Serum lipid response to the graduated enrichment of a Step I diet with almonds: a randomized feeding trial[1–3]

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ABSTRACT

Background: Frequent consumption of nuts may lower the risk of cardiovascular disease by favorably altering serum lipid and lipoprotein concentrations.

Objective: We compared the effects of 2 amounts of almond intake with those of a National Cholesterol Education Program Step I diet on serum lipids, lipoproteins, apolipoproteins, and glucose in healthy and mildly hypercholesterolemic adults.

Design: In a randomized crossover design, 25 healthy subjects (14 men, 11 women) with a mean (± SD) age of 41 ± 13 y were fed 3 isoenergetic diets for 4 wk each after being fed a 2-wk run-in diet (containing 34% of energy from fat). The experimental diets included a Step I diet, a low-almond diet, and a high-almond diet, in which almonds contributed 0%, 10%, and 20% of total energy, respectively.

Results: Inverse relations were observed between the percentage of energy in the diet from almonds and the subject's total cholesterol (P value for trend < 0.001), LDL-cholesterol ($P < 0.001$), and apolipoprotein B ($P < 0.001$) concentrations and the ratios of LDL to HDL cholesterol ($P < 0.001$) and of apolipoprotein B to apolipoprotein A ($P < 0.001$). Compared with the Step I diet, the high-almond diet reduced total cholesterol (0.24 mmol/L or 4.4%; $P = 0.001$), LDL cholesterol (0.26 mmol/L or 7.0%; $P < 0.001$), and apolipoprotein B (6.6 mg/dL or 6.6%; $P < 0.001$); increased HDL cholesterol (0.02 mmol/L or 1.7%; $P = 0.08$); and decreased the ratio of LDL to HDL cholesterol (8.8%; $P < 0.001$).

Conclusions: Isoenergetic incorporation of ≈68 g of almonds (20% of energy) into an 8368-kJ (2000-kcal) Step I diet markedly improved the serum lipid profile of healthy and mildly hypercholesterolemic adults. Total and LDL-cholesterol concentrations declined with progressively higher intakes of almonds, which suggests a dose-response relation. *Am J Clin Nutr* 2003;77:1379–84.

KEY WORDS Serum cholesterol, LDL cholesterol, HDL cholesterol, apolipoproteins, cardiovascular disease, Step I diet, monounsaturated fatty acids, nuts, almonds, humans

INTRODUCTION

Hypercholesterolemia is one of the major risk factors for cardiovascular disease (CVD). A cornerstone of treatment for hypercholesterolemia is dietary modification (1), which has the objective of improving the ratio of LDL to HDL cholesterol. Dietary recommendations for lowering LDL cholesterol have centered on restriction of foods high in saturated fat; modification of visible fats such as butter, margarine, and oils; or both. These diets are low to moderate in total and saturated fat (2), and they require individuals to reduce or avoid certain foods, which is a challenge to long-term adherence (3). Whereas the National Cholesterol Education Program (NCEP) Step I diet is known to reduce LDL cholesterol by 3–10% (4, 5), that reduction is often accompanied by an increase in triacylglycerol and a decrease in HDL cholesterol, both of which contribute to an increased risk of CVD (6). In addition, high-carbohydrate, low-fat diets produce unfavorable shifts in LDL-cholesterol subclasses, which produces further atherogenic risk (7).

Because the NCEP Step I diet falls short of favorably altering a person's overall lipid profile, alternative diets that will accomplish this goal should be identified. In this context, specific whole foods that not only lower LDL cholesterol but also favorably influence other blood lipids could be used as an adjunct to usual or cholesterol-lowering diets. Increased consumption of foods from the whole-grain and legume families was shown to reduce the risk of CVD (8), possibly by lowering LDL cholesterol. Garlic, soy, and fish were shown to lower LDL cholesterol (9, 10) and to favorably modify other lipids and lipoproteins (11). In fact, the latest edition of the American Heart Association dietary guidelines includes specific food recommendations (12).

Epidemiologic studies (13–15) have consistently reported an inverse relation between the incidence of CVD and the frequent consumption of nuts, even after adjustment for confounding variables. Almonds, which are high in monounsaturated fat, are commonly consumed in many parts of the world (16). Almonds are unique in that they have significant amounts of protein and have the highest concentration of α-tocopherol of all nuts (17). A cholesterol-lowering effect of almonds compared with typical Western diets in healthy and hypercholesterolemic subjects was reported in 2 field trials (18, 19) and 1 clinical trial (20). Our study furthers this investigation by comparing the effects of almond consumption in 2 amounts on multiple serum lipid values with those of a Step I diet in a well-controlled experimental setting.

SUBJECTS AND METHODS

Subject recruitment

Potential study subjects were healthy men and women aged 20–60 y. The selection process included the completion of a

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screening questionnaire, an informational group meeting, a personal interview, and cholesterol testing. Exclusion criteria were as follows: fasting serum cholesterol concentrations < 15th or > 90th percentile for age, sex, and race (2); fasting triacylglycerol concentrations > 2.26 mmol (200 mg/dL); current consumption of nuts > 2 times/wk; high intake of caffeinated beverages (> 3 times/d); consumption of > 2 alcoholic drinks/wk; food allergies; cigarette smoking; history of chronic or metabolic disease; regular use of medications known to affect blood lipids; body mass index (BMI; in kg/m^2) > 30; and, for women, either irregular menses or hormone use within the past 5 y.

Study design

This was a randomized, crossover (3 × 3 Latin-square), controlled feeding trial. The sample size was computed on the basis of the decreases in serum cholesterol that were due to the consumption of nuts as reported in previous studies (reviewed in 21), and it provided for a 10% dropout rate. A sample size of 24 was determined to provide > 90% power to detect significant differences between treatments at $P < 0.05$. The Institutional Review Board of Loma Linda University approved the study. All participants gave written informed consent. Participants who successfully completed the study received an incentive of $300.

Diet intervention

The study protocol consisted of a 2-wk run-in (baseline) phase during which the subjects consumed a Western-type diet containing 34% energy from fat and then were randomly assigned to 1 of 6 possible sequences of the 3 treatment diets (Step I diet, low-almond diet, and high-almond diet). The 3 diets were fed to subjects in a crossover fashion for 4 wk each. A wash-out phase was not included between treatment diets because serum lipids and lipoproteins are known to stabilize within 3 wk (22).

The treatment diets were isoenergetic. The low- and high-almond diets were designed to replace 10% and 20%, respectively, of energy of the Step I diet with almonds. This was achieved by making proportional reductions to all foods in the Step I diet menus to accommodate the energy supplied by the almonds. The diets thus differed in the percentage of energy from fat: Step I diet, 30%; low-almond diet, 35%; and high-almond diet, 39%.

The study featured a highly controlled feeding protocol. On Sunday through Friday of each week, subjects ate breakfast and dinner at the Loma Linda University Metabolic Kitchen. Lunch meals and all Saturday meals were packaged for consumption away from the Metabolic Kitchen. All meals were prepared at the Metabolic Kitchen, and 9 menus were used in rotation to provide mealtime variety. Almonds were served on their own or incorporated into cold foods (eg, salads) or hot foods (eg, pizza). Menus were designed for 7 amounts of energy intake, ranging from 7533 to 15 066 kJ/d (1800–3600 kcal/d). Subjects were weighed twice per week, and their energy intake was adjusted as needed to maintain stable body weight.

Quality control

Subjects consumed all research protocol foods provided in the study and refrained from eating any nonprotocol foods. Quality control and compliance with the protocol were ensured among study participants by the following means: *1*) foods were weighed to the nearest gram before being served to the participants; *2*) a senior investigator supervised mealtimes and ensured the complete intake of all study foods; *3*) participants maintained a daily diary in which they recorded any deviations from the study protocol, and the diaries were reviewed by a senior investigator; *4*) randomly collected diet samples (n = 27) covering all 3 treatment diets were homogenized and analyzed (23) for nutrient composition (Covance Laboratories, Madison, WI); and *5*) the fatty acid composition of serum triacylglycerol was analyzed at the end of each diet period at the University of California, Davis, to provide an objective measure of dietary compliance.

Before determining the fatty acid composition of serum triacylglycerol, the lipids from serum were extracted by using chloroform:methanol (2:1, by vol), and individual lipid classes within each extract were separated by preparative thin-layer chromatography. Each lipid fraction was scraped from the plate and transesterified in 3 N methanolic HCl in a sealed vial under nitrogen atmosphere (100 °C for 45 min). The resulting fatty acid methyl esters were extracted from the mixture with hexane containing 0.05% butylated hydroxytoluene and were prepared for gas chromatography by the sealing of the hexane under nitrogen atmosphere. Fatty acid methyl esters were separated and quantified by capillary gas chromatography with the use of a gas chromatograph (model 6890; Hewlett-Packard, Wilmington, DE) equipped with a 30-m papillary column (DB-225MS; J & W Scientific, Folsom, CA) and a flame ionization detector (Agilent Technologies, Inc, Palo Alto, CA).

Data collection

Twelve-hour fasting blood samples were collected from subjects before breakfast on 2 alternate days at the end of each diet phase, including the run-in phase. Subjects were asked not to exercise before blood was drawn on the test days. Within 30 min of collection, the serum was separated by centrifugation at 1500 × g for 15 min at 4 °C. Serum was then aliquoted and stored immediately at −80 °C until it was analyzed. The Nutritional Assessment Core at the University of California, Davis (the Clinical Nutrition Research Unit, which is supported by NIH NIDDK grant 35747), performed the serum lipid and lipoprotein analyses. Concentrations of total, LDL, and HDL cholesterol; triacylglycerol; and glucose (24–26) were determined by enzymatic colorimetric assays with the use of the Bayer 550 Express Chemistry analyzer (Bayer Corp, Tarrytown, NY). Before analysis, HDL was separated from serum with a magnetically enhanced dextran sulfate (molecular weight: 50 000) and magnesium chloride reagent by selective precipitation of apolipoprotein B (apo B)–containing lipoproteins. All samples were analyzed in duplicate with an internal control and standard curve.

The serum concentrations of apo A and apo B (27) were established with the use of a nephelometer (Beckman Coulter, Inc, Brea, CA) to measure the rate of light-scatter resulting from an immunoprecipitation reaction. A commercial calibrator serum was used to set a reference point with which the sample's peak rate of increase of light scatter was compared and then converted into concentration units (mmol/L). Lipoprotein(a) concentrations (28) were measured turbidimetrically (DiSarin Inc, Stillwater, MN).

Statistical analysis

Statistical analyses were performed with SAS software, version 8.0 (SAS Institute Inc, Cary, NC). Tests for trends in blood lipid variables in response to dietary treatments were conducted by analysis of covariance in a mixed-effects model, which included a random-effect term for subjects, a fixed-effect term for period, and a covariate representing the percentage of energy in the diet

TABLE 1
Planned and analyzed composition of the Step I, low-almond, and high-almond diets[1]

	Step I diet		Low-almond diet		High-almond diet	
Nutrient	Planned	Analyzed	Planned	Analyzed	Planned	Analyzed
Energy						
(kJ/d)	10 195	10 133	10 141	10 401	10 090	10 242
(kcal/d)	2437	2422	2424	2486	2412	2448
Protein (% of energy)	13.9	14.0	13.9	13.4	14.0	14.1
Carbohydrate (% of energy)	57.1	55.8[2]	53.0	51.2[2]	48.8	46.0[2]
Fat (% of energy)	31.1	29.9	35.6	35.0	40.2	39.0
SFAs (% of energy)	9.5	8.2	9.2	8.0	8.8	7.7
MUFAs (% of energy)	12.7	12.1	16.3	16.5	19.9	19.4
Oleic acid	11.6	11.6	15.2	16.0	18.9	19.3
PUFAs (% of energy)	6.3	6.2	7.5	7.5	8.7	8.7
Linoleic acid	5.1	5.7	6.4	6.9	7.8	8.1
α-Linolenic acid	0.71	0.65	0.64	0.60	0.57	0.54
Cholesterol (mg/d)	240	202	216	163	192	140
Fiber (g/d)	28.1	—	29.9	—	31.9	—

[1]Planned composition was calculated with the use of FOOD PROCESSOR IV software, version 7.5 (ESHA Research, Salem, OR). Analyzed composition values were obtained from the chemical analysis of samples from the study diets. SFAs, saturated fatty acids; MUFAs, monounsaturated fatty acids; PUFAs, polyunsaturated fatty acids.

[2]Calculated by subtracting the values for fat and protein intake from those for total energy intake.

contributed by almonds. When the trend was significant, differences between pairs of diets were examined by analysis of variance by replacing the covariate in the above model with a fixed-effect classification term for diet. The significance of pairwise differences was assessed with the use of Tukey-Kramer–adjusted *P* values. All results with a *P* value of < 0.05 were considered statistically significant.

RESULTS

Subjects

During the run-in phase, subjects who were not in compliance with the study protocol either voluntarily withdrew ($n = 1$) or were removed from the study ($n = 5$). Of the 27 who were randomly assigned to the treatment diets, 25 completed the study. Two subjects were unable to comply with the diet protocol, and they dropped out of the study. The mean age of subjects who completed the study was 41 ± 13 y (range: 22–53 y). There were 11 women and 14 men in the study, and they represented 4 ethnic groups: white ($n = 10$), Hispanic ($n = 7$), Asian ($n = 5$), and African American ($n = 3$).

Diet composition and compliance

The nutrient composition of the treatment diets calculated with the use of FOOD PROCESSOR IV software, version 7.5 (ESHA Research, Salem, OR) was compared with the analyzed composition established through chemical analysis (**Table 1**). As planned, the percentage of energy from fat was higher in the low-almond (35%) and high-almond (39%) diets than in the Step I diet (30%). The incorporation of almonds into the diets resulted in small decreases in saturated fatty acids, small increases in polyunsaturated fatty acids, and a substantial increase in monounsaturated fatty acids (MUFAs). The nutrient content of the chemically analyzed diets matched that of the planned diets.

A high degree of correspondence was observed between the fatty acid composition of the diets and the serum triacylglycerol fraction in the subjects (**Table 2**). The percentage of total saturated fatty acids in serum decreased and that of MUFAs increased with increasing amounts of almonds in the diet. This trend is similar to that observed for the nutrient composition of the diets, and it validates the consumption of almonds and close adherence to the diets by the study participants.

Serum lipids and lipoproteins

The mean values for serum lipids, lipoproteins, glucose, and body weight at baseline and at the end of each dietary treatment are shown in **Table 3**. As the amount of energy from almonds in the diets increased, a significant dose-response trend was observed for total cholesterol, LDL cholesterol, apo B, LDL:HDL, and apo B:apo A. When compared with the Step I diet, the high-almond diet significantly reduced total cholesterol (0.24 mmol/L), LDL cholesterol (0.26 mmol/L), and apo B (6.6 mg/dL). Compared with the low-almond diet, the high-almond diet significantly reduced total cholesterol (0.19 mmol/L), LDL cholesterol (0.22 mmol/L), and apo B (5.7 mg/dL). LDL:HDL and apo B:apo A were significantly ($P < 0.001$) lower in the high-almond diet than in either the Step I diet or the low-almond diet.

The percentage change in serum lipid, lipoprotein, and apolipoprotein concentrations between baseline and each treatment diet are shown in **Figure 1**. After the high-almond diet phase, total and LDL-cholesterol concentrations were significantly lower than baseline values—7% and 9%, respectively. After the low-almond diet phase, total cholesterol decreased significantly (4%) and LDL cholesterol decreased nonsignificantly (3%) compared with baseline values. Compared with baseline, subjects had lower total and LDL-cholesterol concentrations after the Step I diet, but the difference was not significant. The Step I and low-almond diets reduced HDL cholesterol significantly compared with baseline (3% and 4%, respectively), whereas the high-almond diet did not alter the concentrations of HDL cholesterol. Moreover, LDL:HDL decreased significantly (8.6%) after the high-almond diet compared with baseline. Apo A increased significantly (4%) and apo B decreased significantly (8%) after the high-almond diet compared with baseline. Apo B:apo A decreased significantly after the low-almond (5%) and high-almond (12%) diets.

DISCUSSION

The results of this study indicate that an isoenergetic incorporation of almonds (20% of energy) into a Step I diet markedly

TABLE 2
Fatty acid composition of serum triacylglycerol after the 3 treatment diets[1]

Fatty acid	Step I diet	Low-almond diet	High-almond diet	*P* for trend[2]
	%	%	%	
SFAs	33.2 ± 0.7	33.2 ± 0.7	30.9 ± 0.7	0.01
MUFAs				
Oleic acid	32.7 ± 0.4	34.6 ± 0.4	36.3 ± 0.4	<0.001
PUFAs				
Linoleic acid	21.7 ± 0.1	20.9 ± 0.1	22.1 ± 0.1	0.43
α-Linolenic acid	1.63 ± 0.04	1.45 ± 0.04	1.24 ± 0.04	<0.001
SFAs:MUFAs	0.85 ± 0.02	0.83 ± 0.02	0.75 ± 0.02	<0.001

[1] Least-squares $\bar{x}$ ± SE; n = 25. SFAs, saturated fatty acids; MUFAs, monounsaturated fatty acids; PUFAs, polyunsaturated fatty acids.
[2] ANCOVA test for trend, with the use of the percentage of energy from almonds as the covariate.

improves the serum lipid profile of healthy and mildly hypercholesterolemic adults. Total and LDL-cholesterol concentrations decrease with progressively higher intakes of almonds, which suggests a dose-response relation. A 7% decrease in LDL cholesterol produced by the high-almond diet corresponds to a reduction in the incidence of CVD of ≈11% (1). This has important public health implications.

In addition to reducing LDL cholesterol, the high-almond diet produced marked decreases in apo B concentrations. Apo B is a component of VLDL and LDL in serum and thus reflects the concentration of potentially atherogenic lipoprotein particles, which confer CVD risk (29). We observed a trend of decreasing apo B concentration with increasing amounts of almonds in the diet, which suggested a decrease in both the LDL-cholesterol concentration and the number of LDL particles.

Our study did not show any significant changes in triacylglycerol concentrations with almond consumption. Others have shown that diets enriched with nuts that are high in MUFAs either reduced serum triacylglycerol (30, 31) or produced nonsignificant changes (18, 19) in normolipidemic individuals. A decrease in apo B concentration without a concurrent change in triacylglycerol suggests that there may be changes in the distribution of apo B between the different VLDL particles. Further analysis of VLDL particle size and distribution of apo B may provide a better understanding of potential mechanisms by which almonds and other foods rich in MUFAs influence triacylglycerol concentrations.

Although HDL cholesterol did not show a significant trend with almond consumption in this study, a monotonic trend was observed, ie, LDL:HDL and apo B:apoA decreased as the amount of almonds in the diet increased. Both ratios are considered to be more useful clinically in determining CVD risk than are the individual values of HDL cholesterol and apolipoproteins (32, 33).

Previous studies primarily investigated the lipid-lowering effects of almonds in comparison to those of diets that are high in total and saturated fat. Abbey et al (18) observed decreases in total and LDL cholesterol in normolipidemic men who consumed 84 g almonds/d compared with the values in men who consumed a saturated fat–rich Western diet. In a study of hypercholesterolemic men and women (19), the consumption of 100 g almonds/d produced a decrease in total and LDL-cholesterol concentrations compared with the consumption of a control diet high in saturated fat. In contrast, the current study, with the use of a well-controlled crossover design, found favorable effects of almonds on several serum lipid variables when compared with a widely prescribed cholesterol-lowering diet, the NCEP Step I diet. Our findings are in line with a recent clinical trial in subjects with hyperlipidemia, in which a supplement of almonds in a low-fat diet also resulted in improvements in several serum lipid risk factors for CVD (20).

It has been suggested that nuts in general may contain bioactive components besides fat that may affect LDL cholesterol and other CVD risk factors (34). The decreases in total and LDL cholesterol observed in our study were greater than those estimated from the

TABLE 3
Serum lipids, lipoproteins, glucose, and body weight at the end of each dietary treatment[1]

Variable	Baseline[2]	Step I diet[3]	Low-almond diet[3]	High-almond diet[3]	*P* for trend[4]
Cholesterol					
Total (mmol/L)	5.61 ± 0.14	5.41 ± 0.19[a]	5.36 ± 0.19[a]	5.17 ± 0.19[b]	<0.001
LDL (mmol/L)	3.86 ± 0.15	3.74 ± 0.21[a]	3.70 ± 0.21[a]	3.48 ± 0.21[b]	<0.001
HDL (mmol/L)	1.22 ± 0.04	1.18 ± 0.05	1.17 ± 0.05	1.20 ± 0.05	0.09
LDL:HDL	3.40 ± 0.20	3.40 ± 0.28[a]	3.40 ± 0.28[a]	3.10 ± 0.28[b]	<0.001
Triacylglycerol (mmol/L)	1.37 ± 0.13	1.52 ± 0.19	1.52 ± 0.19	1.47 ± 0.19	0.36
Apolipoprotein A (mg/dL)	135.2 ± 2.9	137.5 ± 4.5	138.6 ± 4.5	140.4 ± 4.5	0.08
Apolipoprotein B (mg/dL)	102.9 ± 4.2	100.3 ± 5.6[a]	99.4 ± 5.6[a]	93.7 ± 5.6[b]	<0.001
Apo B:Apo A	0.78 ± 0.04	0.75 ± 0.05[a]	0.74 ± 0.05[a]	0.69 ± 0.05[b]	<0.001
Lipoprotein(a) (mg/dL)	31.3 ± 5.0	30.9 ± 7.0	29.9 ± 7.0	30.7 ± 7.0	0.84
Glucose (mmol/L)	4.73 ± 0.06	4.70 ± 0.08	4.72 ± 0.08	4.71 ± 0.08	0.64
Body weight (kg)	71.0 ± 2.7	71.0 ± 2.7	71.2 ± 2.7	70.7 ± 2.7	0.11

[1] n = 25. Values within a row with different superscript letters are significantly different, $P < 0.05$ (ANOVA with Tukey-Kramer adjustment).
[2] $\bar{x}$ ± SE. Baseline not included in ANOVA or test for trend.
[3] Least-squares $\bar{x}$ ± SE.
[4] ANCOVA test for trend, with the use of the percentage of energy from almonds as the covariate.

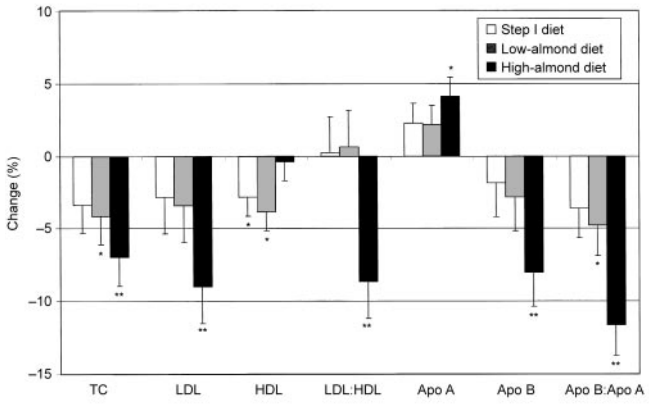

FIGURE 1. The mean (±SE) percentage change in serum lipids, lipoproteins, and apolipoproteins from baseline to the end of the 4-wk treatment diets ($n = 25$). TC, total cholesterol; apo A, apolipoprotein A; apo B, apolipoprotein B. *,**Significantly different from baseline diet (ANOVA with Tukey-Kramer adjustment): $^{*}P < 0.05$, $^{**}P < 0.01$.

fatty acid composition of the diets with the use of predictive equations (35–37). This suggests that the nonlipid components of almonds may play a role in lowering serum lipids. These nonlipid components may reduce other CVD risk factors well. Almonds have a high concentration of α-tocopherol, an antioxidant that has been associated with a lower risk of CVD (15, 38), possibly because it inhibits the oxidation of LDL cholesterol (39). Almonds contain an unusually high amount of protein that is rich in arginine, a known precursor of nitric oxide. Nitric oxide is a potent vasodilator that is known to inhibit platelet adhesion and aggregation (40, 41). Almonds also contain dietary fiber, phytosterols, and other phytochemicals, many of which have cardioprotective effects (42–44). Although our study was not designed to test the independent effects of these bioactive components, it seems reasonable to speculate that many of these factors may act in concert to lower cholesterol or influence CVD risk factors other than blood lipids.

Dietary modification is the cornerstone of the prevention and treatment of CVD (1). However, most cholesterol-lowering diets are restrictive, because they are low in both total and saturated fat. In practice, patients are advised to eliminate or reduce the intake of certain fatty foods to meet the dietary goals of the NCEP, which is a challenge for long-term compliance. In addition, in many patients, the response to these high-carbohydrate, low-fat diets is decreased HDL cholesterol and increased triacylglycerol concentrations (6). The efficacy of cholesterol-lowering diets may be enhanced by the addition of specific whole foods that not only lower LDL cholesterol but also favorably affect other blood lipids (9–11). The frequent consumption of whole grains, legumes, and nuts was shown to reduce the incidence of CVD in several epidemiologic studies (13–15). In our study, a progressively increasing amount of almonds in the diet produced reductions in serum cholesterol in a dose-response manner. Our findings suggest that a 10% isoenergetic replacement of the habitual diet with almonds (≈34 g almonds/8368 kJ) may be sufficient to produce clinically relevant decreases in serum cholesterol. However, to produce reductions beyond those observed with the Step I diet, an isoenergetic replacement of up to 20% with almonds (≈68 g almonds/8368 kJ) may be necessary.

Our study was a controlled feeding study in which the diets fed to subjects were isoenergetic to maintain the same body weight throughout the study. Thus, one of the limitations of our study is that it does not address issues related to the consumption of almonds in free-living conditions. In addition, our study was not designed to test long-term compliance with almond consumption. However, recent research from our laboratory showed that compliance with daily almond consumption for 6 mo was very high among healthy, free-living adults (45). We further showed that incorporating an average of 54.3 g almonds/d (76 kJ/d) in the diet for 6 mo did not result in significant weight changes in these healthy adults. The effects on body weight and serum lipids of long-term consumption of almonds as an addition to the habitual diet remain to be determined.

Our investigation is in general agreement with the new NCEP Adult Treatment Panel III guidelines to liberalize the total amount of fat in the diet. The fatty acid composition of the high-almond diet matches the recommended Therapeutic Lifestyle Changes Diet for saturated fats (<7%), polyunsaturated fats (<10%), and up to 20% of total energy as monounsaturated fats (1). Moreover, results from our study support the inclusion of nuts in cholesterol-lowering diets (12).

The inclusion of almonds and perhaps other MUFA-rich nuts in usual or prescribed cholesterol-lowering diets may benefit both healthy persons and persons with hypercholesterolemia by reducing serum total and LDL-cholesterol concentrations and thus reducing the risk of CVD. Future dietary strategies for lowering serum cholesterol should focus on the consumption of acceptable and tasty whole foods rather than on the restriction of fat.

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