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Effect of Dietetic Intervention in Brazilian Postmenopausal Women with Metabolic Syndrome

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Abstract

The study aim was to investigate the effects of individualized dietary intervention in postmenopausal women with metabolic syndrome. In this randomized and controlled clinical trial, a total of 55 women 40-65 years old presenting metabolic syndrome diagnosis were randomly assigned into intervention and control groups (50.9%). The intervention group received individualized diets prescribed by nutritionists. Participants were assessed at baseline moment (M0) and after a 16 weeks period (M1) for anthropometric, clinical, biochemical, and dietary parameters. The Student's t-test was applied for independent and dependent numerical variables. It was hypothesized that postmenopausal women with metabolic syndrome obtaining dietary intervention would show improvement in the assessed parameters. At M0 there was no significant difference in all parameters between groups, demonstrating homogeneity samples. Yet, at M1, the intervention group showed statistical significant differences: decline in body weight (−3.5 kg), body mass index (−0.3 kg/m^2), waist circumference (−4.1 cm), and systolic (−7.6 mmHg) and diastolic blood pressure (−1.6 mmHg); reductions in plasma levels of total cholesterol, triglycerides, and LDL-cholesterol; as well as increase of HDL-cholesterol and decrease in habitual consumption of energy and dietary lipids. Regarding diet quality, we observed increase of fish, low-fat dairy, fruits, and vegetables consumption; and decrease of butter, margarine, sweets, soft drinks, and fast food consumption. In contrast, the control group showed no improvement in any of these parameters; quite the contrary, they even showed significant weight gain (+0.6 kg). The intervention group had 18.5% reversal rate in metabolic syndrome diagnosis, showing that individualized nutrition care was effective in controlling the metabolic syndrome.

Keywords: Metabolic syndrome; Dietary modification; Women; Nutrition; Chemical analysis; Intervention studies

Introduction

Metabolic Syndrome (MS) is a complex disorder, characterized by interrelated metabolic abnormalities [1]. Abdominal obesity is considered a trigger for MS [2], which represents a risk factor for diseases interconnected by the genesis of insulin resistance [3].

The most recent definition for MS was described by the International Diabetes Federation (IDF) in 2009 [4], featuring MS as an association of factors like: hypertriglyceridemia (plasma triglyceride levels greater than or equal to 150 mg/dL), low plasma levels of HDL cholesterol (less than 40 mg/dL for men and 50 mg/dL for women), increased plasma glucose levels (greater than or equal to 100 mg/dL), type 2 diabetes mellitus diagnosis, and/or elevated systemic arterial blood pressure (greater than or equal to 130/85 mmHg). Such concept can be easily employed as a diagnostic tool in clinical practice to ascertain the patient's metabolic and cardiovascular risk, which serves as a guide for their treatment [5].

This syndrome entails a greater risk for the development of type 2 diabetes mellitus and cardiovascular diseases. In association with high cardiovascular morbidity and mortality, MS is currently regarded as an epidemic worldwide that is progressively reaching alarming numbers [6]. Approximately 26% of the North-American adult population suffers from it [7]. In Brazil the prevalence of MS in the general population is not known, but it seems that in postmenopausal women it ranges from 39% to 61% [8-10]. Postmenopausal women appear to be a major risk group for MS, since menopause causes the loss of ovarian function and subsequent decline of endogenous estrogen. Estrogen deficiency leads to an alteration in lipid metabolism and accumulation of visceral adipose tissue [11,12]. Cho et al. [13] found that postmenopause status was an independent risk factor for MS and all of its individual components.

Despite all, the precise etiology of MS still remains unclear, but it is known to be a complicated interaction between genetic, metabolic, and environmental aspects [14]. Among the environmental aspects, eating habits are of fundamental importance in this disorder's prevention and treatment. Therefore, the eating behavior modification and the weight loss are considered the primary strategies to control MS, enabling reduction of its risk factors by educational intervention that focuses on nutritional aspects [1,15].

Significant associations between dietary patterns and development of MS are documented in the scientific literature [16]. Cross-sectional studies point to a lower prevalence of MS in populations that habitually consume healthy diets rich in fruits, vegetables, fish, and whole grains and poor in saturated fat, cholesterol, and carbohydrates with high glycemic index [17-20], particularly among postmenopausal women [21]. Accordingly, it was hypothesized that postmenopausal women With MS who followed an individualized dietary intervention for a period of up to 16 weeks would show health improvements.

However, there is a lack of studies that have evaluated the effects of a dietary intervention in the presence of MS as a unique independent disease [22]. And in Brazil, there are only a few studies on metabolic syndrome in the general population and even fewer are those that investigate it in postmenopausal women [10]. Therefore, the objective of this study was to evaluate the effects of an individualized dietary intervention in postmenopausal women presenting MS.

Methods and Materials

This study had a longitudinal and randomized controlled clinical trial design, with the voluntary participation of overweight and obese adult women with MS, according to the diagnostic criteria defined by IDF [4]. The participants were allocated randomly to the intervention and control groups. This latter group was subjected to no more than superficial advice regarding the importance of a lifestyle change for controlling MS, while keeping their habitual diet. The intervention group suffered a dietary intervention with energetic restriction by means of an individualized nutritional follow-up on a monthly basis. Both groups were advised not to start any kind of regular physical activity during the follow-up period of the study. The trial lasted 16 weeks and the assessments were conducted at the beginning (M0) and at the end of the study (M1).

The subjects met the following inclusion criteria: age between 40 and 65 years old, BMI between 25 and 39.9 kg/m^2, and postmenopausal. The exclusion criteria were clinical diagnosis of congestive cardiac failure, endocrinopathies (except for dyslipidemiae and diabetes mellitus type 2), uncompensated chronic diseases, renal or hepatic insufficiency, under continuous use of corticoid-base drugs, and/or having underwent dietary treatment in the six months prior to the beginning of the study.

The recruitment of participants was conducted at the Community and Family Clinic of Integral Medicine of a university hospital in the city of Rio de Janeiro, Brazil. All women in the sample received detailed information regarding the study's nature, purpose, and types of evaluation and signed an informed consent prepared in accordance with the Resolution 196/96 of the Brazilian Health Ministry's National Health Council. This project was approved by the Research Ethics Committee of the "Pedro Ernesto" University Hospital (HUPE) of the Rio de Janeiro State University (UERJ) – protocol #1811.

Anthropometric and dietary assessments were conducted at the Physiology Laboratory Applied to Physical Education, while the biochemical analysis was performed by the HUPE's Central Laboratory of Clinical Analysis, following a 12-hour fasting period prior to blood collection.

In relation to the anthropometric assessment, the measurements were taken twice and the mean value was used for data analysis. The data collection procedures obeyed the norms proposed by Norton and Olds [23], including Total Body Mass, stature, waist circumference, and Body Mass Index (BMI) assessment. Other data such as systemic arterial blood pressure, biochemical analysis (plasma levels of HDL and LDL cholesterol, triglycerides, and fasting glucose), and dietary information was collected from each of the study participants. The biochemical parameters were compared to the recommended goal values for the MS treatment according to the I Brazilian Guidelines for the Diagnosis and Treatment of Metabolic Syndrome [24]. Food consumption was investigated by two dietary inquiries: a diet history and a food frequency questionnaire. Energy, macronutrient, fiber, potassium, and calcium intakes from ingested foods and beverages were calculated using the nutrition software Diet Pro 5.0 [25], after being converted from common household measures to its corresponding milliliters or gram weights according to Pinheiro et al. [26]. The food frequency questionnaire applied contained 30 items including different foods and beverages. Consumption frequencies were inquired from each participant in order to construct a qualitative estimation of their food consumption in the previous four months.

Aiming to promote eating habits changes in the intervention group participants, we offered an individualized diet with energy restriction of 500 to 1,000 kilocalories (kcal) per day, calculated based on the diet history, according to the recommendations from the Brazilian Guidelines on Obesity [27] and the I Brazilian Guidelines for the Diagnosis and Treatment of Metabolic Syndrome [24]. The percentages of recommended macronutrients were as follows: 50 to 60% of the Total Energy Value (TEV) from carbohydrates, 25 to 35% of the TEV from lipids, and 0.8 to 1.0 g of proteins/ kg of body weight/ day. The amount of offered dietary fiber was 20 to 30 g/day.

During the whole study period, individual meetings at regular 4-week intervals were conducted in order to reinforce the importance of eating habits modifications and to answer any questions related to the prescribed dietary plan. Fortnightly telephone contacts also served as an impetus to improve diet's adherence. The recommendations were based on whole grains, beans, fish, fruits, and vegetables rich diets. The processed carbohydrates, salt, trans and saturated fat consumption was progressively discouraged.

Data normality and the distribution of the variables were tested by the Kolmogorov-Smirnov test. The frequencies, means, and standard deviations were calculated. The Levene's test was conducted in order to verify the sample homogeneity. The Student's t-test for the independent samples or the Mann Whitney test were used at M0 for comparison between the two groups (intervention and control) and the Student's t-test for dependent samples was applied between the M0 and M1 of each group separately. Categorical data were compared using the $\chi 2$ test. The statistical analysis was conducted utilizing the Statistica 6.0 computer program for Windows [28]. The sample size needed for the study was determined based on the detection of a difference in primary end point (impaired fasting glycemia), with a significance level of 5% and power of 90% [15,16]. The significance level adopted wasa P value $\leq$ 0.05.

Results

Fifty-five women diagnosed with MS were selected and divided into intervention and control groups. Upon comparison, it was observed that there was no significant difference in the anthropometric, biochemical, and dietary variables at M0, which demonstrates the homogeneity of the sample at that time (P > 0.05). About 69% (n=38) of the participants presented overweight or class I obesity, while 31% (n=17) were class II obese individuals. The systolic and diastolic arterial blood pressure were high at M0 in both groups (greater than 120/80 mmHg), as well as the plasma levels of glucose, total cholesterol, triglycerides, and LDL cholesterol. The MS component most frequently found in the participants, aside from abdominal obesity, was the systemic arterial hypertension (92.7%, n=51), followed by hyperglycemia (80%, n=44), reduced plasma level of HDL cholesterol (58.2%, n=32), and hypertriglyceridemia (49.1%, n=27).

The comparison of the demographic, anthropometric, clinical, biochemical, and dietetic characteristics of the sample at M0 may be visualized on Table 1.

Parameters	Intervention Group (N=27)	Control Group (N=28)	p- value
Demographic			
Age (years)	55.26 ± 6.41	57.32 ± 7.00	0.26
Anthropometric			
Total body mass (kg)	81.5 ± 13.7	79.9 ± 13.5	0.67
Body mass index (kg/m^2)	32.1 ± 4.4	33.2 ± 4.6	0.37
Waist circumference (cm)	104.9 ± 11.3	102.3 ± 11.5	0.38
Clinical			
Systolic arterial blood pressure (mmHg)	134.7 ± 18.8	134.4 ± 18.9	0.95
Diastolic arterial blood pressure (mmHg)	82.6 ± 14.4	85.1 ± 16.0	0.84
Biochemical			
Glucose (mg/dL)	118.7 ± 39.1	133.0 ± 56.6	0.35
Triglycerides (mg/dL)	188.5 ± 135.8	158.3 ± 80.2	0.42
Cholesterol (mg/dL)	227.0 ± 44.9	225.6 ± 46.1	0.91
HDL cholesterol (mg/dL)	51.1 ± 8.9	48.0 ± 9.1	0.20
LDL cholesterol (mg/dL)	141.3 ± 44.3	142.8 ± 43.4	0.89
Dietetic			
Energy (kcal)	1921 ± 426	2039 ± 362	0.27
Carbohydrates (%)	47.6 ± 11.75	51.4 ± 7.9	0.16
Proteins (g/kg/day)	1.2 ± 0.5	1.1 ± 0.3	0.92
Lipids (%)	32.6 ± 7.6	30.4 ± 6.9	0.26
Saturated Fatty Acids (%)	7.14 ± 3.3	8.7 ± 3.0	0.17
Monosaturated Fatty Acids (%)	5.6 ± 3.7	7.9 ± 2.5	< 0.01*
Polysaturated Fatty Acids (%)	4.1 ± 2.4	5.5 ± 4.6	0.10
Fibers (g/day)	18.4 ± 4.9	18.1 ± 5.7	0.78
Calcium (mg/day)	876.8 ± 492.5	1647.9 ± 279.1	0.01*
Potassium (mg/day)	1647.9 ±864.7	1935.9 ± 826.0	0.60

Table 1: Comparison of the demographic, anthropometric, clinical, biochemical, and dietetic parameters between intervention and control groups at M0

Values are means ± standard deviation. The Student's t-test for independent samples was used for between-group comparisons.

* express significant difference with P < 0.05. HDL: high density lipoprotein; LDL: low density lipoprotein.

Table 2 presents the comparison between the status of the participants at M0 and at M1, i.e., following the 16-week intervention of the anthropometric, biochemical, and dietary parameters of the women studied. There was a mean reduction of 3.5 kg in total body mass in the intervention group (P<0.01) and an increase of 0.6 kg in the control group, both of which being statistically significant (P=0.03). A significant reduction in BMI (0.28 kg/m^2) (P<0.01), in waist circumference (- 4.1 cm) (P<0.01), and in the arterial systolic (7.6 mmHg) (P<0.01) and diastolic (1.6 mmHg) blood pressure (P=0.02) was observed in the intervention group at M1. Plasma levels of triglyceride, total cholesterol, and LDL cholesterol in the intervention group decreased significantly at M1 (P ≤ 0.01). HDL cholesterol levels increased significantly following the intervention (P<0.01) (Table 2). Of the 49% (n=27) participants from the intervention group who presented HDL cholesterol levels below 50 mg/dL, 32.7% (n=18) reached this target (HDL ≥ 50 mg/dL) after the 16 weeks of the study.

Of the 27 women from the intervention group, five no longer had a MS diagnosis at the end of the study, which amounted to an 18.5% reversal rate in MS during this follow-up period. The dietary intervention reduced the prevalence of MS in the intervention group when compared to the control group. In relation to the dietary variables, a significant reduction in the consumption of energy (P<0.01), lipids (P<0.01), and saturated fatty acids (P=0.03) was observed in the intervention group at M1 (Table 2).

The percentages of macronutrients in relation to the TEV were calculated in order to compare them to the values set forth by the I Brazilian Guidelines for the Diagnosis and Treatment of Metabolic Syndrome [24]. The nutritional composition of the habitual diet of the participants from both groups presented percentages of lipids in accordance with the recommendations from the beginning of the study. However, the percentages of carbohydrates for the intervention group were below the recommended levels, which were corrected at the end of the study. The amount of protein consumed initially and offered throughout the study was around 1.1 to 1.2 g/kg/day, slightly above the recommendation of 0.8 to 1.0 g/kg/day, or 15% of the TEV until the end of the study.

Values are means ± standard deviation. The Student's t-test for dependent samples was used for intra-group comparisons. * express significant difference with P<0.05. M0, baseline moment; M1, after 16 weeks intervention; SAP, Systolic arterial blood pressure; DAP, Diastolic arterial blood pressure; HDL, high density lipoprotein; LDL, low density lipoprotein.

Parameters	**Intervention group (N=27)**		**P-value**	**Control Group (N=28)**		**P-value**
	M0	**M1**		**M0**	**M1**	
	X ± DP	**X ± DP**		**X ± DP**	**X ± DP**	
Anthropometric						
Total body mass (kg)	81.5 ± 13.7	78.0 ± 13.1	< 0.01*	79.9 ± 13.5	80.5 ± 13.7	0.03*
Body mass index (kg/m^2)	32.1 ± 4.4	31.82 ± 4.6	< 0.01*	33.2 ± 4.6	33.5 ± 4.4	0.67
Waist circumference (cm)	104.9 ± 11.3	100.8 ± 10.4	< 0.01*	102.3 ± 11.5	102.3 ± 11.2	0.72
Clinical						
SAP (mmHg)	134.7 ± 18.8	127.1 ± 18.9	< 0.01*	134.4 ± 18.9	137.8 ± 20.1	0.33
DAP (mmHg)	82.6 ± 14.4	81.0 ± 17.3	0.02*	85.1 ± 16.0	83.7 ± 9.2	0.55
Biochemical						
Glucose (mg/dL)	118.7 ± 39.1	107.4 ± 21.2	0.09	133.0 ± 56.6	138.6 ± 63.1	0.18
Triglycerides (mg/dL)	188.5 ± 135.8	151.8 ± 84.7	0.04*	158.3 ± 80.2	167.0 ± 72.0	0.44
Cholesterol (mg/dL)	227.0 ± 44.9	213.0 ± 45.6	0.01*	225.6 ± 46.1	229.3 ± 45.8	0.46
HDL cholesterol (mg/dL)	51.1 ± 8.9	52.04 ± 10.7	< 0.01*	48.0 ± 9.1	51.1 ± 8.5	0.93
LDL cholesterol (mg/dL)	141.3 ± 44.3	130.6 ± 43.7	0.01*	142.8 ± 43.4	144.8 ± 43.5	0.69
Dietetic						
Energy (kcal)	1921 ± 426	1495 ± 359	< 0.01*	2039 ± 362	2041 ± 410	0.12
Carbohydrates (%)	47.6 ± 11.75	50.9 ± 4.7	0.14	51.4 ± 7.9	50.5 ± 10.1	0.60
Proteins (g/kg/day)	1.2 ± 0.5	1.1 ± 0.2	0.63	1.1 ± 0.5	1.2 ± 0.35	0.81
Lipids (%)	32.6 ± 7.6	24.8 ± 9.24	< 0.01*	30.4 ± 6.9	30.4 ± 9.9	0.98
Saturated Fatty Acids (%)	7.14 ± 3.3	6.22 ± 3.84	0.03*	8.7 ± 3.0	8.2 ± 2.7	0.51
Monosaturated Fatty Acids (%)	5.6 ± 3.7	6.9 ± 3.2	< 0.01*	7.9 ± 2.5	7.5 ± 3.5	0.17
Polysaturated Fatty Acids (%)	4.1 ± 2.4	6.2 ± 6.22	0.10	5.5 ± 4.6	4.6 ± 4.0	0.38
Fibers (g/day)	18.4 ± 4.9	18.1 ± 5.7	0.80	18.1 ± 5.7	20.5 ± 7.4	0.36
Calcium (mg/day)	876.8 ± 492.5	1053.1 ± 533.0	0.20	1647.9 ± 279.1	859.3 ± 497.6	0.02*

Potassium (mg/day)	1647.9 ± 864.7	1935.9 ± 826.0	0.69	1935.9 ± 826.0	1778.6 ± 978.6	0.43

Table 2: Comparison of the anthropometric, clinical, biochemical, and dietetic parameters between M0 and M1 from the intervention and the control groups.

On Table 3, the comparison between the answers to the food frequency questionnaires provided by the participants' groups at M0 and M1 may be observed.

In relation to the diet quality evaluated by the food frequency questionnaire (Table 3), it was observed that the type of meat most frequently consumed by individuals from both groups was chicken, while fish was the least. However, the consumption frequency of fish and of low fat dairy products increased in the intervention group. As for leafy vegetables, there was a consumption frequency increase in the intervention group and a decrease in the control group. The fruits consumption frequency increased and that of margarine/ butter, sweets, soft drinks, and fast food decreased in the intervention group after the follow up.

Regarding the control group, the instructions provided about the lifestyle change importance did not result in any positive and statistically significant change in the anthropometric, biochemical, and dietary parameters studied.

Foods	Consumption percentage (%)											
	Intervention (n=27)						Control (n=28)					
	4x/weekor more		2 to 3x/week		Up to 1x/week		4x/week or more		2 to 3x/ week		Up to 1x/week	
	M0	M1	M0	M1	M0	M1	M0	M1	M0	M1	M0	M1
Beef	41*	23*	41	44	18*	33*	25	15	39	45	36	40
Chicken	59	59	37	41	4	--	39	45	43	41	18	14
Fish	--	--	15	29	82	71	--	--	18	14	82	86
Low Fat Milk and Dairy	37*	81*	16	22	47	41	25	21	4	--	71	79
Whole Fat Milk and Dairy	22	--	--	--	78	100	29	14	4*	24*	67	62
Vegetables A	41	78	22	11	37	11	50	53	25	36	25	11
Vegetables B	49	70	40	19	22	--	65	71	21	25	14	4
Fruits	70	89	15	7	15	4	60	71	11	11	29	18
Margarine/ Butter	56	34	11	4	33	62	74	57	7	11	19	32
Sweets	4	--	37	19	59	85	14	18	39	46	47	36
Soft Drinks	41	11	26	23	33	66	10	11	39	39	51	50
Fast Food	--	--	15	4	85	96	--	--	--	11	100	89

Table 3: Comparison of the food frequency questionnaires taken by the participants of the intervention and control groups at M0 and M1.

Values are percentages. M0, baseline moment; M1, after 16 weeks intervention.

* P<0.05 by the t-student test

Discussion

In the present study, the effect of the individualized hypocaloric diet applied independently as a lifestyle change strategy for 16 weeks in the treatment of women with MS resulted in significant reductions in total body mass, BMI, waist circumference, systolic and diastolic arterial blood pressure, increase in plasma levels of HDL cholesterol, reduction in plasma level of total cholesterol, triglycerides, and LDL cholesterol and a decrease in the habitual consumption of energy and dietary lipids, along with an increase in the consumption of monosaturated fatty acids. The dietary modification proposed was well tolerated and there was good adhesion by the intervention group to the diet prescribed. The reversal of 18.5% in the prevalence of MS in the intervention group demonstrated the effect of the dietary intervention in the treatment of MS.

The isolated effect of the dietary intervention in the control of MS components was investigated. Other randomized clinical trials have already tested the effect of the diet and/or of regular physical activity in individuals with MS, finding independent and statistically significant effects for isolated or combined interventions. In 2007, a Norwegian study, lasting one year, verified the effect of diet and/or physical exercise in the reversal of MS in 137 adult individuals.

Following both interventions, only 14 participants (32.6%) still presented MS. In the diet group, this number increased to 22 (64.7%) and in the exercise group, to 26 individuals (76.5%) [29]. Another study, including 90 overweight men presenting MS, obtained similar results. The participants followed a very low energy diet for a two months period, followed by regular physical activity for a six months period. In the first phase, there was a mean weight loss of 14.2 ± 4.0 kg, while in the second phase the reduction was of only 4.8 ± 0.8 kg. The authors concluded that the addition of physical exercise to the dietary counseling was not as effective as the dietary intervention by itself [30].

A study conducted with 41 Italian MS patients found positive effects of the isolated dietary intervention with energy restriction of 500 kcal/day during six months. There was a decrease of 8.5% in the total body mass, in addition to a waist circumference reduction, an improvement of plasma levels of HDL cholesterol, triglycerides, and glucose, and a MS reversal of 37% of the participants [31].

Pimentel et al. [32] conducted a controlled study with 51 Brazilian participants, where 21 participated in a nutrition education program with nutritionists for a 12 months period. The authors reported significant health improvements in the intervention group and, therefore, concluded that nutritional intervention alone is capable of significantly improving all the anthropometric, dietary, and metabolic parameters. These findings corroborate the ones found in this study with Brazilian postmenopausal women.

In the present study, following the proposed intervention, there was a significant reduction in the systolic and diastolic arterial blood pressure in the intervention group. Such reductions were also observed in individuals with MS subjected to a lifestyle change program. The diet denominated Dietary Approach to Stop Hypertension, when associated with a behavioral intervention during six months, seemed to improve the effect of the lifestyle change in 399 North American individuals with MS, more expressively reducing diastolic arterial blood pressure, insulin resistance, and plasma levels of total cholesterol [33].

As for the biochemical analysis performed in this study's intervention group; plasma glucose levels were the only variable that did not suffer a statistically significant reduction, however, we could notice an expressive decrease. The mean glucose levels went from 118.7 mg/dL to 107.4 mg/dL, reducing about 10% of glucose levels found at M0. Cox et al. [34] were able to find reduction of 40% in glucose plasma levels when they recruited 60 Australian men aged 20 to 50 years old that underwent to an energy restriction of 1,000 to 1,500 kcal/day also for a 16 weeks period. According to the authors, interventions in the lifestyle may prove to be important strategies to improve glucose tolerance, reduce insulin resistance, and control type 2 diabetes mellitus.

All participants from the intervention group in our study were encouraged to substitute foods with high for those with low glycemic indexes. Modifying the type of carbohydrate consumed seems to influence the endogenous cholesterol metabolism. A study in Finland, with 74 individuals presenting MS, demonstrated that those submitted to the consumption of whole grains, for a 12 weeks period, presented a reduction of 6 to 10% in the synthesis and increase of 9% in the re-absorption of endogenous cholesterol, which may be clinically favorable to the individuals with MS [35]. The mean reduction of 14 ± 0.7 mg/dL in plasma cholesterol levels found in the present study was statistically significant and may have suffered influence from the modification in the type of carbohydrate consumed.

In 2007, 25 sedentary Colombians with MS and overweight were submitted to four months of a weight loss program which included an energy restriction of 500 kcal/day. There was an increase in insulin sensitivity and a reduction in insulin resistance and in oxidation of the LDL cholesterol. Although no differences were found in HDL cholesterol levels, the alterations presented by the biomarkers of oxidative stress (LDL and HDL cholesterol oxidation, myeloperoxidase and apolipoprotein A) may be beneficial and significant with slight reductions in total body mass, of approximately 5 to 10% [36]. These biomarkers of the inflammatory state are considered good predictors for cardiovascular risk and are directly related to MS and to other complications associated to obesity [37].

In the present study, there was a mean decrease of about 4.3% in total body mass. This weight loss was similar to the ones commonly observed in controlled clinical trials for a long-term treatment for obesity. According to a literature review including five intervention studies, the percentage of weight reduction at the end of the follow up oscillated between 3 and 9%. The authors also took notice of the scarcity of publications involving this type of study design with the Latin American population [38].

Energy restriction imposed on the current study participants was of approximately 500 kcal/day, which represented 22% reduction in daily energy intake according to the diet history collected at the study beginning. The negative energy balance may be more effective on improving the MS components than the body mass decrease that results from it. A study conducted in Israel submitted 12 obese individuals presenting MS to a hypocaloric diet for six months, followed by an isocaloric diet, and another six consecutive weeks of energy restriction. It was observed that during the body mass maintenance phase (isoenergetic diet); the increase in insulin sensitivity was lower than in the other two phases [39].

Diets that are poor in saturated fats and rich in polysaccharides may lead to a moderate weight loss and an improvement in plasma cholesterol levels in individuals with overweight and MS [40]. In the actual study, the diets prescribed to the intervention group participants were. Besides that, the offered nutritional advices stimulated the consumption of low glycemic index carbohydrates and of monounsaturated and polyunsaturated fatty acids. The type of diet offered may cause impact in the individuals' body mass, as well as metabolic and cardiovascular risk [41]. The hypolipidic diet may result in an increase of triglycerides plasma levels and a decrease in HDL cholesterol plasma levels, because it probably offers great amounts of carbohydrates [42], although the opposite (low carbohydrate diets or at least low glycemic index carbohydrate diets) seemed to improve health [43]. On the other hand, according to a review published by Aude et al. [44] regarding dietary interventions in MS patients, the hyperproteic diet may produce adverse effects in the plasma levels of total or LDL cholesterol.

Another research studied the effect of the hypocaloric diet offered for a six months period to 93 insulin resistant women from New Zealand. They concluded that it led to the improvement of MS components in the two studied groups, one with a diet rich in fiber and another with a diet rich in protein and unsaturated fat. And they found a statistically significant reduction of waist circumference [45], as also observed in the present study.

Regarding micronutrients, the control group did not achieve the recommendations of the Dietary Reference Intake [46-48] for any of the calculated minerals. As for the intervention group, at the end of the study, they were consuming a larger amount of calcium (reaching the DRI recommendations for 31 to 50 years age group), but it was not statistically significant. Despite the nonsignificant increase in potassium intake, the recommendations were not reached in either of the two groups, as well as the fiber intake.

When investigating the food frequency questionnaire applied, it was observed that women in the intervention group increased the frequency of habitual consumption of fish, low fat dairy products, leafy vegetables, and fruits and reduced intake of butter, margarine, sweets, soft drinks, and fast food. Williams et al. [17] associated a healthy diet rich in fruits, vegetables, fish, wine, rice, and pasta and poor in fried foods, sausages, and potatoes, with a lower prevalence of MS. Among the other dietary factors inversely related to the MS development, there would be: greater consumption of fruits and vegetables, low intake of sweetened beverages [18]; greater consumption of dairy products in general [49,50]; greater intake of whole grains and fiber [19,20], and greater consumption of foods rich in antioxidants such as retinyl esters, carotenoids, and vitamins C and E [51].

The food groups preferentially consumed can influence total body mass on the long term basis. A Canadian study on changes in dietary pattern was conducted with 248 adults over a six years period. The reported decrease in the consumption of fatty foods and increase consumption of fruits and low fat milk, demonstrated by specific questionnaires, was associated with a lower increase in body mass and adiposity over the study's time [52]. The possible protective effects of dairy products were also inversely proportional to the MS frequency in men, according to a study conducted by means of dietary inquiries with 4,976 individuals [53].

The incorrect eating habits, commonly seen in overweight and obese people, show the need for nutrition education, according to a study with 67 overweight Spanish women who declared an intake of cereals, legumes, fruits, vegetables, and fish in quantities lower than the recommended [54].

The present study has demonstrated that the individualized dietary intervention for 16 weeks may be effective in the control of MS components. The results obtained from this controlled and randomized study showed that effective changes in eating habits incited by a short-term individualized nutritional monitoring promoted significant improvement in all MS components. Therefore, dietary intervention may be a safe strategy for the treatment and reversal of the MS.

The non-pharmacological MS treatment should be encouraged and more controlled epidemiological studies should be conducted to evaluate the long-term benefits of hypocaloric diets in individuals of different age groups.

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References

1. Mclellan KCP, Barbalho SM, Cattalini M, Lerario AC (2007) Type 2 diabetes mellitus, metabolic syndrome and change in lifestyle. Brazilian Journal of Nutrition 20: 515-524.
2. Ntandou G, Delisle H, Agueh V, Fayomi B (2009) Abdominal obesity explains the positive rural-urban gradient in the prevalence of the metabolic syndrome in Benin, West Africa. Nutr Res 29: 180-189.
3. Lerario DD, Gimeno SG, Franco LJ, Iunes M, Ferreira SR (2002) [Weight excess and abdominal fat in the metabolic syndrome among Japanese-Brazilians]. Rev Saude Publica 36: 4-11.
4. Alberti KG, Eckel RH, Grundy SM, Zimmet PZ, Cleeman JI, et al. (2009) Harmonizing the metabolic syndrome: a joint interim statement of the International Diabetes Federation Task Force on Epidemiology and Prevention; National Heart, Lung, and Blood Institute; American Heart Association; World Heart Federation; International Atherosclerosis Society; and International Association for the Study of Obesity. Circulation 120: 1640-1645.
5. Carrasco Naranjo F (2006) [Metabolic syndrome: More definitions for a new disease?]. Nutr Hosp 21: 222-223.
6. Ribeiro Filho FF, Mariosa LS, Ferreira SR, Zanella MT (2006) [Visceral fat and metabolic syndrome: more than a simple association]. Arq Bras Endocrinol Metabol 50: 230-238.
7. Ford ES, Giles WH (2003) A comparison of the prevalence of the metabolic syndrome using two proposed definitions. Diabetes Care 26: 575-581.
8. Silveira IL, Maranhão TM, Azevedo GD (2007) Metabolic syndrome in postmenopausal women: higher prevalence in the Northeastern Region of Brazil than in other Latin American countries and the influence of obesity and socioeconomic factors. Climacteric 10: 438-439.
9. Petri Nahas EA, Padoani NP, Nahas-Neto J, Orsatti FL, Tardivo AP, et al. (2009) Metabolic syndrome and its associated risk factors in Brazilian postmenopausal women. Climacteric 12: 431-438.
10. Figueiredo Neto JA, Figuerêdo ED, Barbosa JB, Barbosa Fde F, Costa GR, et al. (2010) Metabolic syndrome and menopause: cross-sectional study in gynecology clinic. Arq Bras Cardiol 95: 339-345.
11. Ruan X, Jin J, Hua L, Liu Y, Wang J, et al. (2010) The prevalence of metabolic syndrome in Chinese postmenopausal women and the optimum body composition indices to predict it. Menopause 17: 566-570.
12. Cicero AFG, Dormi A, D'Addato S, Gaddi AV, Borghi C (2010) Long-term effect of a dietary education program on postmenopausal cardiovascular risk and metabolic syndrome: the Brisighella Heart Study. Journal of Women`s Health 19: 133-137.
13. Cho GJ, Lee JH, Park HT, Shin JH, Hong SC, et al. (2008) Postmenopausal status according to years since menopause as an independent risk factor for the metabolic syndrome. Menopause 15: 524-529.
14. Feldeisen SE1, Tucker KL (2007) Nutritional strategies in the prevention and treatment of metabolic syndrome. Appl Physiol Nutr Metab 32: 46-60.
15. Mann JI (2006) Nutrition recommendations for the treatment and prevention of type 2 diabetes and the metabolic syndrome: an evidenced-based review. Nutr Rev 64: 422-427.
16. Esmaillzadeh A, Kimiagar M, Mehrabi Y, Azadbakht L, Hu FB, et al. (2007) Dietary patterns, insulin resistance, and prevalence of the metabolic syndrome in women. Am J Clin Nutr 85: 910-918.
17. Williams DE, Prevost AT, Whichelow MJ, Cox BD, Day NE, et al. (2000) A cross-sectional study of dietary patterns with glucose intolerance and other features of the metabolic syndrome. Br J Nutr 83: 257-266.
18. Yoo S, Nicklas T, Baranowski T, Zakeri IF, Yang SJ, et al. (2004) Comparison of dietary intakes associated with metabolic syndrome risk factors in young adults: the Bogalusa Heart Study. Am J Clin Nutr 80: 841-848.
19. McKeown NM, Meigs JB, Liu S, Saltzman E, Wilson PW, et al. (2004) Carbohydrate nutrition, insulin resistance, and the prevalence of the

metabolic syndrome in the Framingham Offspring Cohort. Diabetes Care 27: 538-546.

20. Sahyoun NR, Jacques PF, Zhang XL, Juan W, McKeown NM (2006) Whole-grain intake is inversely associated with the metabolic syndrome and mortality in older adults. Am J Clin Nutr 83: 124-131.
21. Cho YA, Kim J, Cho ER, Shin A (2011) Dietary patterns and the prevalence of metabolic syndrome in Korean women. Nutr Metab Cardiovasc Dis 21: 893-900.
22. Steemburgo T1, Dall'Alba V, Gross JL, Azevedo MJ (2007) [Dietary factors and metabolic syndrome]. Arq Bras Endocrinol Metabol 51: 1425-1433.
23. Norton K, Olds T (1996) Anthropometric: A textbook of body measurements for sport and health courses. University of New South Wales Press, Sydney.
24. Brazilian Society of Cardiology (2005) I Brazilian Guideline for Diagnosis and Treatment of Metabolic Syndrome. Brazilian Archives of Cardiology 84: 15-24.
25. Monteiro JBR, Esteves EA (1999) Diet Pro: Support system to the assessment and prescription of diets. (2ndedn) Agromídia Software, Federal University of Viçosa, Minas Gerais.
26. Pinheiro ABV, Lacerda EMA, Benzecry EH, Gomes MCS, Costa VM (1994) Table for evaluation of food consumption in household measures. (2ndedn) Federal University of Rio de Janeiro, Rio de Janeiro.
27. Brazilian Association for the Study of Obesity and Metabolic Syndrome (2010) Brazilian Guidelines on Obesity. ABESO, São Paulo.
28. (2000) Obesity: preventing and managing the global epidemic. Report of a WHO consultation. World Health Organ Tech Rep Ser 894: i-xii, 1-253.
29. Anderssen SA, Carroll S, Urdal P, Holme I (2007) Combined diet and exercise intervention reverses the metabolic syndrome in middle-aged males: results from the Oslo Diet and Exercise Study. Scand J Med Sci Sports 17: 687-695.
30. Kukkonen-Harjula KT, Borg PT, Nenonen AM, Fogelholm MG (2005) Effects of a weight maintenance program with or without exercise on the metabolic syndrome: a randomized trial in obese men. Prev Med 41: 784-790.
31. Muzio F, Mondazzi L, Sommariva D, Branchi A (2005) Long-term effects of low-calorie diet on the metabolic syndrome in obese nondiabetic patients. Diabetes Care 28: 1485-1486.
32. Pimentel GD, Portero-McLellan KC, Oliveira EP, Spada AP, Oshiiwa M, et al. (2010) Long-term nutrition education reduces several risk factors for type 2 diabetes mellitus in Brazilians with impaired glucose tolerance. Nutrition Research 30: 186-190.
33. Lien LF, Brown AJ, Ard JD, Loria C, Erlinger TP, et al. (2007) Effects of PREMIER lifestyle modifications on participants with and without the metabolic syndrome. Hypertension 50: 609-616.
34. Cox KL, Burke V, Morton AR, Beilin LJ, Puddey IB (2004) Independent and additive effects of energy restriction and exercise on glucose and insulin concentrations in sedentary overweight men. Am J Clin Nutr 80: 308-316.
35. Hallikainen M, Toppinen L, Mykkänen H, Ågren JJ, Laaksonen DE, et al. (2006) Interaction between cholesterol and glucose metabolism during dietary carbohydrate modification in subjects with the metabolic syndrome. American Journal of Clinical Nutrition 84: 1385-1392.
36. Rector RS, Warner SO, Liu Y, Hinton P, Sun GY, et al. (2007) Exercise and diet induced weight loss improves measures of oxidative stress and insulin sensitivity in adults with characteristics of the metabolic syndrome. American Journal of Physiology: Endocrinology and Metabolism 293: 500-506.
37. Zulet MA, Puchau B, Navarro C, Martí A, Martínez JA (2007) [Inflammatory biomarkers: the link between obesity and associated pathologies]. Nutr Hosp 22: 511-527.
38. Márquez-Ibáñez B, Armendáriz-Anguiano AL, Bacardí-Gascón M, Jiménez-Cruz A (2008) [Review of controled clinical trials of behavioral treatment for obesity]. Nutr Hosp 23: 1-5.
39. Assali AR, Ganor A, Beigel Y, Shafer Z, Hershcovici T, et al. (2001) Insulin resistance in obesity: body-weight or energy balance? J Endocrinol 171: 293-298.
40. Rodríguez-Rodríguez E, Ortega RM, López-Sobaler AM, Andrés P, Aparicio A, et al. (2007) Restricted-energy diets rich in vegetables or cereals improve cardiovascular risk factors in overweight/obese women. Nutrition Research 27: 313-320.
41. Irazusta A, Hoyos I, Irazusta J, Ruiz F, Díaz E, et al. (2007) Increased cardiovascular risk associated with poor nutritional habits in first-year university students. Nutrition Research 27: 387-394.
42. Poppitt SD, Keogh GF, Prentice AM, Williams DE, Sonnemans HM, et al. (2002) Long-term effects of ad libitum low-fat, high-carbohydrate diets on body weight and serum lipids in overweight subjects with metabolic syndrome. Am J Clin Nutr 75: 11-20.
43. Grieb P, KÅ‚apciÅ„ska B, Smol E, Pilis T, Pilis W, et al. (2008) Long-term consumption of a carbohydrate-restricted diet does not induce deleterious metabolic effects. Nutr Res 28: 825-833.
44. Aude YW, Mego P, Mehta JL (2004) Metabolic syndrome: dietary interventions. Curr Opin Cardiol 19: 473-479.
45. McAuley KA, Smith KJ, Taylor RW, McLay RT, Williams SM, et al. (2006) Long-term effects of popular dietary approaches on weight loss and features of insulin resistance. Int J Obes (Lond) 30: 342-349.
46. Food and Nutrition Board / Institute Of Medicine (2011) Dietary Reference Intakes for Calcium and Vitamin D. National Academies Press, Whashington DC.
47. Food and Nutrition Board / Institute Of Medicine (2004) Dietary reference intake for water, potassium, sodium, chloride and sulfate. National Academies Press, Whashington DC.
48. Food and Nutrition Board / Institute Of Medicine (2001) Dietary reference intake for vitamin A, vitamin K, arsenic, boron, chromium, cooper, iodine, iron, manganese, molybdenium, nickel, silicon, vanadium and zinc. National Academies Press, Whashington DC.
49. Pereira MA, Jacobs DR Jr, Van Horn L, Slattery ML, Kartashov AI, et al. (2002) Dairy consumption, obesity, and the insulin resistance syndrome in young adults: the CARDIA Study. JAMA 287: 2081-2089.
50. Azadbakht L, Mirmiran P, Esmaillzadeh A, Azizi T, Azizi F (2005) Beneficial effects of a Dietary Approaches to Stop Hypertension eating plan on features of the metabolic syndrome. Diabetes Care 28: 2823-2831.
51. Ford ES, Mokdad AH, Giles WH, Brown DW (2003) The metabolic syndrome and antioxidant concentrations: findings from the Third National Health and Nutrition Examination Survey. Diabetes 52: 2346-2352.
52. Drapeau V, Després JP, Bouchard C, Allard L, Fournier G, et al. (2004) Modifications in food-group consumption are related to long-term body-weight changes. Am J Clin Nutr 80: 29-37.
53. Mennen LI, Lafay L, Feskens EJM, Novak M, Lépinay P, et al. (2000) Possible protective effect of Bread and dairy products on the risk of the metabolic syndrome. Nutrition Research, 20: 335-347.
54. Rodríguez-Rodríguez E, Perea JM, Bermejo LM, Marín-Arias L, López-Sobaler AM, et al. (2007) [Dietary habits and their relationship with the knowledge on the concept of a balanced diet in a group of young women with overweight/obesity]. Nutr Hosp 22: 654-660.