	59.0
Adiponectin (ng/mL)	4.9	1.2	13.9
HOMA-IR	7.4	2.6	23.0
QUICKI	0.361	0.255	0.477

BMI: Body mass index, WC: waist circumference, LBM: lean body mass, FBM: fat body mass, SBP: systolic blood pressure, DBP: diastolic blood pressure.

Table II
Baseline characteristics of dietary intake of obese women

Parameters	*Mean*	*Minimum*	*Maximum*
Total energy intake (KJ)	7253.4	2655.9	15372.6
Carbohydrates (% TEI)	54.0	34.0	77.0
Protein (% TEI)	18.9	8.0	28.0
Lipids (% TEI)	27.3	12.0	43.0
SFA (% TEI)	10.0	2.0	21.0
MUFA (% TEI)	9.0	2.0	13.0
PUFA (% TEI)*	4.1*	1.0	8.0
Trans FA (g/day)	3.1	0.0	26.9
Cholesterol (mg/day)	211.7	16.3	591.3
Dietetic fiber (g/day)	21.9	5.3	62.2
Soluble fiber (g/day)	5.2	0.8	14.8

*$p < 0.0001$ compared to PUFA and MUFA, TEI: total energy intake, SFA: saturated fatty acid, PUFA: polyunsaturated fatty acid, MUFA: monounsaturated fatty acid, FA: fatty acid.

Was observed PUFA intake lower than MUFA and SFA ($p < 0.0001$).

Table III describes the effect of hypocaloric diet on dietary consumption. Energy intake decreased during the total time of treatment with low-calorie diet compared to baseline. There was a significant decrease in energy intake of about -31%, -23%, -28% at 1st, 2nd and 3rd months, respectively. The women had lower intake of carbohydrates and higher intake of lipids in the first (-9.0% $p < 0.01$ and +5.7%, $p < 0.05$, respectively) and third month (-10.3 % and +8.8%, $p < 0.01$, respectively), while the consumption of *trans* FA was lower during the total time of treatment. They had an important decrease in fiber ingestion ($p < 0.05$) in the first month of treatment, which continued to drop during treatment.

Table IV shows the effect of hypocaloric diet on anthropometric, body composition and blood pressure measures. Body weight loss and consequent reduction of BMI and WC were significant ($p < 0.01$) throughout the treatment period. Despite the percentage of LBM had increased significantly ($p < 0.01$) during treatment, it is observed that the amount of LBM (kg) reduced in average and that the quantity of FBM also decreased; however, it became significant ($p < 0.05$) in the 3rd month. Systolic blood pressure also reduced significantly ($p < 0.05$) during the treatment period, while diastolic blood pressure reduction was not significant.

Table V describes the effect of low-calorie diet on lipid profile, insulin resistance and adipokines. Serum lipids did not change significantly with a reduced calorie diet. The treatment showed a decrease in serum insulin and in HOMA-IR in the first two months ($p < 0.05$). On the contrary, serum concentrations of adiponectin increased significantly ($p < 0.01$) during treatment. Corroborating with the reduction of body fat and weight, serum leptin levels also reduced significantly during treatment ($p < 0.01$), showing a 32% decline in the second month compared to baseline. In the 3rd month, energy intake was related do weight loss (r 0.564, $p < 0.05$) and adiponectin was inversely related to HOMA-IR (r -0.365, $p < 0.05$).

Table III
Dietary consumption by each month of treatment with hypocaloric diet

Parameters	*1st month* Δ ± *SEM*	*2nd month* Δ ± *SEM*	*3rd month* Δ ± *SEM*
Total energy intake (KJ)	-2236.6 ± 848.8*	-1680.2 ± 771.6*	-2083.4 ± 881.9*
Carbohydrates (% TEI)	-9.0 ± 2.8**	-5.2 ± 2.8	-10.3 ± 3.3**
Protein (% TEI)	3.3 ± 1.6	2.1±1.8	1. 5 ± 1.5
Lipids (% TEI)	5.7 ± 2.7*	3.1±2.3	8.8 ± 2.9**
SFA (%TEI)	3.1 ± 1.6	1.6±1.6	5.6 ± 2.0*
MUFA (%TEI)	1.6 ± 1.1	-0.1±1.0	1.9 ± 1.3
PUFA (%TEI)	0.7 ± 0.4	0.5±0.6	0.8 ± 0.6
Trans FA (g/day)	-3.1 ± 1.3*	-3.4±1.5*	-3.2 ± 1.6
Cholesterol (mg/day)	-11.7 ± 34.3	-15.6±38.3	40.4 ± 55.4
Dietetic fiber (g/day)	-6.7 ± 3.2*	-4.5±3.9	-6.9 ± 4.1
Soluble fiber (g/day)	-1.5 ± 1.0	-1.1±1.0	-1.5 ± 1.1

*p < 0.05. **p < 0.01. Δ: mean difference compared to the baseline, TEI: total energy intake, SFA: saturated fatty acid, PUFA: polyunsaturated fatty acid, MUFA: monounsaturated fatty acid, FA: fatty acid.

Table IV
Effect of hypocaloric diet on anthropometric measures, body composition and blood pressure parameters

Measurements	*1st month* Δ ± *SEM*	*2nd month* Δ ± *SEM*	*3rd month* Δ ± *SEM*
Body weight (kg)	-2.0 ± 0.2**	-2.7 ± 0.4**	-3.0 ± 0.5**
BMI (kg/m²)	-0.8 ± 0.1**	-1.0 ± 0.2**	-1.1 ± 0.2**
WC (cm)	-1.7 ± 0.4**	-2.2 ± 0.5**	-3.2 ± 0.6**
LBM (kg)	-0.1 ± 0.3	-0.7 ± 0.3	-0.6 ± 0.4
LBM (%)	1.4 ± 0.4**	1.3 ± 0.4**	1.6 ± 0.4**
FBM (kg)	-0.8 ± 0.7	-1.3 ± 0.7	-1.7 ± 0.7*
FBM (%)	-0.1 ± 0.8	-0.4 ± 0.7	-0.8 ± 0.7
SBP (mmHg)	-5.7 ± 2.1*	-3.8 ± 1.8*	-5.0 ± 1.9*
DBP (mmHg)	-1.7 ± 2.2	-3.8 ± 2.0	-2.8 ± 2.2

*p < 0.05; **p < 0.01. Δ: mean difference compared to the baseline, BMI: Body mass index, WC: waist circumference, LBM: lean body mass, FBM: fat body mass, SBP: systolic blood pressure, DBP: diastolic blood pressure.

Table V
Effect of hypocaloric diet on lipid profile, insulin resistance and adipokines

Measurements	*1st month* Δ ± *SEM*	*2nd month* Δ ± *SEM*	*3rd month* Δ ± *SEM*
Total cholesterol (mg/dL)	-0.9 ± 4.6	-10.8 ± 6.6	4.5 ± 4.5
HDL-c (mg/dL)	-1.2 ± 1.5	-1.0 ± 2.6	-1.8 ± 2.3
LDL-c (mg/dL)	2.0 ± 4.6	-5.0 ± 5.3	5.3 ± 3.7
Triglycerides (mg/dL)	-7.7 ± 9.9	-4.8 ± 10.2	4.8 ± 10.7
Uric acid (mg/dL)	-0.1 ± 0.2	-0.3 ± 0.1	-0.1 ± 0.1
Fasting glucose (mg/dL)	-1.6 ± 2.3	-3.1 ± 1.6	1.0 ± 1.8
Insulin (μU/mL)	-6.7 ± 2.9*	-6.6 ± 3.0*	1.0 ± 3.2
Leptin (ng/mL)	-5.8 ± 1.8**	-8.3 ± 1.5**	-5.7 ± 1.9**
Adiponectin (ng/mL)	0.8 ± 0.3**	1.1 ± 0.5*	1.0 ± 0.4**
HOMA-IR	-1.7 ± 0.8*	-1.8 ± 0.8*	0.1 ± 0.8
QUICKI	0.019 ± 0.009*	0.009 ± 0.008	-0.015 ± 0.009

*p < 0.05; **p < 0.01. HDL-c: high-density lipoprotein cholesterol, LDL-c: low-density lipoprotein cholesterol, VLDL-c: very-low-density lipoprotein cholesterol, HOMA-IR: Homeostatic Model Assessment - Insulin Resistance.

Discussion

This study showed that obese women submitted to a low-calorie diet treatment loosed weight, improved insulin resistance and other metabolic abnormalities characteristic of obesity.

Adherence to dietary treatment is very important. In the present study there was reduction of energy intake during the three-month intervention, with greater emphasis in the first month. These data confirm previous studies that also reported success in adherence to dietary treatment for weight loss[20,21]. The progressive increase in energy intake after the initial reduction may have occurred due to adaptive thermogenesis. The decreased energy expenditure when the intake is limited is a counter-productive response during a hypocaloric diet, contributing significantly to lower efficacy of long-term treatment for obesity.

As a consequence of lower caloric intake, mainly carbohydrates, there was a reduction in body weight and BMI of these women, both reaching 3.5% in the last month. The percentage of LBM increased during the study, but mainly due to the reduction of FBM, which remained significant in the third month.

The low-calorie diet alone has been an intervention strategy for weight loss in several studies[22,23]. In a systematic review of 21 studies, in which the diet was the main intervention, we observed that diet alone promoted greater weight loss up to 36 months from the start of the intervention, compared to nutritional guidelines only. Lofgren et al.[24] evaluated in premenopausal women, the response of a hypocaloric diet for 10 weeks with a reduction of 26.6% of VET and observed a reduction of 4.6% of body weight, slightly higher than our study. However, the amount of weight loss varies widely among studies due to different strategies, duration of interventions, age, degree of overweight, etc[22].

The present study showed that a significant percentage of weight loss could promote improvement in some clinical and metabolic parameters. Although other studies did not show higher percentages of weight loss, they showed that even small weight losses contribute to important health benefits[20,21,24,25]. This is probably due to a decrease in WC, which was gradual and reached a higher percentage in the last month of treatment. It is well known that the excess adiposity in the abdominal region is more frequently associated with metabolic abnormalities, such as insulin resistance and atherosclerosis[26]. These findings about treatment with hypocaloric diet and decreased in WC are in agreement with other studies[24,27].

Generally, treatments with low-calorie diets lead to body weight loss. Regarding body composition, there is loss of both adipose tissue and LBM, depending on physical activity. However, in this study, was observed that the loss of body fat promoted relative increase in LBM, i.e., the increase in the percentage of LBM leads to a misconception of its increase. In fact, fat mass has decreased more than lean mass. It is recommended that the loss of lean mass does not exceed 30% of the weight lost. Therefore, maintaining the largest possible amount of lean mass is essential[28]. Thus, this study agrees with these recommendations.

Systolic blood pressure reduced during the study. Our results corroborate with those reported by Al-Sarraj et al.[29], which showed a decrease in both SBP and DBP after treatment with calorie-restricted diet for six weeks, followed by fat-restricted diet for six weeks. In addition, weight loss greater than 5% showed a significant reduction in SBP[25]. The decrease in SBP showed possible relationship with reduced serum insulin, since high concentrations of serum insulin is a contributing factor in the pathophysiological mechanism of systemic arterial hypertension. Therefore, it is likely that a significant reduction in serum insulin had contributed to reduction in SBP.

Insulin resistance in obese subjects markedly increases the risk of coronary artery disease[30]. As expec-

ted, was found that the amount of weight lost had a significant effect on the degree of improvement in insulin sensitivity. The women in this study showed a significant reduction in insulinemia in the first two months of treatment. Regarding insulin concentrations, the mean HOMA-IR was also reduced in the same period, while QUICKI, which evaluates sensitivity to insulin, showed an effect opposite to HOMA-IR in the first month. These results corroborate with the study conducted by Samaha et al.[20], which showed an improvement in insulin sensitivity in obese subjects, after six months of a carbohydrate-restricted diet. Thus, despite the greater weight loss at the end of our study, adherence to treatment and increased serum adiponectin seem to be the main factors related to improve insulin sensitivity.

Adiponectin, a protein secreted by adipocytes, is related to improved insulin sensitivity[31]. It increases insulin sensitivity by increasing fatty acid oxidation and glucose uptake in skeletal muscle and adipose tissue and decreased hepatic glucose release[32].

Adiponectin levels increased during the study, in agreement with reduction of HOMA-IR, weight and WC, which means that insulin resistance reduced with the treatment. The data of adiponectin levels are in agreement with those of Al-Sarraj et al.[29], which showed an increase in adiponetin average (1.58 mg/L) after 12 weeks of treatment with fat-restricted diet. Considering that adiponectin also acts as a protective effect on cardiovascular function[33], the increase observed in this study can be considered a good perspective of the effect of low-calorie diet.

Leptin is another hormone secreted by adipose tissue and is directly related to BMI[34]. Leptin production and its action maintain a regulatory control of energy balance in the body. Insulin is the major regulator of leptin production by adipose tissue in the healthy state. Insulin infusions increase circulating leptin concentrations in humans. Insulin-stimulated leptin production appears to involve increased glucose metabolism while blockade on glucose transport or glycolysis inhibits leptin expression and secretion in isolated adipocytes. Insulin and leptin concentrations decrease during fasting and energy-restricted diets, independent of body fat changes, ensuring that feeding is triggered before body energy stores become depleted[35].

The reduction in leptin after weight loss is variable. Some authors reported reductions of 22%[36] and 45%[37] on serum. While other authors have reported minor loss (5.9%) but significant and simultaneous loss of 7.5% of BFM (kg) and 6.5% of WC (cm)[24]. Still, De Luis et al.[25] showed that regardless of the amount of body weight lost, leptin significantly reduced in groups with weight loss greater and lesser than 5%. Throughout the treatment, the women had significantly lower concentrations of leptin, which is consistent with the reduction of BMI and WC and confirms previous studies[24,25].

In conclusion, the results suggest that short-term nutritional treatment with a balanced hypocaloric diet, in relation to macronutrients, reduces body weight and total body fat, mainly found in the abdominal region, and efficiently improves insulin sensitivity decreasing cardiovascular risk in obese women.

Authors' contributions

GVBH – Followed the subjects, carried out immunoassays, statistical analyses and draft the manuscript; SKU - Followed the subjects, carried out immunoassays, participated to draft the manuscript; FNN - Carried out biochemistry assessment; EGM - Participated in study design;

GR - Participated in study design, statistical analyses and to draft the manuscript; MCFP - Participated in study design, statistical analyses and to draft the manuscript. All authors read and approved the final manuscript.

Competing interests

The authors declare that they have no competing interests.

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