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

Obesity

High-Oleic Peanuts: New Perspective to Attenuate Glucose Homeostasis Disruption and Inflammation Related Obesity

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Objective: To evaluate the effects of acute and daily consumption of high-oleic peanuts (HOP) on inflammation and glucose homeostasis in overweight/obese men.
Methods: In a 4-week randomized clinical trial, males with body mass index of 29.8 ± 2.3 kg/m^2 and aged 18-50 years were assigned to the groups: control (CT, $n = 22$); conventional peanuts (CVP, $n = 22$); or HOP ($n = 21$). They followed a hypocaloric-diet with or without 56 g/day of CVP or HOP. Main outcomes were changes in fasting blood biomarkers and postprandial insulin, glucose, tumor necrosis factor-alfa (TNF-α), and interleukin-10 (IL-10) responses after acute peanut intake.
Results: At baseline, HOP showed significantly lower postprandial responses of glucose, insulin, and TNF-α than CVP and CT. Changes in fasting blood biomarkers did not differ between groups after the 4-week intervention. However, within groups, total cholesterol decreased in CT, and all groups reduced High-density lipoprotein (HDL-c). Triglycerides were reduced in HOP and CVP. IL-10 increased significantly in all groups while only the CT and CVP showed increased TNF-α after intervention.
Conclusion: Acute high-oleic peanut consumption leads to stronger moderation of postprandial glucose, insulin, and TNF-α concentrations than CVP and control meal intake. Whether daily intake of high-oleic peanuts has additional benefits to CVP remains uncertain.

Obesity (2014) **22**, 1981–1988. doi:10.1002/oby.20825

Introduction

Obesity is associated with increased risk for the development of metabolic syndrome, type 2 diabetes, and cardiovascular disease (CVD) (1). Further, increased body fatness is associated with higher circulating concentrations of inflammatory biomarkers, which negatively influence the cardiovascular system and glucose homeostasis (2-5). Tumor necrosis factor-alfa (TNF-α), a proinflammatory cytokine, induces the phosphorylation of the serine residues of the insulin receptor substrate, disrupting insulin signaling by reducing GLUT-4 synthesis and translocation culminating in hyperinsulinemia and/or insulin resistance (6). Western diets can contribute to these complications by continuous stimulation of the endocrine pancreas leading to a repeated or chronic hyperinsulinemia (7). Little is known about postprandial variations in circulating inflammatory markers, but insulin resistance exacerbates the postprandial inflammatory response, which in turn, can increase insulin resistance (8).

Saturated fatty acids (SFA) are more prone to storage than monounsaturated fatty acids (MUFA), promote atherogenesis and increase inflammation in adipose tissue (9-12). SFA replacement by MUFA can reduce total cholesterol and low-density lipoprotein (LDL-c) (12-15). Moreover, this substitution may improve glucose homeostasis and body weight management (12). Peanuts are a rich source of MUFA and their consumption is associated with improved postprandial profiles of inflammatory markers and lipids (16,17). Consistent with this evidence, preliminary findings indicate that recently bred high-oleic peanuts (HOP) improve the serum lipoprotein profile compared to a control diet (15). However, this requires replication and effects of HOP on inflammatory markers have not been evaluated. We hypothesized that inclusion of HOP in a hypocaloric-diet would improve the inflammatory blood biomarker profile of overweight and obese individuals. Further, because low-grade inflammation is associated with insulin resistance (2), the intake of HOP was also posited to moderate postprandial glucose and insulin responses. The purpose of this trial was to evaluate the effects of acute and daily consumption of high-oleic, compared to a conventional peanuts (CVP), on inflammation, glucose homeostasis, and lipid biomarkers in overweight and obese men.

Methods

Participants

One hundred and fifty men underwent a brief nutritional screening. Eligibility included age between 18 and 50 years, body mass index

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Funding agencies: Supported by CNPq, CAPES, and the Peanut Collaborative Research Support Program.
Author contributions: The study design involved all authors except from VSM. The data collection was performed by RDMA, APBM, and VSM. RDMA conduced data analysis, data interpretation, and wrote the manuscript. NMBC, RM, RCGA, and JB reviewed the manuscript and NMBC approved the paper.
Received: 7 January 2014; **Accepted:** 9 June 2014; **Published online 27 June 2014. doi:10.1002/oby.20825**

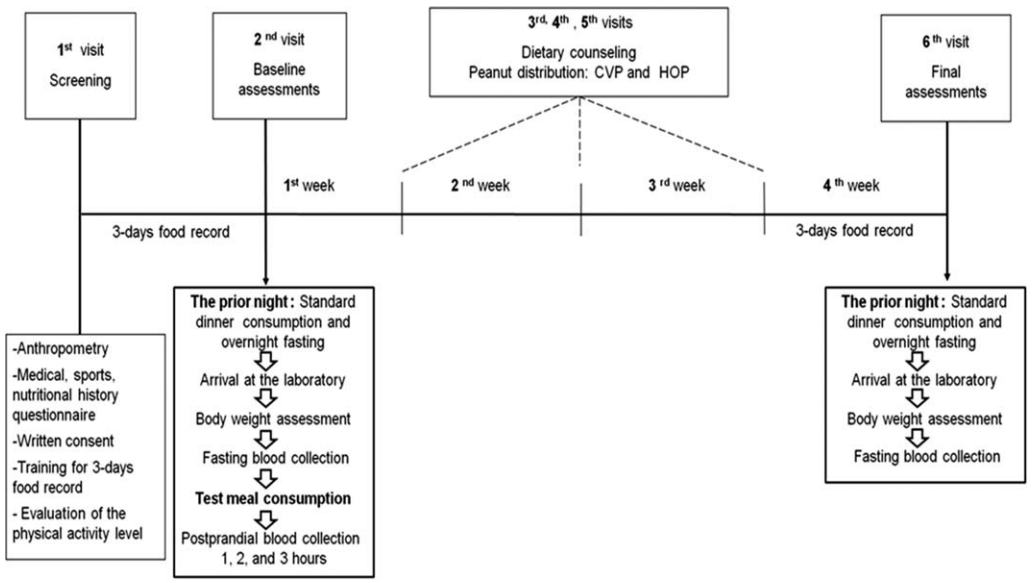

Figure 1 Study design. CVP, conventional peanuts group and HOP, high-oleic peanuts group.

(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 that might affect study outcomes over the 3 months prior to study initiation and high alcohol intake (>168 g/week). The study was approved by the Ethical Committee on Human Research of the Federal University of Viçosa (number: 185/2011). All participants provided informed consent.

Study design

This was a 4-week randomized, parallel-arm trial. Participants were assigned to one of three groups: control (CT); CVP; and HOP. The study design is represented in Figure 1. Participants consumed a standard dinner the night prior to assessments. After an overnight fast, a catheter was introduced into an antecubital vein for blood sample collection. Body composition was also assessed. Then, participants consumed a test meal within 15 min and after 1, 2, and 3 h, blood samples were drawn. During the next 4 weeks, participants followed a hypocaloric-diet and they were asked to maintain their customary physical activity level. At the end of the intervention period, the fasting measurements were repeated.

Dietary intervention

Each subject's daily energy requirement was calculated and 250 kcal was subtracted for the dietary prescription to promote ≈1 kg of weight loss during the trial. All the experimental diets provided 15% of energy from protein, 30% from fat, and 55% from carbohydrate. All groups consumed a hypocaloric-diet. The CT diet did not include any peanuts, but the CVP and HOP diet included a daily portion of 56 g of conventional or HOP, respectively. Participants were free to eat the peanut portion any time of the day, yet, they were asked to record the time in a notepad daily and 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, the total energy prescription was comparable on all three treatments. Since 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

A standard dinner was consumed the night prior to the assessments and it consisted of one pack of instant plain noodles (109 g-Nissin®) with 5 g of grated parmesan cheese, and 200 mL of grape juice.

On tests day, participants consumed their group-specific test meal within 15 min. All test meals provided 25% of each subject's daily energy requirement. They 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 the calories from carbohydrates, 16% from protein, and 49% from fat.

The portions of CVP and HOP offered to the participants contained 13.6 and 12.8 g of carbohydrates, 16.8 and 16.3 g of protein, 24.0 and 24.7 g of fat, and 5.0 and 5.5 g of dietary fiber (0.2 and 0.7 g of soluble; 4.8 and 4.8 g of insoluble), respectively. The fatty acid methyl esters were determined by gas chromatography following the protocol proposed by Folch et al. (18) and Hartman and Lago (19). Oleic fatty acid represents 51.0% of total fat in CVP and 81.5% in HOP (Table 1). Control biscuits were developed in the laboratory to offer a similar amount of total carbohydrates, protein, fat, and fiber, and energy density as CVP.

Dietary intake assessment

Participants completed 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).

TABLE 1 Percentage of fatty acids in relation to total fatty acids of the conventional peanuts and high-oleic peanuts, and control biscuits

Fatty acid	Conventional peanuts (IAC-886)	High-oleic peanuts (IAC-505)	Control biscuits
Lauric acid (C12:0)	-	-	0.43
Palmitic acid (C16:0)	8.78	5.23	12.76
Heptadecanoic acid (C17:0)	0.46	0.18	0.27
Stearic acid (C18:0)	2.14	2.08	8.08
Elaidic acid (C18:1n9t)	-	-	7.11
Oleic acid (C18:1n9)	50.96	81.47	35.16
Linolelaidic acid (C18:2n6t)	-	-	0.96
Linoleic acid (C18:2n6)	31.93	3.87	32.48
Arachidic acid (C20:0)	0.82	1.19	0.53
Gamma-linolenic acid (C18:3n6)	-	-	0.16
Eicosenoic acid (C20:1n9)	0.82	1.45	1.06
Alpha-linolenic acid (C18:3n3)	0.28	0.44	1.44
Behenic acid (C22:0)	2.59	2.68	-
Erucic acid (C22:1n9)	-	0.17	-
Lignoceric acid (C24:0)	1.46	1.65	-
Total SFA	16.25	13.01	22.07
Total MUFA	51.78	83.09	36.21
Total PUFA	32.21	4.30	34.08
Total Trans	-	-	8.07

− = not detected. Values are means of triplicates.

Measurements and calculations

All measurements, except postprandial blood collections, were completed at baseline and after 4 weeks. Participants were instructed not to consume caffeine or alcohol, to refrain from noncustomary physical activity, and to maintain a regular sleep-wake schedule (8 h/night) over the 72 h before assessments. Participants fasted overnight. Height and weight were assessed while the participants were standing straight, barefoot, and wearing light shorts. Body composition was assessed by dual-energy X-ray absorptiometry (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. A catheter was introduced into an antecubital vein and blood samples were collected at fasting and at the 1st, 2nd, and 3rd postprandial hour at baseline. Samples were centrifugated (2.200*g*, 15 min, 4°C), aliquoted, and stored at −80°C for further analysis.

Fasting and postprandial plasma TNF-α and IL-10 were analyzed by multiplex bead-based Luminex™ xMAP technology (Luminex™ 200 and xPonent/Analyst software) using commercial assay kits (Millipore's MILLIPLEX MAP Human Cytokine Panel-CYTOMAG-60k). Serum lipids, glucose, insulin, and high-sensitive C-reactive protein were quantified in fasting serum by automated analyzer systems using commercial assay kits as described elsewhere (20). Serum postprandial glucose and insulin were also analyzed at baseline. The homeostasis model assessment of insulin resistance (HOMA-IR) was calculated according to the equation proposed by Matthews et al. (21). Insulin resistance was classified according to Ascaso et al. (22).

Statistical analysis

Using IL-10 and TNF-α as the primary outcomes, power analyses calculated by the analyst procedures of the statistical analysis system (SAS) package indicated that a sample of 21 per group would permit detection of a 5% change of IL-10 and TNF-α with 99% power at the 5% level of probability.

The positive incremental area under the curve ($_{pi}$AUC) of postprandial concentrations of glucose, insulin, IL-10, and TNF-α was calculated using GraphPad Prism (Version 5; GraphPad software Inc). This method eliminates possible differences in the fasting condition. Statistics were also performed using SAS. The Shapiro-Wilk and Levene tests were performed to test data for normality and homogeneity of variance, respectively. Accordingly, parametric or nonparametric tests were performed. Results are presented as mean ± SEM. Body weight, dietary, and biochemical variables, including $_{pi}$AUC of postprandial concentrations of glucose, insulin, IL-10, and TNF-α, as well as changes (Δ = Final - Baseline) in variables, were compared between groups using one-way analyses of variance (ANOVAs) followed by Tukey's post hoc test or using Kruskal-Wallis followed by Dunn's post hoc test. Multivariate stepwise analyses followed by Tukey-Kramer post hoc tests were used to assess baseline-adjusted end-of-intervention between-group differences. Two-way repeated-measures ANOVA ($_{RM}$ANOVA) was applied to test the differences throughout the baseline test day for postprandial biochemical variables with test meals and time as repeated factors. Post hoc testing was performed using the Tukey-Kramer test. The pairwise tests (paired *t*-test or the Wilcoxon) were performed to compare habitual and fourth-week dietary intake and changes (Δ) in all variables. Analysis of covariance was used to evaluate whether changes in biochemical and inflammatory markers occurred independently of changes in body composition.

Results

Participants and baseline characteristics

Seventy-six participants were randomly assigned to the trial. Seven participants (9.2%) withdrew and 69 completed the study. Sixty-five participants were included in final assessments (Figure 2). Data from all participants that completed the study were included in baseline analyses as well as in the baseline postprandial analyses of glucose homeostasis and the inflammatory biomarkers.

Baseline weight did not differ between groups ($P > 0.05$; Table 2). Overall, mean participants' BMI was 29.8 ± 2.3 kg/m^2, 59.4% ($n = 41$) were overweight, and 40.4% ($n = 28$) were obese.

Neither peanut group differed from the CT group on baseline biomarkers but the HOP group participants had higher fasting insulin concentrations compared to the CVP participants (Table 2). Seven participants allocated in the HOP group (29.2%), two participants (9.1%) in the CT group, and three (13.0%) in the CVP group were insulin resistant (HOMA-IR > 3.5). Four participants in the CVP and the CT groups, and six in the HOP group had fasting glucose concentrations ranging from 100 to 125 mg/dL. Mean systolic and diastolic blood pressures were 119.6 ± 1.7 mm Hg and 72.4 ± 1.6 mm Hg, respectively, and were not significantly different between

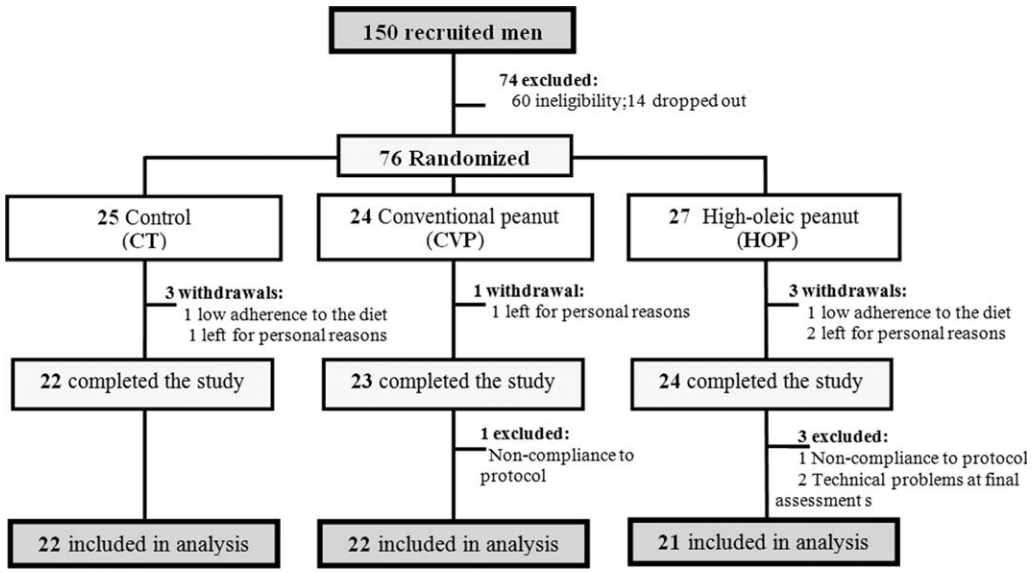

Figure 2 Participants flowchart.

TABLE 2 Fasting characteristics of the participants according to the experimental group at baseline

	CT (n = 22)	CVP (n = 23)	HOP (n = 24)	P value
Age (years)	27.1 ± 1.6	27.6 ± 1.5	27.2 ± 1.6	0.9433
Body weight (kg)	94.4 ± 2.5	93.1 ± 2.0	94.8 ± 2.1	0.8470
BMI (kg/m^2)	29.7 ± 0.6	29.5 ± 0.4	30.1 ± 0.5	0.6813
Total body fat percentage (%)*	33.4 ± 0.9	31.1 ± 1.0	33.5 ± 1.3	0.2314
Total fat mass (kg)*	32.9 ± 1.3	29.3 ± 1.4	31.6 ± 1.6	0.3462
Total lean mass percentage (%)*	62.9 ± 0.9	65.1 ± 1.0	62.9 ± 1.2	0.2859
Total lean mass (kg)*	61.4 ± 0.9	60.6 ± 1.0	58.7 ± 1.2	0.7533
Glucose (mg/dL)	90.9 ± 1.5	92.2 ± 2.4	92.3 ± 2.1	0.9623
Insulin (μU/mL)	8.7 ± 1.4^{ab}	8.2 ± 1.0^{a}	11.2 ± 0.9^{b}	0.0171
$HOMA_{IR}$	2.0 ± 0.3	2.0 ± 0.3	2.6 ± 0.02	0.2221
Total cholesterol (mg/dL)	184.1 ± 9.3	191.2 ± 9.4	183.5 ± 7.8	0.7881
VLDL-c (mg/dL)	23.3 ± 2.1	27.7 ± 3.0	28.5 ± 3.0	0.5897
LDL-c (mg/dL)	120.4 ± 8.1	123.3 ± 9.5	111.8 ± 7.3	0.6043
HDL-c (mg/dL)	40.3 ± 2.6	43.7 ± 3.2	40.0 ± 2.3	0.4151
Triglycerides (mg/dL)	116.7 ± 10.7	160.0 ± 26.1	153.3 ± 18.5	0.4455
Total cholesterol:HDL-c	4.8 ± 0.03	4.6 ± 0.02	4.8 ± 0.03	0.8783
LDL-c:HDL-c	3.1 ± 0.02	2.9 ± 0.02	2.9 ± 0.02	0.6296
Creatinine (mg/dL)	0.91 ± 0.03	0.91 ± 0.04	0.94 ± 0.03	0.7754
Uric acid (mg/dL)	5.4 ± 0.2	5.4 ± 0.3	5.9 ± 0.2	0.0690
hs-CRP (mg/dL)	1.4 ± 0.4	1.5 ± 0.2	1.7 ± 0.3	0.3670
TNFα (pg/mL)	3.8 ± 0.4	4.7 ± 0.4	5.2 ± 0.5	0.0863
IL10 (pg/mL)	2.3 ± 0.3	1.8 ± 0.2	2.2 ± 0.2	0.0809

Values are mean ± SEM. P value column refers to differences between groups (ANOVA or Kruskal-Wallis test followed by Tukey or Dunn's test, respectively). Values with different superscript letters are significantly different ($P < 0.05$). CT, control group; CVP, conventional peanut group; HOP, high-oleic peanut group; BMI, body mass index; $HOMA_{IR}$, homeostasis model assessment of insulin resistance; VLDL-c, very low-density lipoprotein; LDL-c, low-density lipoprotein; HDL-c, high-density lipoprotein; hs-CRP, high sensitivity C-reactive protein; TNF-α, tumor necrosis factor-alfa; and IL-10, interleukin-10. *Subsample CT (n = 12); CVP (n = 17); HOP (n = 18).

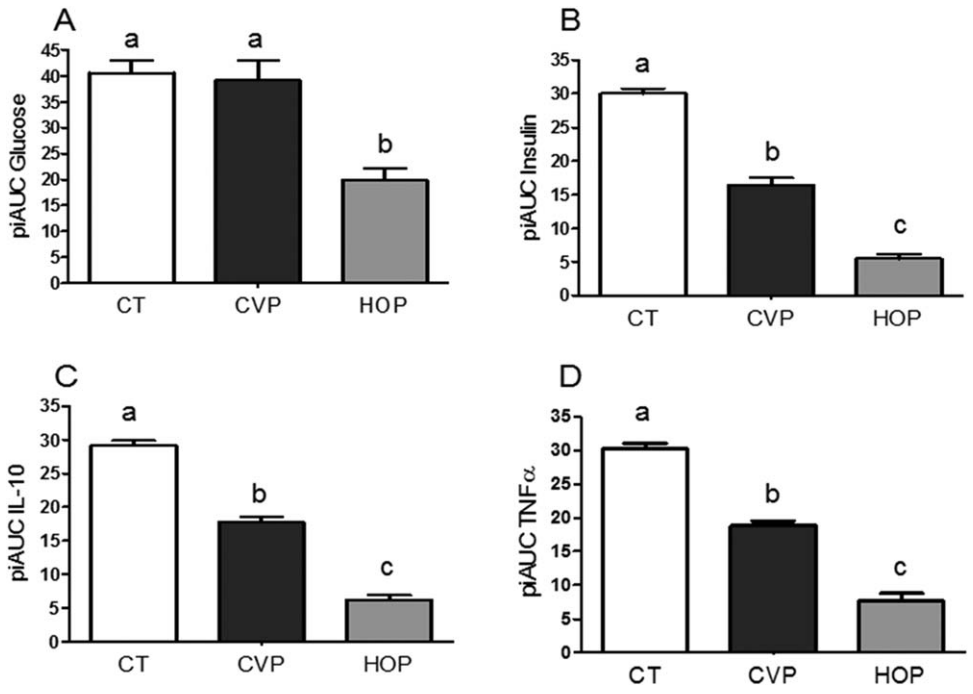

Figure 3 Postprandial response of serum glucose **(A)** and insulin **(B)**, and plasma tumor necrosis factor-alfa (TNF-α) **(C)**, and interleukin-10 (IL-10; D) during 3 h after test meal consumption, expressed as the positive incremental area under the curve ($_{pi}$AUC). Bars with different letters are significantly different (ANOVA; $P < 0.05$). CT, control group; CVP, conventional peanuts group; and HOP, high-oleic peanuts group.

the groups. Habitual dietary intake was also similar between groups (data not shown).

Postprandial inflammatory and glucose homeostasis biomarkers

On the first day of assessments, plasma IL-10 and TNF-α and serum glucose and insulin were measured at the first, second, and third hours after test meal consumption (Figure 1). CT group showed higher plasma responses for insulin, IL-10, and TNF-α than the peanut groups ($P < 0.0001$), with higher glucose response compared to HOP ($P < 0.001$; Figure 3). Furthermore, the CVP group response was higher than the HOP group response for plasma glucose, insulin, IL-10, and TNF-α ($P < 0.0001$; Figure 3). There was no significant group, time, or group × time interaction for postprandial biochemical parameters.

Changes after the 4-week intervention

Body weight and BMI were reduced in all groups ($P < 0.05$) without differences between them ($P > 0.05$). However, although there was no difference between groups ($P > 0.05$), the percentage of changes in comparison to baseline values for body composition variable differed. Total fat mass was significantly reduced in HOP (−1.39 ± 0.29 kg; −5.16%) and in CVP (−1.02 ± 0.33 kg; −3.69%), but not in CT (−0.78 ± 0.32 kg; −2.18%). Total lean mass was significantly reduced only in CT (−1.33 ± 0.30 kg; −2.14%), while in the CVP (−0.42 ± 0.39 kg; −1.59%) and HOP (−0.19 ± 0.33 kg; −1.78%) groups, it did not change significantly. Thus, in the CT group, only 37.1% of the total body weight loss was in fat mass while in the CVP and HOP groups 69.9% and 86.3% of total weight loss was fat, respectively, ($P < 0.05$).

Changes in dietary intake after the intervention are summarized in Table 3. There was no difference between groups for changes in energy intake ($P > 0.05$). MUFA intake increased relative to the CT group and the change was greater for the HOP group compared to CVP group. The CT group reduced their cholesterol intake significantly and the HOP group increased dietary fiber intake ($P < 0.05$). There was no difference between groups in the changes after the intervention, even after adjustments for baseline values (Table 4).

Serum glucose increased in all groups after intervention, yet, this increment was significant only in the CT group and the HOP group ($P < 0.05$) without a significant difference between groups. Changes in insulin and HOMA-IR were not significant. Plasma total cholesterol was decreased significantly only in the CT group ($P < 0.05$), while a significant reduction in very-low density lipoprotein (VLDL) was observed only in the CVP group. All the groups showed a significant decrement in high-density lipoprotein (HDL). Triglyceride levels were significantly reduced in the CVP and HOP groups. The CVP group was the only group that showed a significant increase in low-density lipoprotein (LDL):HDL-c ratio. No significant group differences were detected for hs-CRP. Conversely, IL-10 increased significantly in all groups. The CT and CVP groups had significant increments in TNF-α. Changes in biochemical biomarkers were not affected by changes in body weight and composition ($P > 0.05$).

TABLE 3 Changes in dietary intake (values at week 4 minus at baseline) of energy, macronutrients, cholesterol, and fiber according to the experimental group

	CT (n = 22)	CVP (n = 22)	HOP (n = 21)	P value
Total energy intake (kcal/day)	−552 ± 195	−276 ± 220	−212 ± 147	0.5379
Carbohydrates (g)	−57.8 ± 23.6	−58.5 ± 29.0	−65.1 ± 13.2*	0.977
Proteins (g)	−16.5 ± 9.1	−9.6 ± 9.3	−6.6 ± 7.3	0.785
Total fat (g)	−28.4 ± 8.7*	−0.4 ± 9.6	8.3 ± 11.4	0.0824
Saturated fat (g)	−8.5 ± 2.5*	−1.0 ± 2.3	3.3 ± 4.0	0.097
MUFA (g)	−10.8 ± 2.7[a]*	5.1 ± 2.9[b]	17.8 ± 5.0[c]*	<0.0001
PUFA (g)	−5.5 ± 1.6*	0.9 ± 2.9	−1.9 ± 1.4	0.2125
Cholesterol (mg)	−78.4 ± 26.3*	−94.5 ± 33.6	−58.8 ± 30.0	0.7679
Dietary fiber (g)	1.8 ± 2.5	2.4 ± 3.3	8.9 ± 2.0*	0.0670

Values are mean ± SEM. P value column refer to differences between groups (ANOVA or Kruskal−Wallis test followed by Tukey or Dunn's test, respectively). Values with different superscript letters are significantly different ($P < 0.05$). *Significant difference between final and baseline assessment within group ($P < 0.05$; paired t-test or Wilcoxon test). CT, control group; CVP, conventional peanut group; HOP, high-oleic peanut group; MUFA, monounsaturated fatty acid; and PUFA, polyunsaturated fatty acid.

Discussion

Although changes after the intervention were not significantly different between groups, there was a significant increase in fasting glucose in CT and HOP. However, this increment is not viewed as clinically important, since mean glucose at the final assessment was with the normal range. There were no differences between groups for HOMA-IR at the final assessment nor in its delta, even after adjustments for baseline values. The HOP group had higher values of fasting insulin and HOMA-IR than the CVP group but these differences did not remain after the intervention. The HOP group had a nonsignificant decrement in insulin and in HOMA-IR, while the other groups showed a nonsignificant increment, which is clinically relevant. These findings suggest that high-oleic peanut intake may have contributed to an improvement in insulin sensitivity. This is consistent with findings of the lowest $_{pi}$AUC of postprandial serum insulin and glucose in HOP. Besides, while in the HOP group a nonsignificant decrement in insulin and in HOMA-IR was verified, the other groups showed a nonsignificant increment, which is clinically relevant. These findings were not related to changes in body composition, although adipose tissue is recognized as a highly active metabolic and endocrine organ, which sends and responds to signals that modulate appetite, energy metabolism, and insulin sensitivity (4,23). Changes in body composition are probably related to the increased fat oxidation noted after peanut consumption (24).

Prior work indicates that the acute intake of peanuts (raw, roasted, and ground-roasted) does not alter glycemic responses compared to a control meal (25). In the present study, the glucose and insulin

TABLE 4 Changes in fasting biochemical parameters after 4-week of dietary intervention (values at week 4 minus at baseline)

	CT (n = 22)	CVP (n = 22)	HOP (n = 21)	P value
Glucose (mg/dL)	4.00 ± 1.79*	3.77 ± 2.28	5.76 ± 1.61*	0.5389
Insulin (μU/mL)	0.74 ± 1.49	0.49 ± 0.70	−0.81 ± 0.78	0.2828
$HOMA_{IR}$	0.26 ± 0.36	0.18 ± 0.21	−0.02 ± 0.20	0.3189
Total cholesterol (mg/dL)	−15.77 ± 6.00*	−3.00 ± 6.76	−10.62 ± 7.87	0.4185
VLDL-c (mg/dL)	1.03 ± 2.17	−3.84 ± 1.97*	−4.87 ± 2.55	0.3068
LDL-c (mg/dL)	−12.36 ± 5.84	0.88 ± 5.42	1.39 ± 7.14	0.2165
HDL-c (mg/dL)	−3.95 ± 1.25*	−2.64 ± 1.69*	−3.00 ± 1.29*	0.8432
Triglycerides (mg/dL)	19.05 ± 17.47	−19.18 ± 9.83*	−24.33 ± 12.75*	0.1945
Total Cholesterol:HDL-c	0.00 ± 0.13	0.27 ± 0.13	0.07 ± 0.20	0.4251
LDL-c:HDL-c	−0.06 ± 0.13	0.29 ± 0.14*	0.31 ± 0.16	0.1491
Uric acid (mg/dL)	−0.06 ± 0.12	−0.20 ± 0.11	−0.16 ± 0.12	0.7006
hs-CRP (mg/dL)	0.55 ± 0.40	−0.22 ± 0.18	−0.05 ± 0.24	0.2647
TNFα (pg/mL)	1.59 ± 0.46*	2.14 ± 0.71*	0.69 ± 0.5	0.2261
IL10 (pg/mL)	3.55 ± 2.09*	1.23 ± 0.26*	0.88 ± 0.27*	0.2915

Values are mean ± SEM. P values refer to differences between groups (ANOVA or Kruskal−Wallis). There was no difference between groups even after adjustments for baseline values ($P > 0.05$). *Significant difference between the final and baseline assessment within group ($P < 0.05$; paired t-test or Wilcoxon test). $HOMA_{IR}$, homeostasis model assessment of insulin resistance; VLDL-c, very low-density lipoprotein; LDL-c, low-density lipoprotein; HDL-c, high-density lipoprotein; Hs-CRP, high sensitivity C-reactive protein; TNF-α, tumor necrosis factor-alfa; and IL-10, interleukin-10.

responses after peanut intake, both conventional and high-oleic, were significantly lower than after control biscuit intake. The basis for this difference is unclear because the control biscuits were matched to the peanuts for total fat and fiber content, yet it may be related to the differences in nutrient bioaccessibility. In peanuts, intracellular fat is encapsulated by cell walls, which are resistant to enzymatic degradation in the gastrointestinal tract (26-28), while in control biscuits lipids were not encased in a complex matrix. Thus, in peanuts the amount of fat absorbed depends on the degree of mastication and breakage of the cell walls, affecting the glucose and insulin responses by reducing the gastric emptying rate, meal digestion and absorption rates (25). Besides, the higher polyphenol content of the peanuts may be an explanation as these compounds reduce amylase activity and slow carbohydrate digestion (29).

The differences in the postprandial responses of glucose, insulin, and TNF-α observed between control biscuits and peanuts can be partially explained by the higher content of SFA in the biscuits (22.1% of total fat). SFA can induce insulin and TNF-α release, leading to insulin resistance and hyperglycemia (6,30,31). It is noteworthy that the CVP has higher SFA content than the HOP (16.3% vs. 13% of total fat), which may have contributed to the difference in postprandial insulin and TNF-α response between the CVP and HOP groups. Oleic fatty acid represents 51.0% of total fat in CVP and 81.5% in HOP. It has been suggested that oleic acid is able to reduce the inflammatory effects of SFAs by reducing cellular stearic acid incorporation and nuclear factor-kappaB activation (32). Moreover, the oleic acid from peanut oil is able to reverse the inhibitory effect of TNF-α in insulin production (10).

All the test meals increased the IL-10 postprandial concentration at baseline, which in the long term and associated with weight loss, contributed to a significantly increment in IL-10 fasting concentration. Indeed, changes in IL-10 and TNF-α corroborate the results verified for postprandial measurements at baseline. Besides, all groups had a significant weight loss and an increment in IL-10 ($P < 0.05$), an anti-inflammatory mediator. Generally, a weight loss greater than 5-10% is required to induce significant changes in inflammatory biomarkers (3-5). In the present study, the mean weight loss was only 1.8 ± 0.19% yet a significant increase was observed in IL-10 relative to baseline. Although changes in hs-CRP were not significant, they declined in the peanut groups while an increase was noted in the CT group. The CT and CVP groups had a significant increase in TNF-α. This was not observed in the HOP group, who had the higher MUFA consumption. The high-oleic content of HOP peanuts may have contributed to these findings, since this fatty acid can moderate the inflammatory response (10,32,33).

Regular nut consumption is frequently associated with lower risk for CVD (34). Total blood cholesterol was decreased significantly only in the CT group. This may be explained by the fact that the SFA (g) and cholesterol (mg) intake were significantly reduced only in the CT group. Alper and Mattes did not find a difference in blood total cholesterol after conventional peanut intake (35). Lokko et al. reported a significant decrease in total cholesterol after regular intake of CVP (36). O'Byrne et al. reported a significant decline after daily intake of HOP though a reduction occurred in the control group as well (15). Reductions of cholesterol were only observed in several other studies among those individuals with elevated concentrations (16,37). Participants in the present study had normal cholesterol concentrations so may have been less responsive to the inclusion of peanuts to their diet.

O'Byrne et al. reported a significant decrement in HDL-c with daily intake of HOP as well as in the control group (15). Likewise, in the present study, all the groups showed a significant decline in HDL-c. Conversely, authors of studies that included daily intake of CVP reported significant increases in HDL-c (16,38). An increment in HDL-c was also reported in overweight participants after peanut oil intake for 4 weeks (11). However, no difference in HDL-c was observed after daily peanut intake in the study conducted by Lokko et al. or by Alper and Mattes (35,36).

In the present study, no changes in LDL-c were found after the intervention. Other studies also did not find significant changes in this lipoprotein after peanut intake (11,16,35,36). As there was a significant reduction in HDL-c without changes in LDL-c, the CVP group had a significant increase in LDL-c:HDL-c ratio. Conventional peanut intake did not change the atherogenic index in other studies (16,35,36). Triglyceride levels were significantly reduced in the HOP and CVP groups, while in the CT group there was a non-significant increment. Other studies also report a reduction in triglycerides after CVP intake (16,35-37). One previous study reported that daily HOP intake did not promote changes in triglycerides (15)

Conclusions

Acute peanut intake, specially the high-oleic variety, improves postprandial blood glucose, insulin, and TNF-α concentration compared to a control snack. Thus, HOP for human consumption must be better explored. Whether chronic consumption of HOP will lead to a reduction of CVD risk warrants further consideration. O

Acknowledgments

The authors thank technicians, faculties, and students who cooperated in data collection and analysis. They also thank the Instituto Agronômico de Campinas and CAP agroindustrial who donated the peanuts for the study.

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