Obesity

Effects of Canola and High-Oleic-Acid Canola Oils on Abdominal Fat Mass in Individuals with Central Obesity

Xiaoran Liu[1], Penny M. Kris-Etherton[1], Sheila G. West[1,2], Benoît Lamarche[3], David J.A. Jenkins[4], Jennifer A. Fleming[1], Cindy E. McCrea[2], Shuaihua Pu[5], Patrick Couture[3], Philip W. Connelly[6], and Peter J.H. Jones[5]

Objective: To determine the effect of diets low in saturated fatty acids and high in monounsaturated fatty acids (MUFA) or polyunsaturated fatty acids on body composition in participants at risk for metabolic syndrome (MetS).

Methods: This study was a randomized, crossover, controlled feeding study. Participants ($n = 101$, ages 49.5 ± 1.2, BMI 29.4 ± 0.4 kg/m^2) were randomized to five isocaloric diets containing treatment oils: Canola, CanolaOleic, CanolaDHA, Corn/Safflower, and Flax/Safflower. Each diet period was 4 weeks followed by a 2- to 4-week washout period.

Results: Canola (3.1 kg, $P = 0.026$) and CanolaOleic oil diets (3.09 kg, $P = 0.03$) reduced android fat mass compared with the Flax/Saff oil diet (3.2 kg), particularly in men. The decrease in abdominal fat mass was correlated with the reduction in blood pressure after the Canola (systolic blood pressure: $r = 0.26$, $P = 0.062$; diastolic blood pressure: $r = 0.38$, $P = 0.0049$) and CanolaOleic oil diets (systolic blood pressure: $r = 0.39$ $P = 0.004$; diastolic blood pressure: $r = 0.45$, $P = 0.0006$). The decrease in abdominal fat mass also was associated with a reduction in triglyceride levels after the CanolaOleic oil diet ($r = 0.42$, $P = 0.002$).

Conclusions: Diets high in MUFA (compared with PUFA) reduced central obesity with an accompanying improvement in MetS risk factors. Diets high in MUFA may be beneficial for treating and perhaps preventing MetS.

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[1] Department of Nutritional Sciences, The Pennsylvania State University, University Park, Pennsylvania, USA. Correspondence: Penny Kris-Etherton (pmk3@psu.edu) [2] Department of Biobehavioral Health, The Pennsylvania State University, University Park, Pennsylvania, USA [3] Institute of Nutrition and Functional Foods, Laval University, Quebec City, Quebec, Canada [4] Department of Nutritional Sciences, University of Toronto, Toronto, Ontario, Canada [5] Richardson Center for Functional Foods and Nutraceuticals, University of Manitoba, Winnipeg, Manitoba, Canada [6] Keenan Research Centre for Biomedical Science of St. Michael's Hospital, Toronto, Ontario, Canada.

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Introduction

Abdominal obesity is an important criterion for metabolic syndrome (MetS) along with glucose intolerance, dyslipidemia, and hypertension (1). MetS increases the risk of cardiovascular disease and type 2 diabetes mellitus (2). Almost 40% of U.S. adults have MetS based on the International Diabetes Federation definition (3). Weight loss is the primary treatment for MetS (4). Evidence is emerging that demonstrates beneficial effects of dietary monounsaturated fatty acids (MUFA) in regulating body weight and cardiometabolic risk factors (5). The Prevención con Dieta Mediterránea (PREDIMED) study showed that long-term consumption of a high-MUFA diet provided by extra virgin olive oil or mixed nuts reduced central obesity (6). In addition, studies have shown that MUFA-enriched diets (21%–23% of energy) reduce abdominal fat and central obesity (7,8). A short-term study (3 weeks) by Kien et al. (9) reported that a diet rich in oleic acid versus palmitic acid was associated with decreased android adiposity in healthy men and women. In the OmniHeart Trial, a diet with 21% of energy from MUFA (and 10% from polyunsaturated fatty acids, PUFA) lowered blood pressure and coronary heart disease risk compared with the higher carbohydrate diet (5). Others have shown that high-MUFA (20%–23% of energy) diets improved the lipid/lipoprotein profile (10,11), as well as insulin sensitivity and/or glycemic control (10,12). Our understanding of the role that MUFA play in cardiometabolic disease risk reduction is in the early stages, and further research is needed to clarify the specific effects that MUFA have on central obesity, which is causally related to other MetS criteria.

This study was conducted to evaluate the effects of five vegetable oil blends varying in MUFA and PUFA on body composition changes and cardiometabolic risk factors in individuals with or at risk for MetS. Our hypothesis was that high-MUFA diets would beneficially affect central obesity in individuals with or at risk for MetS and also reduce other cardiometabolic risk factors.

Methods

Participants

We have published detailed information about the study design, participant characteristics, and diet design (13). Briefly, 130 participants were studied at three research centers: University of Manitoba (Canada), Laval University (Canada), and the Pennsylvania State University (United States). Inclusion criteria were men and women 20 to 65 years of age and BMI between 22 and 40 kg/m^2 with central obesity (waist circumference: men $\geq$94 cm; women $\geq$80 cm) plus at least one other MetS criterion. Criteria included raised fasting blood glucose ($\geq$5.6 mmol/L), decreased high-density lipoprotein cholesterol (men $\leq$1.0 mmol/L, women $\leq$1.3 mmol/L), increased triglycerides (TG) ($\geq$1.7 mmol/L), and elevated blood pressure (systolic blood pressure, SBP $\geq$130 mm Hg or diastolic blood pressure, DBP $\geq$85 mm Hg). Exclusion criteria were thyroid disease, diabetes mellitus, kidney disease, liver disease; current smokers; or consuming more than two alcoholic drinks per week. Individuals taking any medications for dyslipidemia, hypercholesterolemia, or inflammation were not eligible to participate in the study. Recruitment criteria were based on the International Diabetes Federation criteria for MetS (14). Participants were recruited via flyers, local newspapers, radio advertisements, and campus mail. The study was approved by the Ethics Committee of University Manitoba, Laval University, and Pennsylvania State University and carried out in accordance with the Helsinki Declaration.

Study protocol

A randomized, crossover, five-period, controlled feeding study was conducted. Each treatment period lasted 4 weeks and was separated by a 2- to 4-week break during which time participants followed their habitual diet. After meeting eligibility criteria, participants were randomly assigned to a sequence of five experimental diets. The study coordinators and participants were blinded to the treatments as were all laboratory staff.

Dietary intervention

Test diets were created using Food Processor SQL software, version 10.8 (ESHA Research, Salem, OR). Participants were fed an isocaloric diet (calculated by Harris-Benedict equation) and advised to follow their routine physical activity practices. Baseline body weight was used to calculate basal metabolic rate (BMR). For men: BMR = 66 + [13.7 × weight (kg)] + [5 × height (cm)] – [6.8 × age (years)]. For women: BMR = 655 + [9.6 × weight (kg)] + [1.8 × height (cm)] – [4.7 × age (years)]. Weight was measured daily (Monday through Friday). During the feeding periods, participants were provided with all of their food, which was prepared in the metabolic kitchen at each research center.

The macronutrient profiles of the study diets were based on a typical American diet with 50% of energy from carbohydrate, 35% of energy from fat (18% from treatment oils), and 15% of energy from protein. Five treatment oils, Canola (conventional canola oil), CanolaOleic (high-oleic-acid canola oil), CanolaDHA (high-oleic-acid canola oil with DHA), Corn/Saff (corn/safflower oil), and Flax/Saff (flax/safflower oil), were studied and incorporated into smoothies that participants consumed twice daily. The smoothie contained the treatment oil, frozen unsweetened strawberries, orange sherbet, and nonfat milk. The quantity of oil was calculated based on participant energy needs. For a 3,000 kcal diet, 60 g of treatment oil per day was required to provide 18% of total energy. Each smoothie contained 100 g orange sherbet, 100 g nonfat milk, 100 g frozen unsweetened strawberries, and 30 g oil. Compliance was verified by assessing the total plasma fatty acid profile at the end of each intervention period, as described previously (15). In brief, fasted blood samples were collected. Fatty acid methyl esters were analyzed using an Agilent 6890N gas chromatograph equipped with a flame ionization detector (Agilent Technologies, Ontario, Canada). The quantity of each fatty acid was calculated by using the corresponding peak area divided by the total area of the fatty acids assayed.

The macronutrient profiles of the study diets are presented in Figure 1. The fatty acid profiles of treatment oils are presented in Supporting Information Table S1. A subgroup of participants ($n = 27$) from the University of Manitoba site prepared their own meals in a communal kitchen under supervision due to cultural habits and consumed smoothies with the treatment oils that were pre-made and delivered from the University of Manitoba. Since they did not receive a controlled feeding regimen, these participants were excluded from the analysis (15). Two participants who lost more than 5% of body weight due to illness unrelated to the study were also excluded from the analysis (Figure 1).

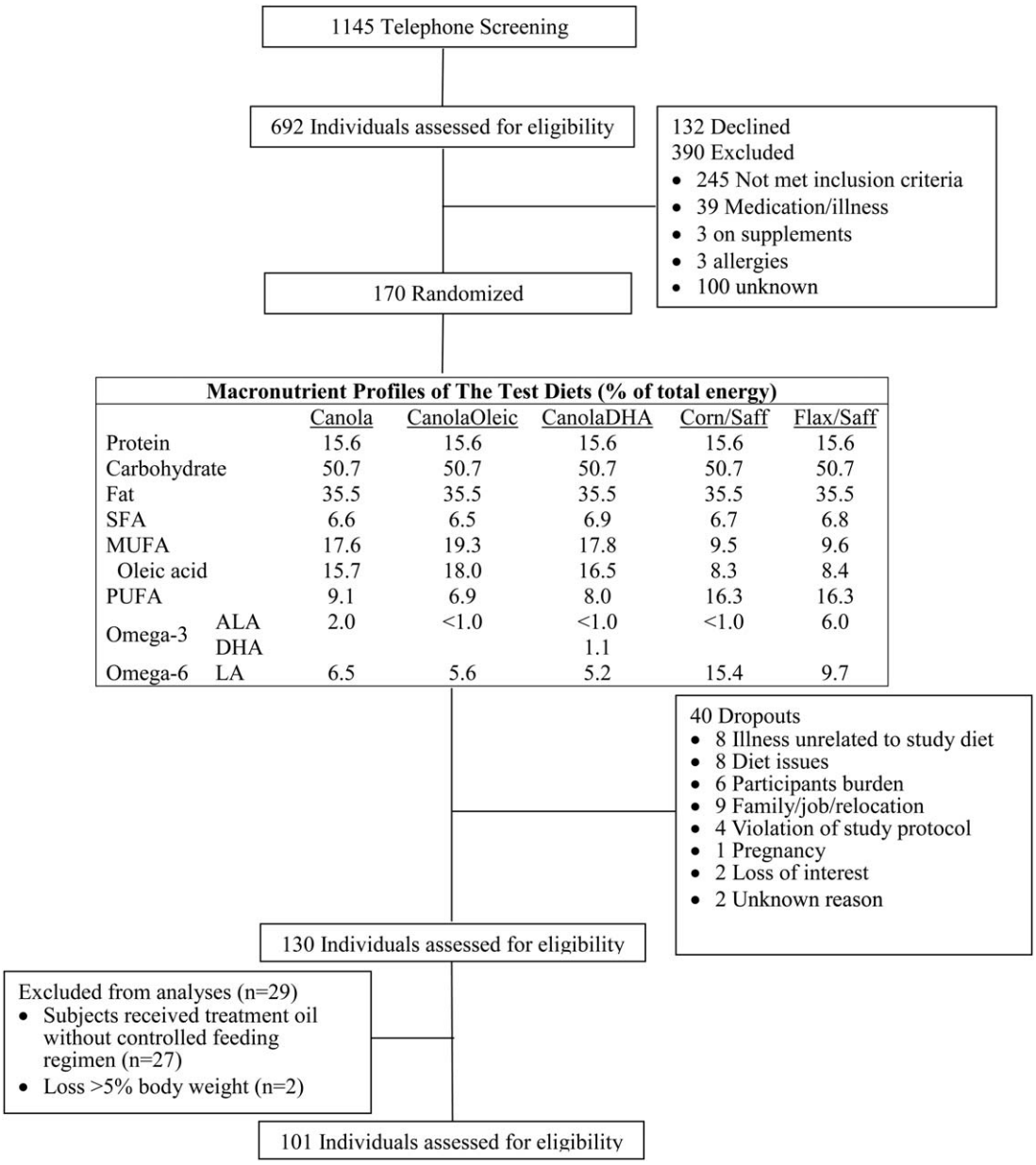

Figure 1 Participant screening, enrollment, and the macronutrient profiles of the study diets in the COMIT study.

Main outcome and measures

Dual-energy X-ray absorptiometry (DXA) measurements. Body composition was assessed by DXA according to the manufacturer's recommendations (Lunar Prodigy Advance, Madison, WI; QDR-4500W; Hologic Corp, Waltham, MA). Total and regional body composition was determined with Prodigy Encore 2005 software (version 9.30.044) and APEX System software (version 4.0). Criteria used to identify the anatomical region of interest were identical across all sites. A subset of the study population from two sites ($n = 54$) had baseline body composition assessment (measured at the beginning of the study). All sites performed DXA scans at the end of each diet period ($n = 101$).

Statistics

Variables were tested for normality and reported as the means ± SE. Statistical analyses were performed using SAS 9.2 (SAS Institute, Cary, NC) mixed model (PROC MIXED) procedure. Models included the following factors as fixed effects: diet, period, age, gender, diet by period, diet by gender interaction with participants, and center as random effects. No diet carry-over effect was observed. A Tukey adjustment was used for multiple comparisons. Within the 54 participants who had baseline body composition assessment, changes from baseline were calculated by subtracting the day 1 measurements from the day 29 measurements. For end-of-diet measurements, the sample size of $n = 101$ offered at least 90% of power to detect a mean of

TABLE 1 Subject metabolic characteristics at screening ($n = 101$)

	All participants ($n = 101$)	Males ($n = 50$)	Females ($n = 51$)
Anthropometric measurements			
Age	49.5 ± 1.2	46.6 ± 1.9	52.2 ± 1.5
Body mass (kg)	85.8 ± 1.5	95.8 ± 1.9[a]	75.9 ± 2.1[a]
Height (m)	1.7 ± 0.01	1.7 ± 0.01	1.6 ± 0.01
BMI (kg/m^2)	29.4 ± 0.4	30.2 ± 0.6	28.6 ± 0.6
Waist circumference	101.9 ± 1.1	107.0 ± 1.2[a]	96.8 ± 1.4[a]
Metabolic syndrome risk factors			
Glucose (mmol/L)	5.2 ± 0.1	5.2 ± 0.1	5.3 ± 0.2
HDL-cholesterol (mmol/L)	1.3 ± 0.03	1.1 ± 0.04	1.4 ± 0.1
Triglycerides (mmol/L)	1.8 ± 0.09	2.0 ± 0.2	1.7 ± 0.1
SBP/DPB (mm Hg)	122/78	123/78	118/78
No. metabolic syndrome risk factors per participant			
1 factor	57	25	32
2 factors	24	17	7
3 factors	19	7	12
4 factors	1	1	

Values are expressed as means ± SEM.
[a]Values were significantly different between genders, $P < 0.0001$.
[b]HDL-cholesterol, high-density lipoprotein cholesterol; SBP, systolic blood pressure; DBP, diastolic blood pressure.

3% differences in android fat mass between treatments with $\alpha = 0.05$. For changes from baseline in response to treatment diets, the sample size of $n = 54$ had 80% of power to detect a mean of 3% of android fat mass change between treatment with $\alpha = 0.05$. The Benjamini–Hochberg procedure was used to control for the false discovery rate. With the Benjamini-Hochberg critical value for a false discovery rate of 10%, the corrected level of significance was 0.03. Pearson correlation was used to calculate the correlation coefficient.

Results

Participant characteristics

Participant screening and enrollment in the study are shown in Figure 1. The study was conducted between July 2010 and April 2012. One hundred and one participants completed all five controlled feeding diet periods and were included in the analysis. The mean energy intake for all participants was the same between treatments (Supporting Information Table S1). Participants' baseline anthropometric and metabolic characteristics are summarized in Table 1. The majority of participants were Caucasian (95%). All participants had at least one MetS criterion in addition to central obesity, which characterized them as a population with or "at risk" for MetS (14). Specifically, 39 participants had elevated BP, 44 had elevated TG, 49 had low high-density lipoprotein cholesterol, and 34 had elevated fasted glucose. In addition to increased waist circumference, 50 participants had one additional criterion for MetS, 33 participants had two additional criteria, 16 had three additional criteria, and 2 had all five criteria for MetS. Fifty-one participants met the criteria for MetS.

Adherence

Plasma fatty acid profiles at the end of each diet period confirmed diet adherence (Supporting Information Table S2). Between-diet comparisons revealed that total MUFA concentration was the highest in participants on the CanolaOleic oil diet (18.5 ± 0.3, $P < 0.0001$), followed by the Canola oil diet (17.9 ± 0.2, $P < 0.0001$) versus the other diets. The Corn/Saff oil diet resulted in the highest total n-6 PUFA (38.7 ± 0.3, $P < 0.0001$), followed by the Flax/Saff oil diet (35.9 ± 0.3, $P = 0.0043$). The Flax/Saff oil diet resulted in the highest ALA (1.7 ± 0.04, $P < 0.0001$). The CanolaDHA oil diet resulted in the highest total n-3 PUFA plasma concentration (9.6 ± 0.1), mainly contributed by DHA (7.1 ± 0.08) and EPA (1.6 ± 0.06).

Outcomes

Correlations between android fat mass and cardiometabolic risk factors ($n = 54$). Baseline correlations between android fat mass and cardiometabolic risk factors ($n = 54$) are presented in Table 2. Android fat mass was positively correlated with C-reactive protein ($r = 0.28$, $P = 0.04$), TG levels ($r = 0.27$, $P = 0.04$), SBP ($r = 0.32$, $P = 0.02$), and DBP ($r = 0.32$, $P = 0.019$). Changes in android fat mass from baseline after the Canola and CanolaOleic oil diets were positively associated with decreases in SBP (Canola, $r = 0.26$, $P = 0.06$; CanolaOleic, $r = 0.39$, $P = 0.004$) and DBP (Canola, $r = 0.38$, $P = 0.005$; CanolaOleic, $r = 0.45$, $P = 0.0006$). Changes in android fat mass were positively correlated with decreases in plasma TG levels after consumption of the CanolaOleic oil ($r = 0.42$, $P = 0.002$) and the Flax/Saff oil ($r = 0.41$, $P = 0.002$) diets. Thus, as android fat mass decreased, there was a corresponding decrease in both SBP and DBP in the Canola and CanolaOleic oil diets, and a decrease in TG with the CanolaOleic oil diet.

Between-diet comparisons following the end-of-diet period body weight and body composition ($n = 101$). Body composition results are presented in Table 3. Participants had lower body weights at the end of the Canola ($P = 0.007$) and CanolaOleic oil

TABLE 2 Correlations between android fat mass and cardiovascular risk factors at baseline and after test diets ($n = 54$)

	Baseline		Canola		CanolaOleic		CanolaDHA		Corn/Saff		Flax/Saff	
	Android fat mass		Changes of android fat mass from baseline									
	r	*P*	*r*	*P*	*r*	*P*	*r*	*P*	*r*	*P*	*r*	*P*
TG	0.27	0.045	0.07	NS	0.42	0.0017	0.09	NS	0.26	NS	0.41	0.002
CRP	0.28	0.0043	0.0012	NS	0.013	NS	0.11	NS	0.11	NS	0.19	NS
SBP	0.31	0.023	0.26	0.06	0.39	0.004	0.28	0.044	0.19	NS	0.37	0.0052
DBP	0.32	0.019	0.38	0.0049	0.45	0.0006	0.30	0.028	0.21	NS	0.35	0.010

CRP, C-reactive protein; DBP, diastolic blood pressure; NS, nonsignificant; SBP, systolic blood pressure; TG, triglyceride.

diets ($P = 0.02$) compared with the Flax/Saff oil diet. There was a strong trend for the Canola oil diet to decrease trunk fat mass compared with the Flax/Saff oil diet ($P = 0.05$). There was a diet effect on android fat mass ($P = 0.01$). Participants had a lower android fat mass in response to the two high-MUFA diets, CanolaOleic oil diet (3.09 ± 0.1 kg, $P = 0.03$), and the Canola oil diet (3.1 ± 0.1 kg, $P = 0.03$) compared with the Flax/Saff oil diet (3.2 ± 0.1 kg). The decrease in android fat mass was affected by gender ($P = 0.03$ for diet by gender interaction). Specifically, males had a lower android fat mass (3.3 ± 0.1 kg) as well as the android-to-gynoid fat mass ratio (0.71 ± 0.02) in response to the CanolaOleic oil diet compared with the Flax/Saff oil diet (3.5 ± 0.1 kg, 0.73 ± 0.03, $P = 0.003$, $P = 0.0067$, respectively). In contrast, there were no differences in females across the five treatment diets.

Between-diet comparisons for changes in android fat mass from baseline ($n = 54$). Android fat mass was decreased from baseline on the Canola oil ($P = 0.04$) and CanolaOleic oil diets ($P = 0.007$); the reduction in response to the CanolaOleic oil diet was greater than that for the Flax/Saff oil diet ($P = 0.02$) (Figure 2). No between-diet differences were observed among the Canola, CanolaDHA, and Corn/Saff oil diets in android fat mass. A gender difference was observed in android fat mass in response to the experimental diets ($P = 0.02$). Male participants experienced a significant android fat mass loss ($P = 0.02$) from baseline after the CanolaOleic oil diet compared with the Flax/Saff oil diet.

Discussion

Our results add to the emerging evidence that dietary MUFA decrease central obesity and improve cardiometabolic health compared with PUFA, specifically with ALA (16). After just 28 days, the two MUFA-rich diets, CanolaOleic oil and Canola oil diets, reduced android fat mass (about 3%) compared with a n-6 PUFA-enriched Flax/Saff oil diet (which was high in ALA). The changes in android fat mass were accompanied by a reduction in the android-to-gynoid fat mass ratio after the CanolaOleic oil diet in males. The reduction was due to a decrease in central body fat mass, rather than a redistribution of adipose tissue to the lower body, as indicated by no change in the gynoid fat mass. The CanolaOleic oil diet provided the most MUFA (19.3% of energy) compared with the Canola and CanolaDHA oil diets (17.6% and 17.8% of energy, respectively). In the Canola and CanolaOleic oil diets that decreased android fat mass, plasma oleic acid concentration was highest (14.9% and 15.6%, respectively) compared with the other diets. These findings add to the evidence base that dietary MUFA beneficially affect central adiposity in an isocaloric setting. In support of our findings, Gillingham et al. (7) reported a tendency for a reduction ($P = 0.055$) in the android-to-gynoid fat mass ratio after consumption of a high-oleic canola oil diet that provided 21% MUFA from canola oil for 28 days in hypercholesterolemic individuals. Results from the PREDIMED study corroborate our findings in which diets high in MUFA have a beneficial effect on reducing central obesity and cardiovascular disease risk. The PREDIMED study reported a decrease in waist circumference, which is a surrogate marker for central obesity, and demonstrated cardiovascular benefits in response to long-term consumption of Mediterranean diets supplemented with extra virgin olive oil or mixed nuts (6,17,18).

The mechanisms for the reduction in body weight and android fat mass may include a greater oxidation rate and/or increased energy expenditure in response to the consumption of a high-MUFA diet. Previous studies have shown that diets high in MUFA affect fat balance, body mass, and possibly energy expenditure (16,19,20). Paniagua et al. (8) reported that a MUFA-rich diet compared with a carbohydrate-rich diet resulted in greater fat oxidation rates and a decrease in the abdominal fat-to-leg fat ratio in insulin resistant participants. In addition, Schmidt et al. (21) reported that the fractional oleic acid oxidation rate was significantly greater (21%) than for palmitic acid in healthy men ($n = 10$) after consuming meals that provided 16.5% energy from oleate and 16.3% from palmitate during an 8-h postprandial period. Kien and Bunn (19) demonstrated that consumption (for 28 days) of a high-oleic-acid diet (1.7% palmitic acid and 31.4% oleic acid) increased the fatty acid oxidation rate in women compared with the control diet (8.4% palmitic acid and 13.1% oleic acid). In the same study, a high-oleic-acid diet increased daily energy expenditure compared with a high-palmitic-acid diet (16.8% palmitic acid, 16.4% oleic acid) in men (19). Collectively, the results from other studies are suggestive of increased oxidation and energy expenditure mechanisms that may explain the decrease in body weight and android fat mass in response to the two high-oleic-acid diets evaluated in this study. In contrast, a recent study by Kien et al. (22) reported that a lower rate of total fatty acid oxidation was observed when palmitic acid (PA: 16.0% of energy; OA: 16.2% of energy) was replaced with oleic acid (OA: 28.8% of

TABLE 3 Body weight and composition after each test diet (all participants $n = 101$, males $n = 50$, females $n = 51$)

	Canola	CanolaOleic	CanolaDHA	Corn/Saff	Flax/Saff	*P*
Body mass						
All	86.6 ± 1.5[a]	86.8 ± 1.5[a]	86.9 ± 1.5[ab]	87.0 ± 1.5[ab]	87.3 ± 1.56[b]	0.0073
Male	95.3 ± 1.2[ab]	95.0 ± 1.9[a]	95.4 ± 1.9[ab]	95.5 ± 1.9[ab]	96.0 ± 1.9[b]	0.01*
Female	76.8 ± 1.6	77.3 ± 1.6	77.3 ± 1.6	77.2 ± 1.6	77.4 ± 1.6	NS
Total fat mass						
All	31.2 ± 0.8	31.2 ± 0.8	31.3 ± 0.8	31.4 ± 0.8	31.6 ± 0.8	0.083
Male	30.7 ± 1.1	30.4 ± 1.1	30.6 ± 1.1	30.5 ± 1.1	31.1 ± 1.2	NS
Female	31.7 ± 1.2	32.1 ± 1.2	32.0 ± 1.2	32.3 ± 1.2	32.0 ± 1.2	NS
Total lean mass						
All	51.3 ± 1.2	51.3 ± 1.2	51.4 ± 1.2	51.4 ± 1.2	51.4 ± 1.2	NS
Male	60.7 ± 1.2	60.8 ± 1.2	61.0 ± 1.2	61.0 ± 1.2	60.9 ± 1.2	NS
Female	42.1 ± 0.7	42.1 ± 0.7	42.1 ± 0.7	42.0 ± 0.6	42.1 ± 0.7	NS
Trunk fat mass						
All	16.7 ± 0.5[†]	16.7 ± 0.5	16.9 ± 0.5	17.0 ± 0.5	17.0 ± 0.5[†]	0.013
Male	17.9 ± 0.7[ab]	17.6 ± 0.6[a]	17.9 ± 0.7[ab]	17.9 ± 0.7[ab]	18.2 ± 0.7[b]	0.04*
Female	15.6 ± 0.7[a]	15.8 ± 0.7[ab]	15.9 ± 0.7[ab]	16.1 ± 0.7[b]	15.8 ± 0.7[ab]	0.04*
Trunk lean mass						
All	24.2 ± 0.6	24.2 ± 0.6	24.3 ± 0.6	24.3 ± 0.6	24.3 ± 0.6	NS
Male	28.2 ± 0.7	28.3 ± 0.7	28.4 ± 0.7	28.5 ± 0.7	28.4 ± 0.7	NS
Female	20.2 ± 0.4	20.2 ± 0.4	20.3 ± 0.4	20.2 ± 0.4	20.2 ± 0.4	NS
Android fat mass						
All	3.1 ± 0.1[a]	3.09 ± 0.1[a]	3.2 ± 0.1[ab]	3.1 ± 0.1[ab]	3.2 ± 0.1[b]	0.010*
Male	3.4 ± 0.1[ab]	3.3 ± 0.1[a]	3.4 ± 0.1[ab]	3.4 ± 0.1[ab]	3.5 ± 0.1[b]	0.0027*
Female	2.9 ± 0.1	2.9 ± 0.1	2.9 ± 0.1	2.9 ± 0.1	2.9 ± 0.1	NS
Android lean mass						
All	3.6 ± 0.1	3.6 ± 0.1	3.7 ± 0.1	3.6 ± 0.1	3.7 ± 0.1	NS
Male	4.3 ± 0.1	4.3 ± 0.1	4.3 ± 0.1	4.3 ± 0.1	4.3 ± 0.1	NS
Female	2.9 ± 0.1	3.0 ± 0.1	3.1 ± 0.1	3.0 ± 0.1	3.0 ± 0.1	NS
Gynoid fat mass						
All	5.3 ± 0.2	5.3 ± 0.2	5.3 ± 0.2	5.3 ± 0.2	5.3 ± 0.2	NS
Male	4.9 ± 0.2	4.9 ± 0.2	4.9 ± 0.2	4.8 ± 0.2	4.9 ± 0.2	NS
Female	5.7 ± 0.2	5.7 ± 0.2	5.8 ± 0.2	5.7 ± 0.2	5.7 ± 0.2	NS
Gynoid lean mass						
All	7.7 ± 0.2	7.7 ± 0.2	7.7 ± 0.2	7.7 ± 0.2	7.7 ± 0.2	NS
Male	9.0 ± 0.2	0.9 ± 0.2	9.1 ± 0.2	9.1 ± 0.2	9.1 ± 0.2	NS
Female	6.4 ± 0.1	6.4 ± 0.1	6.3 ± 0.1	6.4 ± 0.1	6.3 ± 0.1	NS
A/G						
All	0.61 ± 0.02	0.60 ± 0.02	0.61 ± 0.02	0.62 ± 0.02	0.62 ± 0.02	0.05
Male	0.72 ± 0.02[ab]	0.71 ± 0.02[a]	0.72 ± 0.02[ab]	0.72 ± 0.03[ab]	0.73 ± 0.03[b]	0.0067*
Female	0.51 ± 0.02	0.51 ± 0.02	0.51 ± 0.02	0.51 ± 0.02	0.51 ± 0.02	NS

Values are expressed as means ± SEM in kilograms.
Values in a row with different superscript letters indicate significant differences between diets, $P < 0.05$.
*Diet by gender effect.
[†]Canola versus Flax/Saff ($P = 0.0518$).
NS, nonsignificant; A/G, android-to-gynoid fat mass ratio.

energy) in 18 healthy participants. The authors suggested that the discrepancy with their previous finding about fatty acid oxidation could be due to the methods of food delivery and the high serum concentration of estradiol, which may have masked the effects of MUFA on fatty acid oxidation.

Related to this, MUFA have been shown to activate PPAR-δ and thereby increase fatty acid oxidative capacity (23,24), a finding that provides a plausible molecular mechanism for the body weight and android fat mass decreases we report herein for the diets highest in MUFA. Moreover, a derivative of oleic acid, oleoylethanolamide,

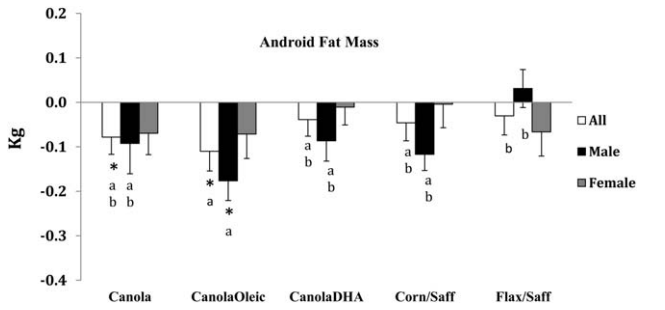

Figure 2 Android fat mass changes in response to five experimental diets. *Values were significantly different from baseline, $P < 0.05$. Values with different superscript letters indicate significant differences between diets, $P < 0.05$.

has been shown to induce lipolysis through the activation of PPAR-α, which provides a possible mechanism of action for the favorable body composition changes in participants on the high-MUFA diets (25-27). In addition, a higher MUFA diet has been associated with lower inflammatory activity that may regulate adipose tissue mass (28). Recent evidence has shown that inhibition of inflammasome-mediated caspase-1 activity and reduction of obesity-associated inflammation (i.e., interleukin-1β) attenuated an increase in body weight despite a similar daily food intake in animal models (29,30). High MUFA consumption may also contribute to this particular pathway in regulating body weight and fat mass.

Numerous studies have shown that android fat mass is positively correlated with BP, TG, and C-reactive protein levels (31-33). In our study, the reduction in android fat mass was positively correlated with decreases in cardiometabolic risk factors including TG, SBP, and DBP after the Canola, CanolaOleic, CanolaDHA, and Flax/Saff oil diets, but not the Corn/Saff oil diet. Previous evidence has shown that dietary MUFA decrease SBP (5). A recent cross-sectional study reported that MUFA intake (ranging from 8.1% kcal to 12.2% kcal), especially oleic acid from vegetable oil sources, may contribute to the prevention of high BP (34). Research has shown that weight loss and a reduction in android adiposity decrease MetS risk (35). The positive correlation between the reduction in android fat mass and the decrease in cardiometabolic risk factors demonstrates unique effects of dietary MUFA on MetS criteria that could be due to a decrease in android adiposity. Fatty acids have depot-specific effects on lipid accumulation in that MUFA preferentially accumulate in subcutaneous adipose tissue in contrast to SFA that preferentially accumulate in visceral adipose tissue (36-38). Consumption of a high-oleic-acid diet may therefore contribute to preferential fat deposition in subcutaneous adipose tissue and thereby decrease visceral adiposity and accompanying adverse metabolic effects.

A strength of this study is the controlled diet design, high rates of dietary adherence as evidenced by plasma fatty acid concentrations, and the delivery of the treatment oils, all of which were incorporated into smoothies for easy and accurate administration of the specified dose of oil. Second, the crossover study design minimized the influence of genetic polymorphisms that contribute to variations in diet responsiveness and consequently interindividual differences in results among study participants (39). Third, results presented herein are applicable to a large proportion of the U.S. population with almost 40% of adults having MetS.

Limitations of the study include the 4-week duration of each diet period. Further studies are needed to evaluate the specific effects of a high-MUFA diet longer term on body weight and android fat mass changes in free-living individuals on self-selected diets. As is always the case, it not possible to definitively conclude that increasing MUFA versus decreasing PUFA accounts for the observed effects. Nonetheless, increasing MUFA occurs both at the expense of decreasing PUFA (specifically ALA) and decreasing SFA. One recent study showed that erythrocyte linoleic acid was associated with decreased trunk fat mass in men and women (40). In conjunction with our finding, this suggests that varying PUFA (LA vs. ALA) may have a different effect on android fat mass. It will be important to conduct additional studies that confirm increasing MUFA is the mechanistically important event. In addition, while the baseline body composition is a reasonable covariate for assessing the effects of the experimental diets, comparisons with the baseline diet are limited because it was always ingested first and nutrient composition was only estimated based on the habitual intake of a Western population. Nonetheless, it is highly improbable that the SFA content of the habitual diets of our participants was as low as it was in the experimental diets, nor was MUFA as high.

This study has important clinical implications. Our results add to the emerging evidence base that diets high in MUFA have beneficial effects on central obesity and cardiometabolic risk factors.

Conclusion

In summary, short-term consumption of diets high in MUFA provided by Canola oil and CanolaOleic oil was associated with a reduced android fat mass in participants with or at risk for MetS. These changes were associated with favorable shifts in cardiometabolic risk factors. Importantly, our findings provide evidence for a beneficial effect of dietary MUFA in lowering cardiometabolic risk that we suggest is mediated by a decrease in android fat mass.

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