

Short-term changes in lipoprotein subclasses and C-reactive protein levels of hypertriglyceridemic adults on low-carbohydrate and low-fat diets

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Abstract

Diets designed to promote weight loss and improve atherogenic lipid profiles traditionally include a reduction in total fat and, in particular, saturated fats. This study was designed to test the efficacy of a low-fat diet vs a carbohydrate (CHO)–restricted (low-CHO) diet in hypertriglyceridemic patients on lipid profile, weight loss, high-sensitivity C-reactive protein (hs-CRP), and satiety. Twenty-eight hypertriglyceridemic subjects (based on fasting triacylglycerol [TG] levels exceeding 1.69 mmol/L) were randomized to either the low-CHO or low-fat diet for 8 weeks. Fasting bloods were acquired at weeks 0 and 8 and analyzed for lipids and hs-CRP. Body weight and other anthropometric measures were also obtained. Three random 24-hour food recalls were used to assess compliance during the trial and 2 recalls before randomization to permit individualized dietary education. A significant time-by-treatment interaction was observed ($P = .045$), wherein the small low-density lipoprotein cholesterol concentrations were reduced by 46% in the low-CHO–assigned subjects and increased by 36% for those assigned the low-fat plan. The observed decrease in TG (18%) among low-CHO subjects, in contrast to the 4% increase for low-fat group, was not significant, nor were there significant differences in hs-CRP, overall dietary compliance, satiety, or the magnitude of body weight loss between groups (low-CHO group, -3.8% vs low-fat group, -1.6%). Favorable reductions in small low-density lipoprotein concentrations after 8 weeks suggest that a moderately restricted carbohydrate diet (20% CHO as energy) can promote a less atherogenic lipid profile when compared to the low-fat diet.

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Abbreviations: CHO, carbohydrate; hs-CRP, high-sensitivity C-reactive protein; HTG, hypertriglyceridemic; LDL, low-density lipoprotein; low-CHO, carbohydrate restricted; HDL, high-density lipoprotein; IDL, intermediate-density lipoprotein; NMR, nuclear magnetic resonance; TG, triacylglycerol; VLDL, very low-density lipoprotein.

1. Introduction

Controversy continues regarding the best dietary strategy to recommend for hypertriglyceridemic (HTG) individuals who seek weight loss. Achieving sustainable weight loss

remains an important challenge for overweight and obese patients. However, because excess body fat is accompanied often either by atherogenic dyslipidemia (increased triacylglycerols or TG, reduced high-density lipoprotein [HDL] cholesterol, and a preponderance of small dense low-density lipoprotein [LDL] particles [1] or LDL pattern B, where 40% of total LDL are small LDL particles [2]), it is important to identify the dietary composition that promotes an improved lipid profile, whether weight loss is achieved. Based on a

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recent meta-analysis [3] and a clinical trial [4], the low-carbohydrate (low-CHO) diet may promote more favorable triacylglycerol (TG) and lipoprotein subclass changes than does the low-fat diet.

Obesity is also characterized by an inflammatory state, often reflected by elevated plasma C-reactive protein (CRP) levels [5], which fall with weight reduction [6,7]. Limited conflicting information is available regarding whether dietary manipulation alters plasma CRP levels, independent of weight loss [8–11]. Because high-sensitivity CRP (hs-CRP) has been shown to be highly predictive of future vascular events [12,13], it was important to assess whether dietary changes might reduce circulating hs-CRP levels. Thus, our objectives were to (1) determine whether a carbohydrate (CHO)-restricted (or low-CHO) diet reduces circulating concentrations of hs-CRP and atherogenic lipids including TG more than the low-fat diet and (2) to measure compliance to each diet using 3 24-hour recalls during the intervention similar to other dietary trials [14–16]. Thus, we hypothesized that subjects following a low-CHO diet for 8 weeks would exhibit lower concentrations of TG and hs-CRP compared to similar HTG patients following a low-fat diet.

2. Methods and materials

2.1. Study participants

Subjects (13 men and 15 women) were recruited from University Cardiologists of Rush University Medical Center who had reported fasting TG concentrations >1.69 mmol/L. Three participants with type 2 diabetes mellitus were also included. Exclusion criteria were as follows: TG concentrations >6.78 mmol/L, renal insufficiency, liver disease, type 2 diabetes mellitus, currently on a weight loss regimen, and on a low-CHO or low-fat diet at time of the study initiation. Current medications or supplements used were maintained throughout the study. All subjects provided written consent to participate in this study, which was approved by the institutional review board at Rush University Medical Center.

2.2. Design

This was an 8-week, parallel-group design study with subjects randomized to either a low-CHO diet or a low-fat diet (the usual approach by the preventive cardiology team). Subjects were asked to maintain their current level of physical activity throughout the 8-week dietary intervention period.

2.3. Baseline visit

Two 24-hour recalls were acquired during the 7-day period before the baseline visit to assess dietary preferences and compliance to alcohol restriction and fasting for the phlebotomy. Intake of stanol-containing products such as Benecol (McNeil Nutritionals, Ft. Washington, PA), Take Control (currently known as Promise active; Unilever, Englewood Cliffs, NJ), and others or fish oil or fiber sup-

plements was recorded throughout the trial, and subjects were asked to maintain current intake but not to add any of these potentially lipid lowering products to their diet. Participants fasted for a minimum of 12 hours, abstained from alcoholic beverages for 48 hours, and were encouraged to drink 8 to 16 oz of water before the blood draw. Anthropometric measurements including subject height, weight, abdominal circumference [17], sagittal diameter (Harpenden Anthropometer; Hoktain, Pembroke, UK) [18], and percent body fat by the near-infrared interactance (Futrex 5000A, Palm Desert, Calif) were obtained by the same investigator. Duration and frequency of physical activity was determined at baseline and at study conclusion using questions from the Futrex 5000A protocol [19,20].

2.4. Study interventions: diets

The purpose of this study was to test the short-term efficacy of a low-fat vs a low-CHO diet on weight loss, fasting lipid profiles (in particular, TG), and hs-CRP. The study was designed to be free-living, that is, no foods were provided to subjects. The low-fat diet was based on the standard dietary approach to lower elevated TG (including weight loss) using the Therapeutic Lifestyle Changes portion of the Adult Treatment Panel Report III [21]. The low-CHO diet was similar to the popular “Atkins” diet but not as restrictive on quantity of carbohydrates allowed [22]. In the latter dietary approach, subjects may consume their food ad libitum but must limit carbohydrate intake; no regard is given to total energy or total fat except in terms of saturated fat intakes. Diet instructions and handouts (including 7-day meal plans) were tailored to the individual’s food habits based on nutrition history questions and the 24-hour recall acquired before the baseline clinic visit. Subjects received dietary instructions by a clinical dietitian on alternating weeks throughout the study.

2.4.1. Low-carbohydrate diet

The goal of this diet was to have 15% of energy from CHO, 20% to 30% of energy from protein, and the remaining portion as fat (55%–65%) with saturated fat intake less than 10% of energy. The proportion of energy as fat is typical for a carbohydrate-restricted dietary plan [8,22]. Subjects were given guidelines to avoid alcohol and simple sugars; CHO-rich foods such as breads, cereals, rice, pasta muffins, chips, pretzels, and crackers; select vegetables (potatoes, corn, peas, and dried beans); dairy foods; fruits; and fruit juices. No specific targets were imposed for the monounsaturated and polyunsaturated fatty acid intake or total calories.

2.4.2. Low-fat diet

The low-fat dietary plan was based on the Therapeutic Lifestyle Changes portion of the Adult Treatment Panel Report III [21] where subjects are advised to restrict calories, particularly from fat, to produce weight loss. Thus, a goal of 104.6 kJ/kg body weight was recommended. The targets as a percentage of energy were as follows: for CHO, 50% to 60% (with an emphasis on complex CHO); for protein, 15%; and

Table 1

Physical characteristics of subjects according to dietary group assignment at baseline^{1,2}

Variable	Low-CHO (n = 10)	Low-fat (n = 13)
Age (y)	57.0 ± 9.8	48.4 ± 13.0
Proportion male (%)	50	46
BMI (kg/m ²)	34.5 ± 6.8	29.6 ± 4.4
Weight (kg)	91.3 ± 26.9	90.6 ± 91.0
Sagittal diameter (mm)	278.1 ± 44.9	247.4 ± 41.2
Abdominal circumference (cm)	105.1 ± 13.5	98.3 ± 14.7
Body fat (%) ^a	35.3 ± 6.8	29.4 ± 6.2

BMI, body mass index. Values represent means ± SD except those identified.

^a Body fat (%) was based on a 2-compartment model using Futrex 5000A; these measures were significantly different between groups on the basis of Mann-Whitney *U* test (*P* = .045).^{1,2}

for fat, 30% or less of energy with a reduced intake of transfatty acids and saturated fat intake below 10% of energy. The addition of stanols (2 g/d) and water-soluble fiber (10–35 g/d) was not incorporated into this diet as advocated at 6 weeks [21] because the diet treatments were only 8 weeks long.

2.5. Biochemical analyses

Screening TG analysis to determine eligibility of patients comprised using the Cholestech LDX System (Cholestech Corp, Hayward, Calif), using nuclear magnetic resonance (NMR) (LipoScience, Raleigh, NC), or through standard chemical analysis by the Rush University Medical Center Laboratory. At both baseline and 8-week visits, plasma samples were held at 4°C until shipped to LipoScience within 4 days of collection for subsequent NMR and hs-CRP analysis. Nuclear magnetic resonance spectroscopy [23] was used to quantify lipid and lipoprotein concentrations, as well as lipoprotein particle sizes. Determination of hs-CRP concentrations was performed with the Diagnostic Products Immulite 2000 analyzer (Block Scientific, Nutley, NJ); assay limits ranged from below 0.1 to above 10.0 mg/L. Urinary ketones (Multistix, Bayer Corporation, Elkhart, Ind) were measured at the last study visit only.

2.6. Dietary information and assessment of compliance

Two unannounced 24-hour recalls were obtained the week before randomization and 3 during the intervention. Recalls were analyzed using the Interactive Healthy Eating Index Web site (<http://www.usda/cnpp>). Dietary estimates were averaged across 2 recalls for baseline and across the 3 recalls at intervention. Compliance to the low-CHO diet was defined as CHO intake of 29% or less of energy and for the low-fat diet and fat intake of 30% or less of energy. Although compliance to dietary targets was a focus of the dietary instruction, noncompliance was not a criterion for exclusion from subsequent analyses. The validity of self-report measures, in particular, target components of a dietary pattern through use of multiple 24-hour recalls as a measure of adherence, has been demonstrated by others [14–16,24].

Overall satiety was measured at the completion of the 8 weeks using a survey developed by Layman et al [25]. The instrument contained 7 questions with a Likert scale format where responses ranged from 1 (dissatisfied or unsatiated) to 7 (satisfied or satiated).

2.7. Statistical analyses

SPSS for Windows (Version 13, SPSS, Inc, Chicago, Ill) was used for statistical analysis. Histograms were used to assess normality. Nonnormal data were transformed using logarithmic or square root transformations. A .05 significance level was used for all statistical tests. Repeated-measures analysis of variance was performed to test significance with time, dietary treatment, and lipid outcome measures at 0 and 8 weeks (within-subjects factor, time and between-subjects factor, dietary treatment). Both primary outcome variables, TG and hs-CRP, were logarithmically transformed to approximate normality. However, because distributions of

Table 2

Changes in baseline lipids, lipoprotein subclasses and hs-CRP by diet group¹

	Low-CHO (n = 10)		Low-fat (n = 13)	
	Week 0	Week 8	Week 0	Week 8
	Mean ± SD			
Triacylglycerols (TG) (mmol/L)	1.62 ± 0.64	1.33 ± 0.61	2.00 ± 1.03	2.08 ± 1.52
VLDL TG (mmol/L)				
Large	0.32 ± 0.25	0.26 ± 0.28	0.49 ± 0.62	0.36 ± 0.56
Medium	0.79 ± 0.52	0.55 ± 0.39	1.01 ± 0.71	1.22 ± 1.07
Small	0.15 ± 0.07	0.14 ± 0.09	0.13 ± 0.11	0.14 ± 0.15
VLDL size (nm)	46.6 ± 4.4	47.3 ± 6.4	41.3 ± 14.6	47.8 ± 5.5
Total cholesterol (mmol/L)	4.74 ± 0.78	4.47 ± 0.45	4.77 ± 1.10	4.73 ± 1.19
IDL cholesterol (mmol/L)	0.03 ± 0.08	0.02 ± 0.05	0.02 ± 0.04	0.02 ± 0.04
LDL cholesterol (mmol/L)	2.95 ± 0.62	2.83 ± 0.45	2.92 ± 1.01	2.88 ± 1.10
Large	0.87 ± 0.75	1.10 ± 0.54	0.88 ± 1.09	0.85 ± 0.73
Medium	1.22 ± 0.85	1.27 ± 0.54	0.77 ± 0.67	0.28 ± 0.42
Small ^a	0.81 ± 0.85	0.44 ± 0.53	1.26 ± 1.05	1.72 ± 1.34
LDL size (nm)	20.5 ± 0.6	20.8 ± 0.4	20.3 ± 0.7	20.2 ± 1.0
LDL particles (nmol/L)	1346 ± 280	1216 ± 210	1405 ± 534	1457 ± 657
HDL C (mmol/L)	1.16 ± 0.21	1.13 ± 0.20	1.14 ± 0.47	1.10 ± 0.43
Large	0.41 ± 0.16	0.44 ± 0.13	0.46 ± 0.43	0.49 ± 0.38
Medium	0.17 ± 0.12	0.18 ± 0.14	0.14 ± 0.11	0.09 ± 0.12
Small	0.58 ± 0.14	0.50 ± 0.12	0.54 ± 0.13	0.52 ± 0.10
HDL size (nm)	8.8 ± 0.4	8.7 ± 0.2	10.5 ± 5.1	8.8 ± 0.5
hs-CRP (mg/L) ^b	3.7	1.5	1.3	1.9
	(1.1, 10.3) ^c	(0.8, 6.7)	(0.6, 3.0) ^c	(1.0, 3.4)

Lipid profiles were determined using nuclear magnetic resonance spectroscopy.

^a Significant differences between dietary groups over time by repeated measures analysis of variance (treatment × time interaction, *P* = .045).^b Values represent median and (interquartile range).^c Significant differences between groups at baseline on basis of Mann-Whitney *U* test (*P* = .032).

hs-CRP were so highly skewed even with transformation, the Mann-Whitney U test was used to test for differences between groups with respect to change in hs-CRP. A χ^2 test was used to test for differences in compliance (yes/no) by diet treatment. A Mann-Whitney U test was also used to test for differences in perceived satiety between dietary groups.

3. Results

3.1. Baseline characteristics of study participants

Exactly 28 subjects were recruited from June 2003 to March 2004. Two subjects (1 from each treatment group) dropped out because of personal reasons, and 3 had missing values (all in low-CHO group). Thus, 23 subjects were included in the final analyses.

Baseline physical and demographic characteristics of the 23 subjects (87% white) who completed the study are provided in Table 1. Men and women were present in nearly equal numbers in each group. There were no significant differences between groups at baseline except for percent body fat ($P = .045$). There was a wide range in body mass index (18.7–49.5), and those of the low-CHO group were marginally higher than those of the low-fat assigned subjects ($P = .051$).

Both baseline and final NMR lipid and lipoprotein values and those of hs-CRP are provided in Table 2. There were no significant differences between groups at baseline, except for hs-CRP concentrations. The low-CHO group had significantly higher hs-CRP concentrations than those assigned to the low-fat treatment at baseline. Moreover, at baseline, more subjects in the low-fat treatment ($n = 7$) exhibited the LDL pattern B (small LDL as 40% of total LDL) compared to those subjects assigned to the low-CHO diet ($n = 3$) (data not shown). However, the average LDL particle size, 20.5 vs

20.3 nm for low-CHO and low-fat groups, respectively, was not different, nor was there a difference in LDL particle concentrations between the groups (1346 vs 1405 nmol/L, respectively) as shown in Table 2.

3.2. Changes in lipids, lipoprotein subclasses, and hs-CRP after 8-week diet interventions

Weight loss did not differ between groups after 8 weeks (low-CHO group loss 1.7 ± 1.5 kg vs low-fat group loss 0.7 ± 1.2 kg). There were no significant changes in body fat nor circumferences after 8 weeks (data not shown).

As depicted in Table 2, plasma TG concentrations were not significantly different within or between groups. The trend for lower TG (an 18% reduction) in the low-CHO–assigned subjects (as opposed to the small increase in the low-fat group) parallels changes observed for the medium very low-density lipoprotein (VLDL) subclass (drop in the low-CHO group and increase in the low-fat group). However, there were no significant changes in these VLDL subclasses or particle size (dietary treatment by time interaction).

There was a large increase (63%) in the medium LDL fraction among the low-fat diet–assigned subjects (63%), whereas the low-CHO subjects had a 4% rise in this fraction; yet, the differences between groups was not significant (diet by time interaction, $P = .09$). A significant time-by-treatment interaction was observed ($P = .045$), wherein the small LDL cholesterol concentrations were reduced by 46% in the low-CHO–assigned subjects and increased by 36% for those assigned the low-fat plan. These changes reflect those for LDL size (diet by time interaction, $P = .063$), with increases among low-CHO–assigned subjects and little change in those assigned to the low-fat regimen. Thus, by 8 weeks, 9 of 13 subjects assigned to the low-fat diet exhibited LDL pattern B, whereas only 1 from the low-CHO treatment was so identified (data not shown). No significant differences between groups across time were observed for hs-CRP levels; however, a median drop from baseline of 60% was observed for the low-CHO group, which reflects a change from high-risk to a medium-risk level ($P = .12$).

3.3. Dietary characteristics, changes, and compliance

Based on 2 diet recalls at baseline, all subjects consumed somewhat similar diets at baseline (Fig. 1) including energy: 7095 ± 2520 kJ for low-CHO and 7911 ± 3211 kJ for low-fat diet–assigned individuals. Over the 8-week period, both groups reported significant decreases in total energy intake (-1620 ± 2219 kJ for low-CHO vs -1013 ± 3152 kJ for low-fat, $P = .036$). However, there was no difference in the magnitude of energy reduction between groups. Similarly, both groups dramatically reduced the proportion of energy from saturated fat from 12% to 5% of energy among the low-CHO individuals and from 12% to 2.5% of energy in the low-fat groups. There were significant changes in the percent of energy from CHO ($P = .015$) and in energy-adjusted fiber intakes ($P = .034$) between groups—reductions in both for the low-CHO individuals with small increases in both for the

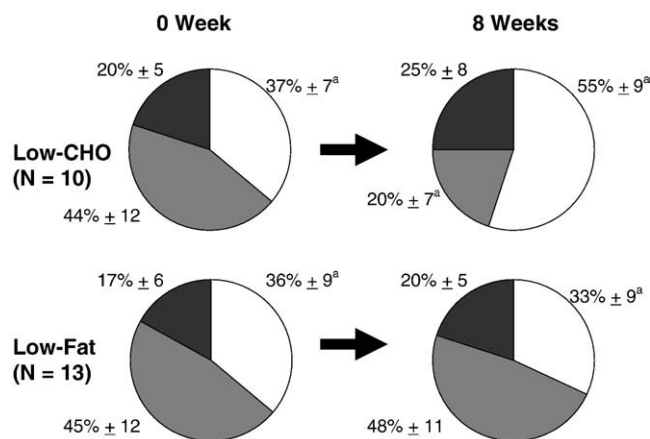


Fig. 1. Distribution of macronutrients (as percentages of energy) in subjects before and after 8 weeks on either a low-CHO diet or low-fat diet. ^aThe proportion energy from saturated fat was 12% for both groups at baseline and was 5% for low-CHO and 2.5% for low-fat. *Statistically significant difference was observed between groups with respect to the change in percentage of energy from CHO using Mann-Whitney U tests ($P < .001$).

low-fat subjects. No other time-by-treatment interactions were observed.

Compliance to the low-CHO and low-fat diets was determined by evaluating the three 24-hour diet recalls conducted during the intervention. Exactly 80% of the subjects on the low-CHO diet were compliant, whereas only 60% of the subjects on the low-fat diet were compliant; however, these differences were not significant.

Finally, scores for satiety and hunger components of the 7-item questionnaire [25] were not different between diet groups. Overall satisfaction scores were slightly higher with the low-fat group (5.5 ± 1.2 ; median, 5.0) vs the low-CHO individuals (4.9 ± 2.1 , median 5.0).

4. Discussion

This short dietary intervention had a significant impact on the concentration of the small LDL particles; these were lowered in subjects on the low-CHO dietary intervention in contrast to those assigned to the low-fat dietary intervention in whom the proportion of such particles increased. Accordingly, the number of individuals manifesting the atherogenic pattern B (predominance of small LDL particles) was reduced from 3 to 1, whereas 9 of 13 on the low-fat exhibited LDL pattern B. Aude et al [26] also observed significant reduction in small LDL subclasses (6%, $P = .02$) with their low-CHO and monounsaturated fat diet but no change (1.4%, $P = .29$) was observed for those on the Step II National Cholesterol Education Program diet. Favorable changes in small LDL subclasses were also observed when patients were on a 26% of energy as CHO diet during weight maintenance and in the 54% CHO group when weight loss occurred [4]. However, a hypocaloric very low-CHO (<10% of energy) in comparison to hypocaloric low-fat (<30% of energy) diet did not alter lipoprotein fractions or LDL size in another 4-week study of women [27]. Others [28] observed reductions in the number of LDL particles (difference = -30 nmol/L, $P = .74$) and in large- and medium-size VLDL particles with both low-CHO and low-fat diets after 6 months. In contrast to these findings, in the present study, we observed a decrease in medium LDL fraction (0.77–0.28 mmol/L) of subjects assigned to the low-fat diet, whereas, in the low-CHO subjects, there was an increase in this fraction (from 1.22 to 1.27 mmol/L).

As expected, TG values fell by 18% on the low-CHO diet compared to those subjects on the low-fat diet in whom these increased slightly (+4%, $P = .42$). Based on the meta-analysis of Dattilo and Kris-Etherton [29], plasma TG levels are expected to decrease by 0.015 mmol/L per kilogram of weight lost. In the present study, we would expect an average decrease of 0.03 mmol/L in TG concentrations of subjects assigned the low-CHO treatment and a decrease of 0.011 mmol/L for those on the low-fat diet. We observed nearly 10-fold decrease of TG (0.29 mmol/L) among low-CHO individuals and increase of 0.08 mmol/L in the low-fat group. In other reports, weight reduction was associated with

a reduction in TG concentrations in individuals on low-CHO diets [22,26,27,30–33]. The magnitude of TG reduction in the current study was less than previously reported [22,26,30], but the present study was at least 4 months shorter in duration. Nor were the subjects similar, because the current study was designed to recruit exclusively HTG individuals, regardless of body weight. Different subject characteristics, proportion of dietary CHO, and the shorter study duration might explain why weight loss was less than that reported in these studies [22,26,31]. The former 2 were 6 months in duration, whereas the Foster et al [31] study was 1 year in length. Moreover, 2 of the 3 studies tested low-CHO diets with a range of 20 to 30 g of CHO daily, much lower than the present effort (50–80 g). Low-CHO diets may result in greater reductions in TG than expected by weight loss alone [29,34,35], which some have attributed to changes in VLDL particle quantity and not size. Thus, with the present findings, marginally significant ($P = .09$) changes in medium VLDL particles with low-CHO treatment are entirely consistent with these longer-term trials.

Decreases in hs-CRP concentrations have been reported in the 6-month randomized clinical trial of low-fat vs low-CHO dietary interventions in 41 subjects [8]. This decrease was proportional to the amount of weight loss ($r = 0.41$, $P = .007$); there was no difference in the magnitude of hs-CRP reduction between the low-CHO (median change, -0.16 mg/L) and low-fat (median change, -0.02 mg/L) groups. Because these investigators also observed greater weight loss in the low-CHO group (-7.6 kg) and the low-fat group (-4.3 kg), the reduction in hs-CRP concentrations was greater than that of the present study. In a trial of 78 severely obese subjects randomly assigned to a low-CHO or hypocaloric low-fat diet for 6 months, reductions in CRP (~ 0.3 – 0.5 mg/dL) were also seen even after adjustment for weight changes [28]. No differences were attributed to dietary manipulation except when individuals ($n = 48$) with the highest CRP (exceeding 3 mg/dL) risk were examined separately. Then, more dramatic reductions (-0.9 mg/dL, after adjustment for weight loss) was observed with the low-CHO treatment in contrast to the low-fat intervention. In the present study, changes in hs-CRP were not related to dietary manipulation or change in body weight ($r = -0.25$, $P = .24$), but these were related to changes in abdominal circumferences ($r = -0.44$, $P = .03$).

Little information is available regarding compliance to low-CHO and low-fat dietary interventions. Most often, compliance has been operationalized through measurement of ketones [8,26,30,32,34] or in terms of the concentration of 1 ketone, β -hydroxybutyrate in the serum [22,32]. No urinary ketones were detectable in any subject in the present study because both diets contained sufficient amounts of carbohydrate to prevent ketosis. Thus, this comparison could not be made. Our approach to document compliance also included assessment through unannounced, repeated telephone 24-hour recalls by a trained dietitian. Based on nutrient analyses of these recalls, subjects did not differ with respect to compliance to either dietary regimen in this short

8-week trial. In another report in which dietary records of participants were assessed [22], adherence to the low-CHO dietary plan appeared to become more difficult without regular dietitian support because the proportion of energy from CHO consumed rose from 15% at 12 weeks to 30% at 24 weeks. This finding led us in part to select 15% of energy as CHO as the target for those assigned to the low-CHO plan but to classify intakes as less than 29% of energy as CHO as compliant to the low-CHO dietary plan in the present study.

There are some limitations to the findings of this study. Sample size is chief among these because individual differences may markedly alter the findings. Assessment of dietary noncompliance was not intended to exclude subjects from analyses but rather to identify (1) how dietary interventions were interpreted by participants in free living situations, (2) the difficulties encountered in adoption of either dietary plan, and (3) dietary patterns common to other dietary studies in which such information is reported. A run-in in longer trial would be preferable to identify who best could adhere to these dietary protocols. Although a trial of longer duration is a logical next step, important changes in lipids have been noted in the present study as well as in other reports of studies of shorter [27,33] or slightly longer duration [8,22,26,31,35,36]. Because TG [36] and hs-CRP [8] are measures that display large within-person variation, more repeat blood sampling on consecutive days may provide a more representative value for the subject.

Diets promoting weight loss and reducing atherogenic lipid profiles have typically been founded on the belief that lowering total dietary fat with particular attention to saturated fats would produce the most effective change in lipids and weight. However, increasingly, data gathered over the last few years challenges that notion and, in fact, indicate that better weight loss, better satiety, and better lipid profiles may result when the diet is low in carbohydrate and no restriction on fat content is adopted [4,8,22,30–35]. In this short 8-week study, improved lipid profiles with respect to LDL patterns and the similar compliance to such dietary plans suggests that further examination of such a pattern is worthy and must be examined in the context of long-term health. More long-term studies including both clinical trial and prospective cohort designs are needed before such a dietary plan can be advocated. This caution is based on the recent findings from the prospective Greek cohort from the European Prospective Investigation into Cancer and Nutrition [37] where the low-CHO, high-protein diet (corresponding to, eg, an increase of protein intake by about 15 g/d and a decrease of CHO intake by about 50 g/d) was associated with a 22% increase in overall mortality. In light of the observed reduction in small LDL particles with a moderately restricted CHO dietary plan in HTG adults, further study of this approach for longer duration is warranted in order to promote alternative, yet potentially effective, dietary plans that are associated with a reduction in cardiovascular events.

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