Original Article
CLINICAL TRIALS: BEHAVIOR, PHARMACOTHERAPY, DEVICES, SURGERY

Obesity

Regular Intake of High-Oleic Peanuts Improves Fat Oxidation and Body Composition in Overweight/Obese Men Pursuing a Energy-Restricted Diet

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Objective: Evaluate the effect of high-oleic and conventional peanuts within a hypocaloric-diet on energy metabolism and body composition.
Methods: This 4-week randomized clinical trial included males with BMI of 29.7 ± 2.4 kg m^{-2} and aged between 18 and 50 years. Participants were assigned to the groups: control (CT, $n = 22$) that followed a hypocaloric-diet; conventional peanuts (CVP, $n = 22$) or high-oleic peanuts (HOP, $n = 21$) that received the hypocaloric-diet including (not adding) 56 g day^{-1} of peanuts. Glucose, fat oxidation, and body fatness and lean mass were the main outcomes.
Results: Body weight and composition did not differ between groups. However, within group total body fat (kg) reduced with CVP and HOP, with a significant decrease in body fat percentage in HOP. While total lean mass (kg) decreased in CT, total lean mass (%) increased in HOP. Truncal lean mass decreased in the CT. At baseline, HOP had greater postprandial fat oxidation than the CVP. After 4-weeks, fasting fat oxidation increased in CVP and HOP. Fat oxidation increased in CT and HOP during the 200 min after meal intake compared to the fasting condition.
Conclusion: Regular peanut consumption, especially the high-oleic type, within a hypocaloric-diet increased fat oxidation and reduced body fatness in overweigh and obese men.

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Introduction

Despite their high-energy density, peanuts may aid in the prevention and management of obesity and it's metabolic complications (1-3). The high protein (≈24%) and dietary fiber (≈8%) contents of peanuts reportedly moderate appetite (1). Additionally, peanuts are a rich source of monounsaturated fatty acids (MUFA) that may increase body fat oxidation (2,4). The main MUFA present in peanuts is oleic acid which is resistant to lipid peroxidation during storage (5-7). Thus, there is interest in producing and promoting the consumption of high-oleic peanuts to improve shelf-life without promoting the risk of obesity and its complications (6). High oleic acid peanuts may also reduce the negative metabolic effects of dietary saturated fatty acids (SFA) (8,9).

O'Byrne et al (1997) demonstrated that high-oleic peanuts improved serum lipoprotein profiles (10). However, the effects of regular intake of high-oleic peanuts on substrate oxidation, body composition, and appetite have not been studied. This trial aimed to compare the effects of daily consumption of high-oleic and conventional peanuts on energy metabolism, appetite, fat oxidation, and body composition in overweight/obese men.

Methods

Participants

One hundred and fifty men were recruited. Eligibility included age between 18 and 50 years, body mass index (BMI) ranging from 26 to 35 kg m^{-2} and stable weight (±3 kg) during the previous 3 months. Individuals with acute diseases and/or eating disorders or any chronic disease other than obesity, were not included. Other exclusion criteria were the use of medications (e.g., β-blockers or diuretics, antibiotics, anti-inflammatory agents) that might affect study outcomes over the 3 months prior to study initiation and high alcohol intake (>168 g $week^{-1}$). The study was approved by the Ethical Committee on Human Research of the Federal University of Viçosa (number: 185/2011). All participants provided a written informed consent.

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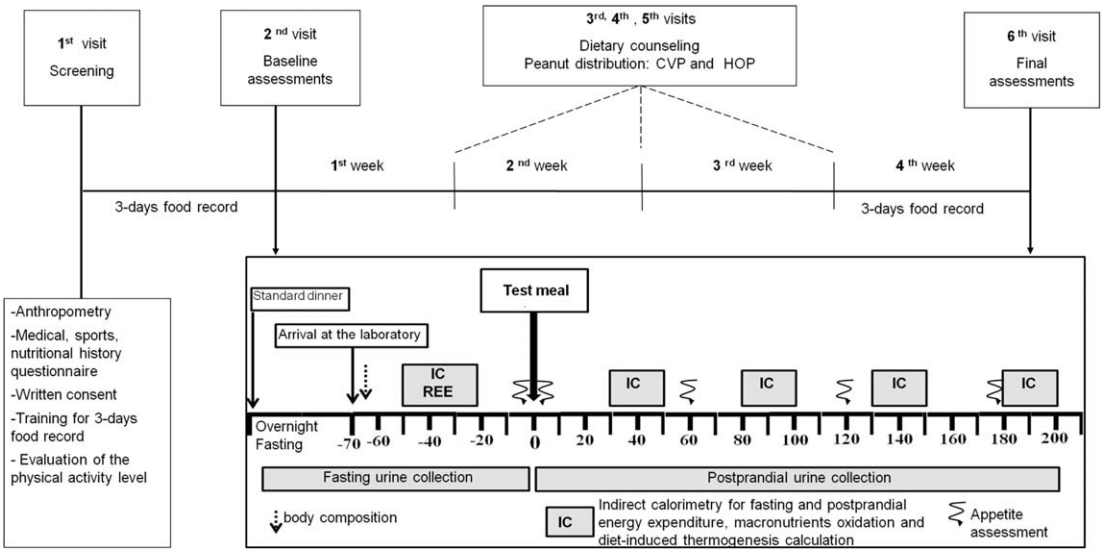

Figure 1 Experimental design. CVP, conventional peanuts group; HOP, high-oleic peanuts group. [Color figure can be viewed in the online issue, which is available at wileyonlinelibrary.com.]

Study design

This was a 4-week randomized, parallel-arm clinical trial. Participants were assigned to groups: control (CT, $n = 22$); conventional peanuts (CVP, $n = 22$); high-oleic peanuts (HOP, $n = 21$). They received a standard dinner the night prior to assessments. After overnight fasting, participants consumed a test-meal within 15 min. Measurements included anthropometry, body composition, appetite, food intake, and energy metabolism. During the next 4 weeks, participants followed a hypocaloric-diet. They were asked to maintain their customary physical activity level. At the end of the intervention period, all measurements were repeated (Figure 1).

Dietary intervention

Each participant's daily energy requirement was calculated, then 250 kcal day^{-1} were subtracted for the dietary prescription. All experimental diets provided 15% of energy from protein, 30% from fat, and 55% from carbohydrate.

The CT group's diet did not include any test food. The CVP and HOP groups' prescriptions were calculated including a daily portion of 56 g of conventional or high-oleic peanuts, respectively. Participants were free to eat the peanut portion any time of the day, yet, they were asked to consume the whole portion at once. The energy provided by peanuts in the CVP and HOP groups was offset in the balance of the diet, thus, total energy prescription was comparable on all three treatments. Because the dietary intervention was in a free-living condition, participants were instructed to use an exchange-based self-selected food list.

Test meal and peanuts

The night prior to each assessment, participants consumed a standard dinner that consisted of instant plain noodles (Nissin®) with grated parmesan cheese, and grape juice (731 kcal; 65.1% from carbohydrate, 7.6% from protein, and 28.3% from fat). This meal was intended to reduce hepatic glycogen oxidation during the fasting state at night, and diet induced thermogenesis (DIT) (11).

After fasting assessments, the test meal was offered according to their group assignment. Each test meal provided 25% of each participant's daily energy requirement. The meals consisted of a strawberry flavored milkshake and 56 g of unpeeled roasted peanuts (conventional, high-oleic) or control biscuits. They had the same volume, energy density, and provided 35% of energy from carbohydrate, 16% from protein, and 49% from fat.

Conventional and high-oleic peanuts were prepared in dry heat then, 56 g of peanuts were vacuum packed and stored. This portion of conventional and high-oleic peanuts contains, respectively, 13.6 and 12.8 g of carbohydrates, 16.8 and16.3 g of proteins, 24.0 and 24.7 g of fat, and 5.0 and 5.5 g of dietary fiber (0.2 and 0.7g of soluble; 4.8 and 4.8 g of insoluble). Oleic fatty acid represents 51.0% of total fat in conventional peanuts and 81.5% in high-oleic peanuts. Control biscuits were developed in the laboratory to offer similar amounts of macronutrients and fiber, and energy density as the peanuts. Its composition was also analyzed. Its ingredients consisted of eggs, whey protein supplement, whole wheat flour, margarine, hydrogenated vegetable shortening, soybean oil, dietary fiber, sesame seed, wheat bran, salt, and powdered yeast.

The milkshake was prepared just before its consumption and consisted of water, ice, whole milk powder, whey protein supplement, soybean oil, and Nesquik® strawberry powder. This milkshake was prepared to complete the energy and macronutrient consistent with the macronutrient proportion previously described.

Dietary intake assessment

Participants provided two 3-day food records (two nonconsecutive week days and one weekend day), before the baseline assessments

and during the fourth week of the study. Food records were analyzed using Dietpro software (version 5.2i).

Appetite assessment

One hundred millimeter visual analog scales were used for appetite assessment. These scales include words anchored at each end, expressing the most positive and negative rating, to assess hunger, satiety, fullness, and prospective food consumption (12). Appetite ratings were recorded immediately before and after test meal consumption, and then hourly for 3 h (Figure 1). Results were expressed as the positive incremental area under the curve ($_{pi}$AUC) of the scores.

Measurements and calculations

All measurements were taken at baseline and after 4-weeks. Participants were instructed not to consume caffeine or alcohol, to refrain from non-customary physical activity, and to maintain a regular sleep-wake schedule (8 h night^{-1}) over the 72 h before assessments. Participants fasted overnight. Body weight, height, and waist and hip circumferences were assessed. Body composition was assessed by dual-energy X-ray absorptiometry (DEXA) (Lunar Prodigy Advance DXA System, GE Lunar) in a subsample (75%; CT $n = 12$; CVP $n = 17$; HOP $n = 18$) due to the equipment schedule availability. The DEXA analyses provided total and regional body fatness, including truncal, android, and gynoid composition. The neck, chest, abdominal, and pelvic areas are included in truncal analyses. The area between the ribs and the pelvis, and is totally enclosed by the trunk region, was considered the android area while the gynoid region includes the hips and upper thighs, and overlaps both the leg and truncal regions (13).

Respiratory gas exchange measurements were performed by indirect calorimetery using a ventilated respiratory canopy (Deltatrac II; Datex Instrumentarium Corporation) in full compliance with the manufacturer guidelines. REE, respiratory quotient (RQ), and substrate oxidation were measured over 30 min under fasting conditions. To evaluate the postprandial metabolic rate for DIT and substrate oxidation, this measurement was performed four times after test meal consumption during 20 min with 30-min intervals, over 200 min (Figure 1). DIT was calculated as the incremental increase in energy expenditure above REE, expressed as percentage of the test meal calories (14). To calculate substrate oxidation, urinary nitrogen was analyzed by the Kjeldahl method (15) in timed urine samples, which were collected after overnight fasting and over 200 min after meal ingestion. Fasting and postprandial substrate oxidations were calculated using standard equations (16) and were expressed as mg per minute.

Statistical analysis

Power analysis was calculated by SAS (Version 9.2) using body fat percentage as the primary outcome. It indicated that a sample of 12 per group would permit the detection of a 5% variance in body fat percentage with 99% power at the 5% level of probability.

The $_{pi}$AUC of appetite scores were calculated using GraphPad Prism (Version 5). Statistics were performed using SAS. The normality and homogeneity of variance were tested by Shapiro-Wilk and Levene tests, respectively. Results are presented as mean ± sem. Variables and their changes (Δ = Final – Baseline) were compared between groups using one-way ANOVA or Kruskal–Wallis followed by Tukey's or Dunn's test, respectively. Two-way repeated-measures ANOVA was applied to test the differences between groups throughout the test days for postprandial metabolic and substrate oxidation with treatment and time as repeated factors. Changes (Δ) in all variables were compared within each group by paired *t* test or Wilcoxon test.

Results

Seventy six participants were randomized to the trial. Seven participants (9.2%) withdrew for personal reasons. Sixty five were included in the analyses due to missing data. Baseline weight and body composition did not differ between groups ($P > 0.05$) (Table 1).

Baseline fasting REE, RQ, and oxidation of carbohydrate and fat were not statistically different between groups. Furthermore, there was no difference between groups for physical activity levels ($P = 0.7650$) or daily energy requirement ($P = 0.8760$). Habitual dietary intake as well as the energy balance, calculated as the difference between caloric intake and total energy expenditure, was not different between groups ($P > 0.05$) (data not shown).

Changes in anthropometry and body composition are presented in Table 2. Body weight, BMI, and waist and hip circumferences were significantly reduced in all groups. The percentage of weight loss did not differ between groups (2.3% ± 0.4% in CT, 1.6% ± 0.3% in CVP, and 1.9% ± 0.4% in HOP). Energy balance was negative in all groups with no statistical differences between them (data not shown), which is consistent with the weight loss results.

Changes in the following variables were not different between groups. However, within group, total body fat mass (kg) was reduced in the CVP and HOP groups ($P < 0.05$). A significant decrease in total body fat percentage was verified only in the HOP group (Figure 2). While a significant decrease in total fat free and lean mass was documented in CT, a significant increase in total lean mass percentage occurred in the HOP group. In the CT group, 62.9% of the total body weight loss was in fat free mass (FFM) while in the CVP group 69.9% of total weight loss was in fat mass (Figure 2). Furthermore, the HOP group had an 86.3% of fat mass loss with a slight FFM loss in relation to total mass lost.

Truncal fat free and lean mass ($P < 0.05$) decreased in the CT group. A significant reduction in gynoid fat percentage and an increase in gynoid lean mass percentage were observed only in the HOP group. Android mass was not significantly changed in any group.

Subjects did not change their physical activity level in comparison to baseline (CT: $P = 1.0$; CVP: $P = 0.73$; HOP: $P = 0.84$), and no difference between groups was measured ($P = 0.97$).

Between groups assessments of energy metabolism variables, as well as changes compared to baseline, were not significantly different. Within group analysis showed that, the REE was not significantly changed (Table 3), but there was a significant decrement in fasting RQ and in carbohydrate oxidation in the CVP group, but not in CT and HOP groups. Indeed, fasting fat oxidation increased significantly in the CVP and HOP groups (Table 3).

TABLE 1 Participants characteristics according to the experimental group at baseline

	CT ($n = 22$)	CVP ($n = 22$)	HOP ($n = 21$)
Age (years)	27.4 ± 1.6	28.0 ± 1.5	26.8 ± 1.9
Body weight (kg)	94.5 ± 2.5	93.4 ± 2.2	95.1 ± 2.4
BMI (kg m^{-2})	29.7 ± 0.6	29.5 ± 0.4	29.9 ± 0.6
Waist (cm)	102.3 ± 2.0	100.9 ± 1.3	101.7 ± 1.8
Hip (cm)	109.1 ± 1.1	108.0 ± 1.1	109.4 ± 1.1
Waist-hip ratio	0.94 ± 0.01	0.93 ± 0.01	0.93 ± 0.01
Body composition (DEXA)	CT ($n = 12$)	CVP ($n = 17$)	HOP ($n = 18$)
Total body fat percentage (%)	33.4 ± 0.9	31.1 ± 1.0	33.5 ± 1.3
Total fat mass (kg)	32.9 ± 1.3	29.3 ± 1.4	31.6 ± 1.6
Total fat free mass (kg)	65.0 ± 0.9	64.1 ± 1.1	62.1 ± 1.3
Total lean mass percentage (%)	62.9 ± 0.9	65.1 ± 1.0	62.9 ± 1.2
Total lean mass (kg)	61.4 ± 0.9	60.6 ± 1.0	58.7 ± 1.2
Truncal fat percentage (%)	36.7 ± 1.4	34.5 ± 1.0	36.8 ± 1.5
Truncal fat mass (kg)	17.1 ± 1.0	14.8 ± 0.7	16.3 ± 1.1
Truncal fat free mass (kg)	28.8 ± 0.6	27.7 ± 0.5	27.1 ± 0.8
Truncal lean mass percentage (%)	27.7 ± 0.6	26.6 ± 0.5	26.1 ± 0.7
Truncal lean mass (kg)	60.7 ± 1.4	62.9 ± 1.0	60.8 ± 1.6
Gynoid fat percentage (%)	38.6 ± 1.0	37.4 ± 1.2	39.4 ± 1.3
Gynoid fat mass (kg)	5.7 ± 0.2	5.0 ± 0.3	5.1 ± 0.3
Gynoid fat free mass (kg)	9.0 ± 0.2	8.3 ± 0.2	8.2 ± 0.2
Gynoid lean mass percentage (%)	58.6 ± 1.0	59.3 ± 1.2	57.6 ± 1.4
Gynoid lean mass (kg)	8.6 ± 0.2	7.8 ± 0.2	7.8 ± 0.2
Android fat percentage (%)	36.7 ± 1.8	33.4 ± 1.0	35.8 ± 1.9
Android fat mass (kg)	2.5 ± 0.2	2.1 ± 0.1	2.3 ± 0.2
Android fat free mass (kg)	4.2 ± 0.1	4.1 ± 0.1	4.0 ± 0.1
Android lean mass percentage (%)	4.2 ± 0.1	4.0 ± 0.1	4.0 ± 0.1
Android lean mass (kg)	62.3 ± 1.7	65.4 ± 1.0	63.0 ± 1.9

Values are mean ± SEM. There was no difference between groups ($P > 0.05$; ANOVA or Kruskal–Wallis). CT, control group; CVP, conventional peanuts group; HOP, high-oleic peanuts group; BMI, body mass index; DEXA, dual-energy X-ray absorptiometry.

Baseline DIT was significantly higher after high-oleic peanuts intake (3.57% ± 0.26%) compared to conventional peanuts (2.60% ± 0.17%). However, there was no significant difference in DIT between the CT (3.21% ± 0.25%) and HOP groups. Conversely, DIT assessed after 4-weeks did not differ significantly between groups (CT: 3.07% ± 0.43%; CVP: 2.38% ± 0.24%; HOP: 2.97% ± 0.21%). Changes within groups were not significant.

As expected, in all groups, on both test days, energy expenditure measured at 50, 100, 150, and 200 min after meal intake was significantly different from fasting values. It was significantly increased at 50 and 100 min after test meal intake compared to the fasting condition. There was no group-time interaction for carbohydrate oxidation. For fat oxidation, different response were verified at baseline compared to final assessments. At baseline, fat oxidation was increased significantly at 200 min after test meal in all groups compared to the fasting condition. Significant increases were observed 200 min after test meal intake in the HOP and CT groups at the final assessments. This did not occur in the CVP group ($P = 0.45$). At baseline, the HOP group had greater fat oxidation than the CVP group after 200 min of meal intake ($P = 0.04$).

The results shown in Figure 3 represent the mean integrated value for appetite scores calculated as the $_{pi}$AUC over the time at baseline and at the final assessments. At baseline, there was no difference between groups for appetite after test meal intake. At the final assessment, there was a significant decrease in "fullness" in the HOP group compared to baseline ($P = 0.013$), but there was no difference between groups. Participants from the CT group reported greater satiety at the final assessment compared to the CVP group ($P = 0.03$). Because prospective food consumption and hunger ratings are reversed scored compared to satiety, taller bars represent lower sensation levels. Thus, only the CT group rated hunger lower at the final assessment compared to baseline ($P = 0.03$). No changes of prospective food consumption were observed.

In the prescribed diet, peanuts contributed a mean of 12.7% of total energy, 2.5% of total carbohydrate, 16.0% of total protein, and 30.3% of total fat. Overall, the prescribed energy restriction represented 8.3% of participants' daily energy requirement. However, dietary data from the fourth week showed that participants in the CT group experienced greater energy restriction (28.4% ± 3.2%) than did the CVP and HOP participants (15.7% ± 4.3% and

TABLE 2 Changes (Δ) in body composition variable after 4-weeks of intervention

	CT ($n = 22$)	CVP ($n = 22$)	HOP ($n = 21$)	*P* value
Body weight (kg)	-2.24 ± 0.35[a]	-1.46 ± 0.29[a]	-1.70 ± 0.33[a]	0.2232
BMI (kg m^{-2})	-0.70 ± 0.11[a]	-0.47 ± 0.09[a]	-0.55 ± 0.10[a]	0.2232
Waist (cm)	-2.06 ± 0.30[a]	-2.00 ± 0.40[a]	-1.58 ± 0.34[a]	0.9057
Hip (cm)	-1.37 ± 0.26[a]	-1.05 ± 0.40[a]	-1.14 ± 0.28[a]	0.7654
Waist-hip ratio	-0.01 ± 0.00	-0.01 ± 0.00	0.00 ± 0.00	0.7691
Body composition (DEXA)	CT (n=12)	CVP (n=17)	HOP ($n = 18$)	*P* value
Total body fat percentage (%)	-0.06 ± 0.28	-0.64 ± 0.34	-0.97 ± 0.31[a]	0.2547
Total fat mass (kg)	-0.78 ± 0.32	-1.02 ± 0.33[a]	-1.39 ± 0.29[a]	0.5138
Total fat free mass (kg)	-1.32 ± 0.28[a]	-0.44 ± 0.37	-0.22 ± 0.31	0.1465
Total lean mass percentage (%)	-0.03 ± 0.30	0.60 ± 0.37	0.94 ± 0.33[a]	0.2582
Total lean mass (kg)	-1.33 ± 0.30[a]	-0.42 ± 0.39	-0.19 ± 0.33	0.1566
Truncal fat percentage (%)	-0.08 ± 0.58	-0.88 ± 0.64	-1.03 ± 0.60	0.6504
Truncal fat mass (kg)	-0.57 ± 0.41	-0.43 ± 0.41	-0.60 ± 0.40	0.9617
Truncal fat free mass (kg)	-0.76 ± 0.20[a]	0.04 ± 0.30	0.19 ± 0.27	0.1091
Truncal lean mass percentage (%)	-0.03 ± 0.63	0.89 ± 0.70	1.08 ± 0.61	0.5932
Truncal lean mass (kg)	-0.77 ± 0.21[a]	0.05 ± 0.30	0.19 ± 0.27	0.1077
Gynoid fat percentage (%)	-0.13 ± 0.34	-0.52 ± 0.46	-1.61 ± 0.56[a]	0.1612
Gynoid fat mass (kg)	-0.17 ± 0.06	-0.11 ± 0.09	0.12 ± 0.26	0.9530[b]
Gynoid fat free mass (kg)	0.04 ± 0.36	0.55 ± 0.49	1.71 ± 0.61	0.2097
Gynoid lean mass percentage (%)	-0.23 ± 0.13	-0.03 ± 0.15	0.24 ± 0.17[a]	0.1456
Gynoid lean mass (kg)	-0.23 ± 0.13	-0.04 ± 0.15	0.23 ± 0.16	0.2023
Android fat percentage (%)	-0.60 ± 0.83	-0.88 ± 1.14	-1.28 ± 0.97	0.9239
Android fat mass (kg)	-0.09 ± 0.09	-0.07 ± 0.09	-0.08 ± 0.06	0.9804
Android fat free mass (kg)	-0.06 ± 0.04	-0.02 ± 0.05	0.04 ± 0.04	0.4869
Android lean mass percentage (%)	0.48 ± 0.85	0.92 ± 1.14	1.36 ± 0.98	0.8792
Android lean mass (kg)	-0.06 ± 0.05	-0.02 ± 0.05	0.04 ± 0.05	0.4300

Values are mean ± SEM. *P* value column refer to differences between groups (ANOVA or Kruskal–Wallis test. There was no difference between group ($P > 0.05$).
[a]Significant difference between baseline and final assessments within group ($P < 0.05$; paired *t* test or Wilcoxon test). CT, control group; CVP, conventional peanuts group; HOP, high-oleic peanuts group; BMI, body mass index; DEXA, dual-energy X-ray absorptiometry.
[b]Kruskal–Wallis test.

15.1% ± 4.5%, respectively) ($P < 0.05$). Energy intake, as well as dietary fiber, did not differ between groups (data not shown).

The HOP group intake of total fat (g) was significantly higher (103.4 ± 10.1 g) than in the CT group (70.2 ± 5.5 g) ($P < 0.01$). The percentage of total fat in relation to total energy intake was significantly lower in the CT group (28.0% ± 0.8%) than in other groups (CVP: 34.5% ± 0.9%; HOP: 35.5% ± 1.4%). The percentage of SFA in relation to total energy intake was not different between groups. However, the HOP group had higher intake of MUFA than the other groups ($P < 0.01$) while the CVP group showed higher intake of polyunsaturated fatty acids (PUFA) compared to the HOP and CT ($P = 0.01$) groups. The CT group had a higher intake of carbohydrate, in percentage, than the other groups ($P < 0.01$).

Discussion

Although the potential metabolic benefits of peanuts and other nuts have been recognized, few studies have include nuts in weight-loss regimens (17). In a recent meta-analyses, a mean body weight reduction of 2.61 kg ($P > 0.05$) was verified in energy-restricted nut-enriched diets (18). A hypocaloric moderate-fat diet that included convention peanuts (whole, butter, and oil) significantly reduced body weight but with no difference compared to a hypocaloric low-fat peanut-free diet (19).

O'Byrne et al. reported that daily intake of high-oleic peanuts within a hypocaloric-diet for 6 months led to significant weight loss (3.6 kg), while body weight was unchanged in their control group (10). In that study, participants from both groups restricted energy intake similarly (10). In the present trial, weight loss was also observed in the control group, but energy restriction was similar in all groups. Although the recommended energy prescription was the same in all groups, the CT group actually consumed less energy than the CVP and HOP groups. Nevertheless, body weight was significantly and similarly reduced in all groups. Thus, the groups consuming peanuts lost more weight relative to the level of their energy restriction. Indeed, by reducing ≈550 kcal day^{-1}. An expected weight loss of 2.0 kg in 4 weeks, which was close to the 2.2 kg found after intervention. The HOP group had a restriction of ≈215 kcal day^{-1} in relation to habitual intake, which leads to a predicted a weight loss of 0.8 kg, but the observed decrement was 1.7 kg. In the CVP group, a reduction ≈275 kcal day^{-1} would theoretically lead to 1.0 kg of weight reduction, but they had a mean weight loss of 1.5 kg.

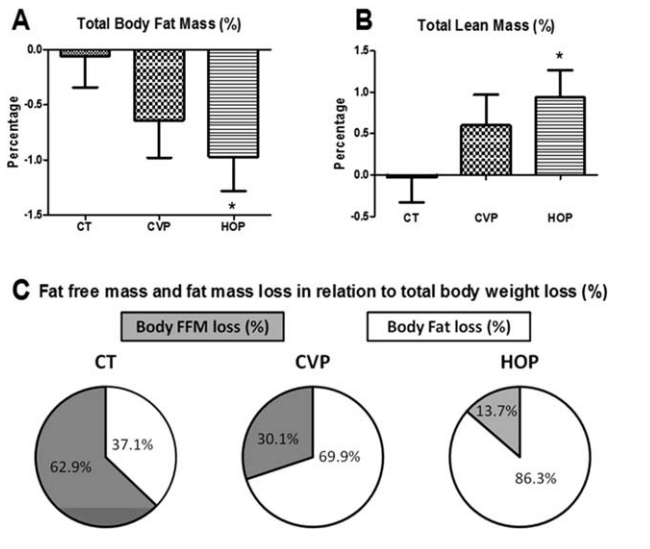

Figure 2 Mean (±SEM) changes in total body fat percentage (**A**), total lean mass percentage (**B**), and in fat free mass and fat mass loss in relation to total body weight loss (%) after intervention (**C**). There was no difference between groups ($P > 0.05$; ANOVA or Kruskal–Wallis). *Significant difference between baseline and final assessments within group ($P < 0.05$; paired *t* test or Wilcoxon test). CT, control group; CVP, conventional peanuts group; HOP, high-oleic peanuts group.

Four mechanisms that have been identified as contributing to the effects of peanut consumption on energy-balance warrant consideration. The first is that peanuts hold high satiety value since they are a rich source of fiber and protein (1,20,21). In this trial, there were no significant differences in appetitive ratings between groups. A recent study reported that conventional peanut consumption lead to greater satiety compared to control treatment after 1 h of its intake (21). Given the groups consuming peanuts ingested less energy, this suggests the inclusion of peanuts in the diet did enhance satiety.

Second, the energy contained in peanuts is not fully bioaccessible, thus they yield less than the predicted amount of energy (22-24). This occurs due to the encapsulation of intracellular fat by cell walls that are resistant to enzymatic and microbial degradation in the gastrointestinal tract. Thus, intact cotyledon cells are lost in the feces (22,23). The magnitude of energy lost by this mechanism is not well characterized. Estimates range from about 5–18% (25,26) of the peanut energy value depending on the amount consumed and the background diet in which it is incorporated. Prior work revealed individuals who added 500 kcal day^{-1} of peanuts to their customary diet did not gain the predicted weight (1). The contribution of inefficient energy absorption to the findings of this trial cannot be determined as no fecal analyses were conducted.

Third, peanut consumption reportedly elevates resting energy expenditure. One trial observed a significant 11% increase in REE without a change of DIT (1). A 5% rise was noted in an 8-week trial of daily intake of conventional peanut oil by overweight participants (2). A later trial revealed a 13% increment in REE associated with daily almond consumption, but the change was not statistically significant (22). However, this was not replicated in the present study as neither REE nor DIT changed over time or varied between treatment groups. DIT in the HOP group was significantly higher than DIT from the CVP group at baseline, but this difference was not sustained over the trial. Likewise, studies with regular almond consumption showed no difference in REE, total energy expenditure, and DIT (22,27).

A fourth potential mechanism that may have contributed to the finding that similar weight loss occurred with a lesser energy restriction among peanut consumers entails an augmentation of fat oxidation with peanut consumption. This has not been well studied. In the present trial, fat oxidation was significantly elevated in the late postprandial period only in the HOP and CT groups. Further, the HOP group had higher late postprandial fat oxidation at the initial test session than the CVP group. Differential oxidation of fatty acids varying in saturation have been reported (1,28) with higher rates noted for monounsaturated fatty acids (28). Consistent with this, the present trial noted that fat oxidation was augmented by consumption of high oleic acid peanuts compared to the conventional variety.

Less is known about the effects of peanut consumption on body composition than body weight. This is an important distinction because a reduction in body fat accompanied by an increment in FFM can result in minor difference in weight yet hold important health benefits (29-32). Besides, differences in body fat distribution (peripheral and central) may have different impacts on metabolic parameters (11,13,33,34). Furthermore, improvement in body composition, including reduced body fat relative to FFM, may aid in to the prevention of weight regain (11,35). Within group analyses revealed significant reductions in total body fat mass (kg) only in groups consuming peanuts. Additionally, a significant decrease in total body fat percentage accompanied by a significant increase in total lean mass percentage was observed only in the HOP group. Moreover, only the HOP group decreased gynoid fat percentage, while its lean mass percentage increased. It has been reported that peanut oil

TABLE 3 Changes (Δ) in fasting energy expenditure, respiratory quotient, and carbohydrate and fat oxidation after the intervention

	CT ($n = 22$)	CVP ($n = 22$)	HOP ($n = 21$)	*P* value
REE (kcal day^{-1})	−21.3 ± 15.4	−26.9 ± 14.1	−0.4 ± 19.5	0.5195
Respiratory quotient	−0.01 ± 0.01	−0.02 ± 0.01[a]	−0.01 ± 0.01	0.7262
CHO oxidation (mg min^{-1})	−19.5 ± 17.4	−30.8 ± 11.8[a]	−16.2 ± 9.9	0.7405
Fat oxidation (mg min^{-1})	3.6 ± 6.5	9.9 ± 4.2[a]	11.1 ± 5.4[a]	0.6063

Values are mean ± SEM. *P* value column refer to differences between groups (ANOVA).
[a]Significant difference between baseline and final assessments within group ($P < 0.05$; paired *t* test or Wilcoxon test). CT, control group; CVP, conventional peanuts group; HOP, high-oleic peanuts group; REE, resting energy expenditure; CHO, carbohydrate.

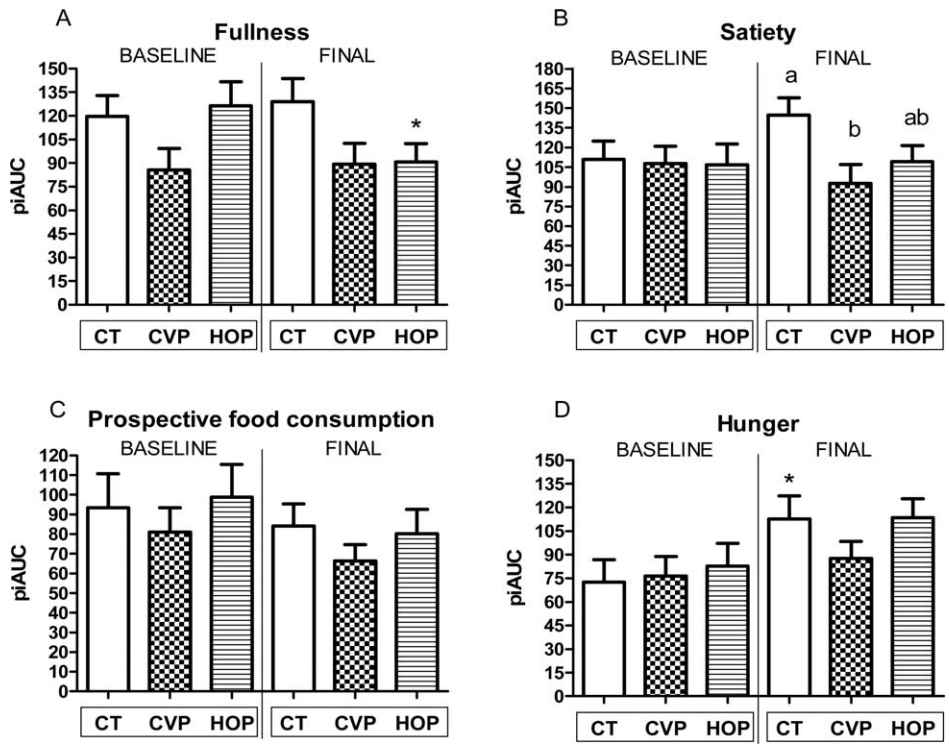

Figure 3 Mean (±SEM) fullness (**A**), satiety (**B**), prospective food consumption (**C**), and hunger (**D**), expressed as the positive incremental area under the curve (piAUC). Bars with different letters are significantly different (ANOVA; $P < 0.05$). *Significant difference between baseline and final assessments within group ($P < 0.05$; paired t test or Wilcoxon test). CT, control group; CVP, conventional peanuts group; HOP, high-oleic peanuts group.

intake in addition to habitual diet, during 8-weeks, did not increased hip circumference, a simple marker of gynoid fatness, while olive oil did (36). In a meta-analysis, nut-enriched diets were compared with control diets and no significant effect of nuts consumption on waist circumference was verified, yet, in studies that imposed an energy restriction its reduction was greater than in weight-maintenance studies (18). In the present trial, all changes in body composition occurred without changes in participants' physical activity so are attributed to the dietary intervention. The reduction in total body fat mass in CVP and HOP groups may be attributable to the significant increase in fasting fat oxidation in these groups. Furthermore, the slightly higher increment in fasting fat oxidation in HOP group compared to CVP group may be a consequence of the higher intake of MUFA in the HOP group (2,4). The CT group actually experienced a reduction of MUFA intake consistent with their lack of change in fat mass. High-oleic peanuts intake contributed to a decrement in body fat (≈3.1%) in the study conducted by O'Byrne el al. (10), although, lean and fat free mass were not measured in that study. Alper and Mattes found no significant difference in body composition after daily intake of conventional peanuts (1).

There are other rich sources of oleic fatty acid other than nuts. Corroborating the present findings, the consumption of an olive oil-enriched diet during 4-weeks by overweight and obese men, decreased fat mass accompanied by an increment in lean body mass without significant change in body weight (37). Moreover, after following a MUFA enriched-diet with a mix of olive oil and nuts for 4-weeks, a significant reduction in body weight and fatness was observed in overweight and obese man (38). Furthermore, although an olive oil-enriched diet did not change body weight and fatness of the obese individuals, it prevented central body fat distribution leading to an improvement in insulin sensitivity (39). When two MUFA-enriched diets within a 6-months weight reduction program were compared (olive oil versus rapeseed), no significant difference between then was verified, yet, both were effective in reducing body weight, fat mass, and waist circumference with an increment in lean mass (40). These results are consistent with the hypothesis that oleic fatty acid improves body composition to reduced cardiovascular disease risk.

Conclusion

The inclusion of peanuts in an energy-restricted diet does not compromise weight loss. Indeed, peanut consumption contributes to higher fat oxidation and improved body composition. This is augmented by ingestion of high oleic peanuts. O

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