

High-monounsaturated fatty acid diets lower both plasma cholesterol and triacylglycerol concentrations¹⁻³

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ABSTRACT

Background: Low-fat diets increase plasma triacylglycerol and decrease HDL-cholesterol concentrations, thereby potentially adversely affecting cardiovascular disease (CVD) risk. High-monounsaturated fatty acid (MUFA), cholesterol-lowering diets do not raise triacylglycerol or lower HDL cholesterol, but little is known about how peanut products, a rich source of MUFAs, affect CVD risk.

Objective: The present study compared the CVD risk profile of an Average American diet (AAD) with those of 4 cholesterol-lowering diets: an American Heart Association/National Cholesterol Education Program Step II diet and 3 high-MUFA diets [olive oil (OO), peanut oil (PO), and peanuts and peanut butter (PPB)].

Design: A randomized, double-blind, 5-period crossover study design ($n = 22$) was used to examine the effects of the diets on serum lipids and lipoproteins: AAD [34% fat; 16% saturated fatty acids (SFAs), 11% MUFAs], Step II (25% fat; 7% SFAs, 12% MUFAs), OO (34% fat; 7% SFAs, 21% MUFAs), PO (34% fat; 7% SFAs, 17% MUFAs), and PPB (36% fat; 8% SFAs, 18% MUFAs).

Results: The high-MUFA diets lowered total cholesterol by 10% and LDL cholesterol by 14%. This response was comparable with that observed for the Step II diet. Triacylglycerol concentrations were 13% lower in subjects consuming the high-MUFA diets and were 11% higher with the Step II diet than with the AAD. The high-MUFA diets did not lower HDL cholesterol whereas the Step II diet lowered it by 4% compared with the AAD. The OO, PO, and PPB diets decreased CVD risk by an estimated 25%, 16%, and 21%, respectively, whereas the Step II diet lowered CVD risk by 12%.

Conclusion: A high-MUFA, cholesterol-lowering diet may be preferable to a low-fat diet because of more favorable effects on the CVD risk profile. *Am J Clin Nutr* 1999;70:1009–15.

KEY WORDS Plasma triacylglycerol, HDL cholesterol, LDL cholesterol, monounsaturated fatty acids, dietary fat, peanuts, peanut products, Step II diet, cardiovascular disease, humans

INTRODUCTION

Diet is the cornerstone of the prevention and treatment of cardiovascular disease (CVD). Currently, National Cholesterol

Education Program/American Heart Association Step I or Step II diets are typically recommended for lowering blood cholesterol concentrations. The primary objective of these diets is to lower saturated fat (8–10% and <7% of energy, respectively), cholesterol (300 or 200 mg/d), and total fat (<30% of energy). Typically, a Step I diet lowers total cholesterol and LDL cholesterol by ≈ 5 –7% (1–3). A Step II diet can lower total cholesterol and LDL cholesterol an additional 3–7% (1, 2). In these diets, saturated fat energy is replaced by carbohydrate, resulting in a low-fat, high-carbohydrate diet. Although these diets have beneficial effects on total cholesterol and LDL cholesterol, they increase plasma triacylglycerol concentrations and decrease HDL-cholesterol concentrations, thereby potentially adversely affecting CVD risk. This has caused some to question whether a Step I or Step II diet is the ideal diet for maximally reducing CVD risk (4, 5).

The alternative diet that has attracted much attention recently is a high-monounsaturated fatty acid (MUFA), cholesterol-lowering diet, in which saturated fat energy is replaced by MUFAs, resulting in a diet higher in total fat (ie, >30% of energy) than a Step I or Step II diet. In contrast with a Step I or Step II diet, a high-MUFA diet does not raise triacylglycerol nor lower HDL-cholesterol concentrations. To date, the primary food source of MUFAs that has been used is olive oil; canola oil has also been used. Little is known about how other food sources of MUFAs, such as peanuts, might affect the plasma lipid response to a cholesterol-lowering diet. Establishing the efficacy of other MUFA sources is important because it will increase the food options available in planning high-MUFA, cholesterol-lowering diets. Accordingly, flexibility in diet planning may enhance compliance with a cholesterol-lowering diet. Thus, the objective of the present study was to compare a Step II diet and 3 high-MUFA diets in which the MUFA source was olive oil, peanut oil, or peanuts and peanut butter with an average American diet (AAD).

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SUBJECTS AND METHODS

Experimental design

A 5-period crossover study design was used. Experimental periods lasted 24 d, followed by a short break (ie, lasting only 4–11 d and planned to avoid feeding subjects on holidays and special events). Breaks were intended to enhance compliance and not to serve as a washout period because changes in plasma lipids and lipoproteins with diet stabilize within 2–3 wk. Every subject consumed each experimental diet in a random, balanced order sequence. All meals were provided and subjects were required to eat the breakfast and dinner on weekdays at the Penn State Metabolic Diet Study Center; lunch and weekend meals were packed for consumption at a time and place of convenience. Subjects were not allowed to consume non-study foods or beverages, except for non-energy-containing seasonings and beverages. Compliance was monitored by body weight measurements and a dietary assessment questionnaire administered daily. Blood samples were collected 2 times within a 1-wk period at the end of each diet period. The length of the diet period was sufficient to achieve stabilization of blood lipids (3).

Subjects

The number of subjects recruited ($n = 26$) was based on our previous clinical studies and on the anticipated plasma lipid response to the experimental diets. Subjects were recruited by using various methods described by us previously (3, 6). Normocholesterolemic (mean total cholesterol: 4.88 mmol/L; mean LDL cholesterol: 3.05 mmol/L) men ($n = 9$) and women ($n = 13$) were studied. HDL-cholesterol concentrations of subjects were between the 25th and 90th percentile based on age and sex of National Cholesterol Education Program standards (1) (mean HDL cholesterol: 1.32 mmol/L; range: 0.78–1.99 mmol/L), and triacylglycerol concentrations were below the 90th percentile based on age and sex (mean triacylglycerol: 1.16 mmol/L; range: 0.5–2.26 mmol/L). All subjects were healthy (as determined by a self-reported health questionnaire and blood chemistry analysis), aged 21–54 y (\bar{x} : 34 y), and had a body mass index (BMI; in kg/m²) of 20–27. Our previous studies have shown that men and women of all ages respond to a cholesterol-lowering diet similarly. Thus, there was no need to control for sex or age (3). This study was approved by the Biomedical Committee of the Institutional Review Board at the Pennsylvania State University.

Experimental diets

The nutrient profiles of 4 cholesterol-lowering diets were compared with that of an AAD (Table 1). One cholesterol-lowering

diet was low in total fat (Step II diet) and 3 were high in total fat (and monounsaturated fat) provided by olive oil (OO), peanut oil (PO), or peanuts and peanut butter (PPB). The AAD was included as a reference diet to approximate the typical diet that is consumed widely today. A Step II diet was used because it is the diet recommended to achieve the maximal cholesterol-lowering response. This low-fat diet provides 7% of energy from SFAs, 25% from total fat, and 200 mg cholesterol/d. The 3 higher-fat (34–36% of energy), cholesterol-lowering diets (ie, OO, PO, and PPB) were designed to provide the same amount of saturated fat and cholesterol as the Step II diet; however, carbohydrate energy was replaced with MUFAs from OO, PO, or PPB. MUFAs provided 17–21% of energy in these diets. The diets were designed to provide the same amount of total fat as the AAD. To achieve this amount of fat in the diet, each MUFA source provided one-half of the fat in the diet. In addition to a somewhat variable MUFA content of these diets, polyunsaturated fatty acids (PUFAs) also varied modestly (ie, from 6% to 10% of energy). Olive oil was selected as one MUFA food source because it has been frequently tested experimentally. Peanut products were also tested because they are a popular food rich in MUFAs and have not been extensively evaluated in experiments. Two experimental diets with different peanut sources were tested to assess whether there might be a lipid-lowering effect associated with the protein moiety of peanuts in addition to that expected from the fat component. The experimental design not only enabled us to compare 2 different food sources of MUFAs but also allowed us to evaluate whether peanut products exert a cholesterol-lowering effect that is independent of MUFAs.

Validation of diet composition

A 7-d menu cycle was planned by using the NUTRITIONIST IV database (N-Squared Computing, First DataBank Division, San Bruno, CA) and all menus were designed to be nutritionally adequate. The macronutrient profiles of the 5 experimental diets were analyzed chemically to validate the diet composition. Validation samples were collected as follows. Food preparation was identical to that used in preparing each experimental diet; the 10.5-MJ (2500 kcal) diet was chosen as the representative sample. Breakfast, lunch, dinner, and a snack for the 7-d menu cycle were combined in a container and frozen at -20°C . The sample was then thawed, finely ground, subsampled, frozen, and stored at -20°C until analyzed. Total fat was determined by ether extraction of the oven-dried sample, protein was determined by using the Kjeldahl method, and carbohydrate was determined from the difference. SFAs, MUFAs, and PUFAs were analyzed by gas chromatography. The

TABLE 1

Assayed values of macronutrients, fatty acids, and cholesterol of the experimental diets¹

Dietary constituent	AAD	Step II	OO	PO	PPB
Carbohydrate (% of energy)	50	59	50	50	47
Protein (% of energy)	16	16	16	16	17
Fat (% of energy)	34	25	34	34	36
SFAs	16	7	7	7	8
MUFAs	11	12	21	17	18
PUFAs	7	6	6	9	10
Cholesterol (mg/d) ²	400	200	200	200	200

¹SFA, saturated fatty acids; MUFAs, monounsaturated fatty acids; PUFAs, polyunsaturated fatty acids; PPB, peanut and peanut butter diet; PO, peanut oil diet; OO, olive oil diet; Step II, American Heart Association/National Cholesterol Education Program Step II diet; AAD, average American diet.

²Estimated by using the NUTRITIONIST IV database (N-Squared Computing, San Bruno, CA).

assayed experimental diets (Table 1) met the target nutrient goals established initially and were consistent with the nutrient database values obtained from the development of the 7-d menu cycle.

Laboratory analyses of lipids, lipoproteins, and apolipoproteins

All blood samples were collected according to a standardized protocol, and serum aliquots were stored at -80°C until the end of the study, when all samples were analyzed. Serum concentrations of total cholesterol, HDL cholesterol, and triacylglycerol were determined by enzymatic assays. HDL cholesterol was determined after precipitation of apolipoprotein (apo) B-containing lipoproteins with dextran sulfate (molecular weight: 50000). LDL-cholesterol concentrations were calculated as mg/dL with use of the Friedewald equation (1): $\text{LDL cholesterol} = \text{total cholesterol} - (\text{HDL cholesterol} + \text{triacylglycerol}/5)$, then converted to mmol/L by dividing by 38.7. The within-laboratory CVs were 1.9% for total cholesterol and $\leq 2.5\%$ for HDL cholesterol. Rate immunonephelometry (Beckman Array; Beckman Instruments, Fullerton, CA) was used to measure apo B and apo A-I; Macra Lp(a) ELISA (Strategic Diagnostics Inc, Newark, DE) was used to determine lipoprotein(a) [Lp(a)] concentrations. The intraassay CV of the apoprotein assays was $<6\%$.

Statistical procedures

All data analyses were performed by using SAS (version 6.11; SAS Institute Inc, Cary, NC). Data are expressed as least-squares means \pm SEs, and any effects of diet or feeding period were tested by using analysis of variance (ANOVA). Tukey-Kramer adjusted P values were used to determine statistical differences between diets for each of the following variables: serum total cholesterol, LDL cholesterol, HDL cholesterol, triacylglycerol, apo A, apo B, Lp(a), ratio of total to HDL cholesterol, and ratio of LDL to HDL cholesterol. Probability values <0.05 were considered significant.

RESULTS

Twenty-two subjects completed all 5 diet periods and 2 completed 4 of the 5 diet periods. These 2 subjects left the study because of waning enthusiasm about continued study participation, principally because of the length of the study (ie, >6 mo).

Two other subjects relocated to different geographical areas early in the study (after participation in 1 or 2 diet periods). Results are reported for 22 subjects (results were identical when data from all 24 subjects were analyzed for the diet periods completed). Compliance with the experimental diets was judged to be excellent on the basis of the results of a variety of assessment techniques described previously (3). Each subject's weight was maintained throughout the study (± 1 kg).

The lipid, lipoprotein, and apolipoprotein data of subjects consuming the 5 experimental diets are given in **Table 2**, and the LDL-cholesterol, HDL-cholesterol, and triacylglycerol responses to the 4 cholesterol-lowering diets are compared in **Figure 1**. All 4 cholesterol-lowering diets reduced total cholesterol by $\approx 10\%$ (NS only for the Step II diet; $P < 0.05$ for the high-MUFA diets) and LDL cholesterol by $\approx 14\%$ compared with the AAD ($P < 0.05$). Changes in apo B paralleled those observed for LDL cholesterol. The high-MUFA diets did not lower HDL cholesterol whereas the Step II diet lowered it by 4% compared with the AAD ($P < 0.05$). Although not significant, there was a trend for HDL cholesterol to be higher in subjects consuming the high-MUFA diets than the Step II diet. There were no significant differences in apo A-I concentrations, irrespective of the diet fed. Triacylglycerol concentrations were significantly higher after consumption of the Step II diet than after all other cholesterol-lowering (high-MUFA) diets ($P < 0.05$; by 21%). Importantly, triacylglycerol concentrations were 13% lower ($P < 0.05$) when subjects consumed the high-MUFA diets than the AAD. Lp(a) concentrations were significantly higher ($P < 0.05$) in subjects when consuming the Step II diet than when consuming the PO diet; no other treatment effects were observed. The ratio of total to HDL cholesterol was significantly higher ($P < 0.05$) in subjects consuming the AAD than in those consuming the high-MUFA diets and not significantly different from that of subjects consuming the Step II diet. The ratio of LDL to HDL cholesterol was significantly lower ($P < 0.05$) in subjects consuming the cholesterol-lowering diets than in those consuming the AAD.

The magnitude of the reduction in LDL-cholesterol concentrations achieved by the 4 cholesterol-lowering diets depended on subjects' initial LDL-cholesterol concentrations (**Figure 2**). In general, subjects with higher LDL-cholesterol concentrations had a greater reduction in LDL cholesterol in response to the cholesterol-lowering diets than did those with lower concentrations.

TABLE 2

Lipid and lipoprotein endpoint results for the experimental diets¹

	AAD	Step II	OO	PO	PPB
Total cholesterol (mmol/L)	5.41 ± 0.23^a	4.92 ± 0.23^a	4.79 ± 0.23^b	4.93 ± 0.23^b	4.82 ± 0.23^b
LDL cholesterol (mmol/L)	3.52 ± 0.20^a	3.01 ± 0.20^b	2.98 ± 0.20^b	3.13 ± 0.20^b	3.03 ± 0.20^b
HDL cholesterol (mmol/L)	1.29 ± 0.11^a	1.24 ± 0.11^b	1.28 ± 0.11^{ab}	1.26 ± 0.11^{ab}	1.26 ± 0.11^{ab}
Triacylglycerol (mmol/L)	1.33 ± 0.13^a	$1.48 \pm 0.13^{a,2}$	1.15 ± 0.13^b	1.18 ± 0.13^b	1.16 ± 0.13^b
Apolipoprotein A (g/L)	1.54 ± 0.10	1.50 ± 0.10	1.52 ± 0.10	1.49 ± 0.10	1.48 ± 0.10
Apolipoprotein B (g/L)	1.01 ± 0.05^a	0.95 ± 0.05^b	0.92 ± 0.05^b	0.95 ± 0.05^b	0.92 ± 0.05^b
Lipoprotein(a) (g/L) ³	$0.12 \pm 0.06^{a,b}$	0.14 ± 0.06^a	$0.12 \pm 0.06^{a,b}$	0.12 ± 0.06^b	$0.13 \pm 0.06^{a,b}$
Total:HDL cholesterol	4.5 ± 0.3^a	$4.3 \pm 0.3^{a,b}$	4.1 ± 0.3^b	4.2 ± 0.3^b	4.1 ± 0.3^b
LDL:HDL cholesterol	3.0 ± 0.3^a	2.7 ± 0.3^b	2.6 ± 0.3^b	2.7 ± 0.3^b	2.7 ± 0.3^b

¹Least-squares mean \pm SE; $n = 22$. Within a row, values with different superscript letters are significantly different, $P < 0.05$ (with Tukey-Kramer adjustment). AAD, average American diet; Step II, American Heart Association/National Cholesterol Education Program Step II diet; OO, olive oil diet; PO, peanut oil diet; PPB, peanut and peanut butter diet.

²Different from AAD, $P = 0.06$.

³ $n = 20$; subjects whose values were <0.05 g/L for all diet treatments were dropped from the analysis (J Judd, personal communication, 1998).

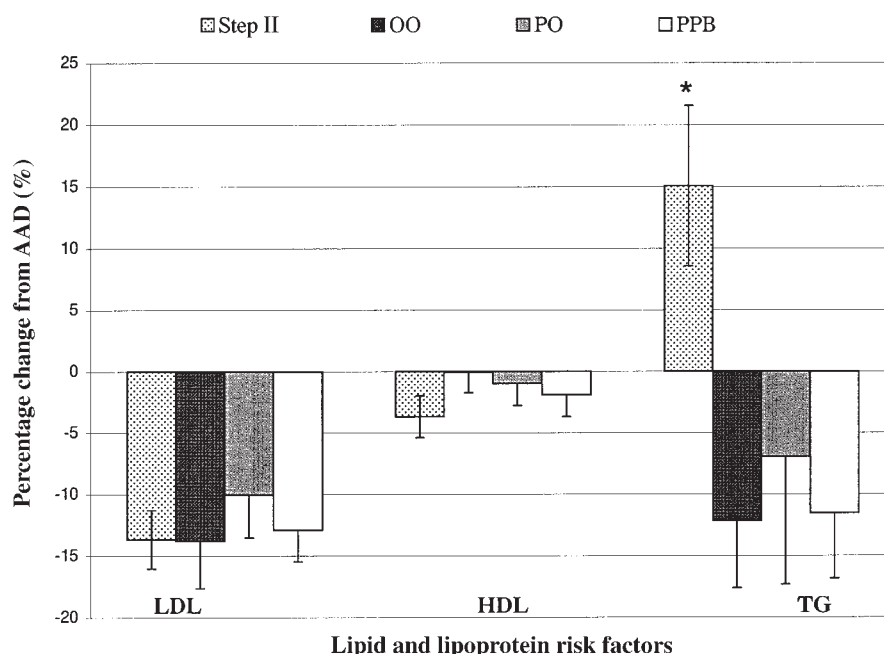


FIGURE 1. Effects of 4 cholesterol-lowering diets [Step II, olive oil diet (OO), peanut oil diet (PO), peanut and peanut butter diet (PPB)] on LDL-cholesterol, HDL-cholesterol, and triacylglycerol (TG) concentrations ($n = 22$) compared with an average American diet (AAD). Significantly different from the other 3 diets, $P = 0.028$. Values are least-squares means \pm SEs.

To assess how changes in plasma lipids correspond to changes in CVD risk status, we estimated the change in risk expected for the lipid and lipoprotein responses as follows: a 1% decrease in LDL cholesterol decreases CVD risk by $\approx 1.5\%$ (7–9), a 1-mg (0.026 mmol/L) decrease in HDL cholesterol increases CVD risk by 2–3% (10), and a 1-mmol/L (89 mg/dL) increase in triacylglycerol increases CVD risk by 25% (11). The average 14% reduction in LDL cholesterol achieved with all 4 cholesterol-lowering diets would be expected to decrease CVD risk by $\approx 21\%$ (Figure 3). The 0.026–0.052-mmol/L (1–2 mg/dL) increase in HDL cholesterol in subjects consuming the higher-fat, cholesterol-lowering diets (OO, PO, and PPB) would further decrease CVD risk by 2–6% compared with a Step II diet. In addition, the average decrease in triacylglycerol of 0.16 mmol/L with the high-MUFA, cholesterol-lowering diets (OO, PO, and PPB) would be expected to decrease the risk of CVD by 4%. Collectively, as much as a 25%, 16%, and 21% decrease in CVD risk would be expected with implementation of the OO, PO, and PPB diets, respectively. In contrast, the Step II diet decreased HDL cholesterol by 0.05 mmol/L and increased triacylglycerol by 0.145 mmol/L, thereby increasing CVD risk by 4.8% and 3.7%, respectively. Overall, the Step II diet would be expected to lower CVD risk by $\approx 12\%$. With use of these estimates, it appears that the high-MUFA diets (the OO, PO, and PPB diets in particular) have a more favorable effect on CVD risk.

DISCUSSION

The results of the present study provide further evidence that higher-fat diets that are high in MUFAs and low in SFAs lower total and LDL cholesterol to a degree similar to that observed for a lower-fat, cholesterol-lowering diet (12–15). Moreover, in agreement with other reports (12, 14), we showed that the high-MUFA

diets studied had the added benefit of not increasing triacylglycerol concentrations or lowering HDL-cholesterol concentrations, as the Step II diet tends to.

The finding that a high-MUFA diet favorably affected LDL-cholesterol as well as HDL-cholesterol and triacylglycerol concentrations (12–14, 16, 17) has important public health implications. The significance of both LDL- and HDL-cholesterol concentrations in affecting CVD risk is well established. Controlled clinical trials have shown that a 1% reduction in total and LDL-cholesterol concentrations results in an $\approx 1.5\%$ decrease in the incidence of CVD (7–9). Moreover, the risk of CVD is increased by 2–3% for every 0.026-mmol/L (1 mg/dL) decrease in HDL cholesterol (10). Recently, an elevated triacylglycerol concentration was shown to be a univariate predictor of CVD (18). Specifically, a 1-mmol/L increase in triacylglycerol is associated with a 14% increase in CVD risk in men and a 37% increase in women (11). That the Step II diet tended to increase Lp(a) concentrations compared with the high-MUFA diets is consistent with results of our previous studies (3) and is of potential significance because an elevated Lp(a) concentration is also associated with an increased risk of CVD (19). Collectively, these findings point to the fact that a high-MUFA diet may be preferable to a low-fat diet because of more favorable effects on the CVD risk profile.

An important finding reported in the present study is the hypo-triacylglycerolemic effect (13% reduction) of a high-MUFA diet compared with an AAD. Specifically, when carbohydrate and total fat intake were constant and SFAs were replaced with MUFAs, there was a significant triacylglycerol-lowering effect observed. This finding has not been typically reported. In large part, this reflects the experimental design that has been used by other investigators to compare the plasma lipid effects of a high-MUFA diet with those of a low-fat diet. These studies have typically replaced carbohydrate with MUFA, which makes it difficult to evaluate the

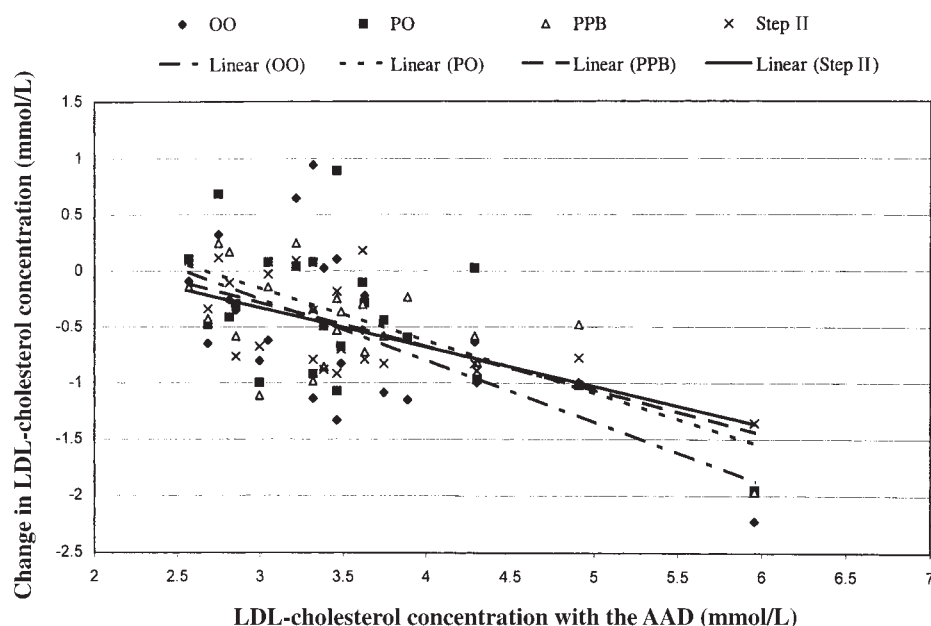


FIGURE 2. Effects of baseline LDL-cholesterol concentrations on LDL-cholesterol response. The Pearson correlation coefficients between LDL cholesterol with an average American diet (AAD) and the change in LDL cholesterol are as follows: $r = 0.416$, $P = 0.002$ for the Step II diet; $r = 0.376$, $P = 0.002$ for the olive oil diet (OO); $r = 0.347$, $P = 0.004$ for the peanut oil diet (PO); and $r = 0.398$, $P = 0.002$ for the peanut and peanut butter diet (PPB). The regression equations are: $y = -0.36x + 29.57$ for the Step II diet, $y = -0.55x + 54.56$ for the olive oil diet, $y = -0.47x + 48.83$ for the peanut oil diet, and $y = -0.39x + 34.86$ for the peanut and peanut butter diet ($n = 22$). The slopes of the lines for each diet were not significantly different.

independent effects of MUFAs when carbohydrate intake changes. This is important, given the triacylglycerol-raising effects of dietary carbohydrate and, specifically, a low-fat, high-carbohydrate diet. Thus, we propose that when carbohydrate is held constant and MUFAs replace SFAs, MUFAs elicit an independent triacylglycerol-lowering effect. We noted a similar response in the Dietary Effects on Lipoproteins and Thrombogenic Activity (DELTA) Study (20), which used an experimental design comparable with that used in the present study.

In addition to the beneficial effects on total cholesterol, LDL cholesterol, triacylglycerol, and HDL cholesterol, MUFA diets have been shown to reduce LDL oxidative susceptibility in vitro (21–24). Limited data suggest that MUFAs also may decrease platelet aggregation (25), increase fibrinolysis (26), and increase bleeding time (27), thereby protecting against thrombogenesis. Rasmussen et al (28) reported that a very-high-MUFA diet (30% of energy from MUFAs) significantly reduced systolic (by 6 mm Hg) and diastolic (by 6 mm Hg) blood pressure in subjects with type 2 diabetes. All of these findings collectively suggest that high-MUFA diets may have multiple benefits for CVD risk factors.

Because most serum triacylglycerol is transported by VLDL, hepatic production or rate of secretion of VLDL triacylglycerol and hydrolysis or removal of circulating triacylglycerol are 2 key determinants of serum triacylglycerol concentrations. The underlying mechanism for the hypotriacylglycerolemic effect of MUFAs is not clear. However, McNamara (29) proposed 2 complementary mechanisms that may be involved: 1) changes in the composition of VLDL and 2) changes in the expressed activities of the enzymes and proteins involved in intravascular processing and catabolism of VLDL, both of which would decrease plasma triacylglycerol concentrations. The fatty acid composition of VLDL triacylglycerol, which is affected by dietary fatty acid composition, is a determi-

nant for the conversion of VLDL into other lipoproteins and the metabolism of triacylglycerols (30). Therefore, the rates of VLDL production and clearance of triacylglycerol may be altered as a result of the amount and type of fat in the diet (31, 32).

Campos et al (31) reported a significant increase in both lipoprotein lipase and hepatic lipase activities in subjects consuming a high-fat (46% of energy) compared activities when consuming a low-fat (24% of energy) diet, which likely accounted for the reported decrease in total triacylglycerol (by 0.56 mmol/L, or 50 mg/dL) resulting from the catabolism and extrahepatic catabolism of triacylglycerol-rich lipoproteins. The finding of Montalto and Bensadoun (32) that oleic acid does not increase lipoprotein lipase secretion in cell culture suggests that the decrease in triacylglycerol reported in the present study may reflect an increase in triacylglycerol removal that is due to an effect of total fat and a specific effect of MUFA. In support of this proposed MUFA-mediated mechanism, Brousseau et al (33) observed significantly lower hepatic apo C-III messenger RNA concentrations in cynomolgus monkeys in response to high-MUFA and -PUFA than in response to high-SFA diets. The authors suggest that the decrease in apo C-III content of HDL in monkeys fed high-MUFA and high-PUFA diets may be due to a reduction in the transfer of VLDL constituents to HDL as a result of a decreased production of nascent VLDL particles. Thus, there are potentially multiple mechanisms that account for the MUFA effect observed. Additional studies are needed to clarify the mechanism or mechanisms by which MUFAs elicit a triacylglycerol-lowering effect.

The results of our study show that another food source rich in MUFAs, peanut products (ie, peanuts, peanut butter, and peanut oil), can be used in designing high-MUFA diets. The availability of an additional food source rich in MUFAs is invaluable within the context of diet planning. With greater flexibility in

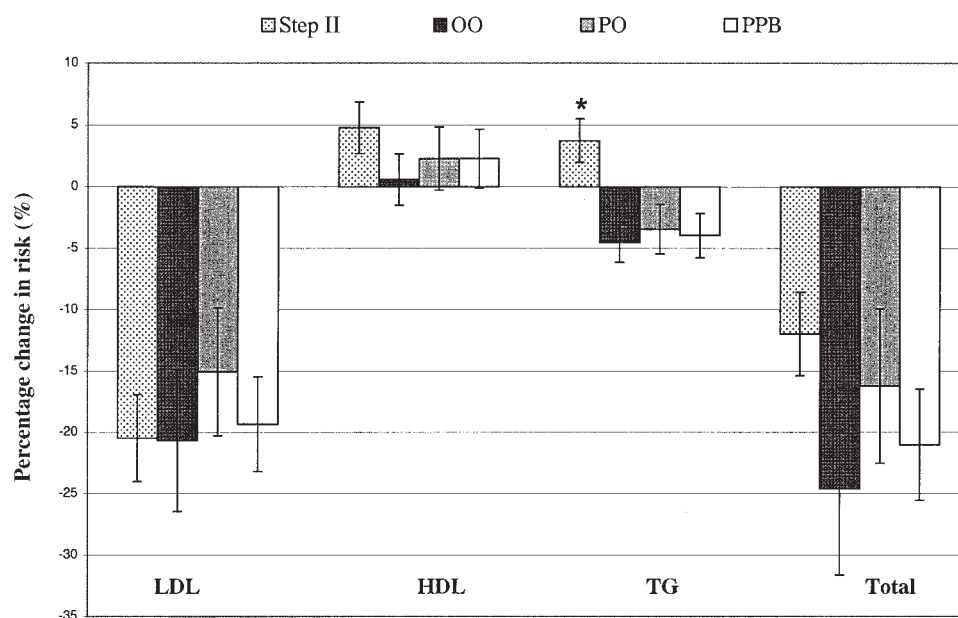



FIGURE 3. Effects of LDL-cholesterol, HDL-cholesterol, and triacylglycerol (TG) concentrations on cardiovascular disease (CVD) risk reduction in response to Step II, olive oil (OO), peanut oil (PO), and peanut and peanut butter (PPB) diets ($n = 22$). *Significantly different from the other 3 diets, $P = 0.005$. Values are least-squares means \pm SEs.

diet planning as a result of multiple food sources of MUFA, it is not unreasonable to speculate that compliance with a cholesterol-lowering diet can be enhanced. Moreover, because peanuts and peanut products also are a rich source of other nutrients, their inclusion in the diet can favorably affect the nutrient profile of the diet. Further impetus for recommending inclusion of nuts in the diet comes from recent epidemiologic studies that show a marked reduction in CVD risk with frequent nut consumption (34–36). Several studies have reported a dose-response relation between nut consumption and the incidence of CVD. For example, consumption of nuts 5 times/wk has been shown to reduce the incidence of CVD by 50%, whereas consumption 1–4 times/wk decreases the incidence by 25% (34). Thus, there is provocative evidence that there are cardioprotective nutrients or factors in nuts.

From a public health perspective, it is now timely to reevaluate what the optimal diet is for lowering risk of CVD. On the basis of the results of the present study, it appears that a high-MUFA, cholesterol-lowering diet is superior to a low-fat diet such as the Step II diet. Although the reduction in CVD risk due to a decrease in LDL cholesterol is similar for both a high-MUFA diet and a low-fat diet (Step II diet), a high-MUFA diet lowers triacylglycerol and does not decrease HDL cholesterol. In contrast, a Step II diet increases triacylglycerol and lowers HDL cholesterol, thereby possibly negating some of the beneficial effects of reducing LDL cholesterol. Collectively, the results of the present study as well as evidence from epidemiologic studies provide compelling evidence for conducting further studies to evaluate the long-term effects of high-MUFA, cholesterol-lowering diets that include peanuts and nuts in different population groups on both total morbidity and mortality and that due to CVD. Currently, however, the evidence available is sufficient to consider a high-MUFA, chole-

sterol-lowering diet that includes peanuts and nuts as an acceptable, and perhaps preferable, dietary approach for most favorably affecting CVD risk status. 

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