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# Influence of body mass index and serum lipids on the cholesterol-lowering effects of almonds in free-living individuals

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**Abstract** *Background and aims:* Short-term (4–9 weeks) human feeding trials have shown nut consumption to reduce serum total cholesterol (TC) and LDL-cholesterol (LDL). We hypothesized that individual levels of BMI, LDL, TC and triglycerides modify the cholesterol-lowering effect of almonds in a 24-week almond supplementation trial in a free-living population.
*Methods and results:* We performed secondary analysis on data from a previously published study. Using a sequential study design, all participants followed their habitual diets during the first six months (control), and then consumed an almond-supplemented diet (habitual + almonds) for another six months. 100 adults enrolled; 19 were lost to attrition. Those who completed the study were men ($n = 43$) and women ($n = 38$) with mean (SD) age 49.4 (13.6) years. During almond supplementation, we found statistically significant changes in TC (−0.22 mmol/L), LDL (−0.22 mmol/L), TC:HDL (−0.35), and LDL:HDL (−0.28) in participants with baseline LDL levels $\geq$ 3.30 mmol/L, but not among normocholesterolemic individuals. Direction and magnitude of change were similar among individuals with TC $\geq$ 5.20 mmol/L but not in the lower strata. Tests of interaction (diet $\times$ TC and diet $\times$ LDL) were significant. Reductions in the ratios TC:HDL, and LDL:HDL were significant among those with BMI $<$ 25 kg/m$^2$, but not in heavier individuals; however, formal tests of interaction did not reach significance.

*Abbreviations:* TC, total cholesterol; LDL, LDL-cholesterol; SD, standard deviation; BMI, body mass index; HDL, HDL-cholesterol; TG, triglycerides.

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*Conclusions:* We provide strong evidence that the cholesterol-lowering effect of almonds is responsive among hypercholesterolemic individuals, and weak evidence that BMI modifies the effect of almonds on serum lipids.


## Introduction

Dietary interventions to modify serum lipoproteins and improve cholesterol levels are well established and supported by the American Heart Association [1]. In the last two decades, nutrition research has placed great attention on consumption of nuts as part of a heart healthy diet in part due to its potential benefits for reducing the risk of coronary heart disease and coronary risk factors [2—4]. Most nuts are rich sources of monounsaturated fatty acids, and the essential polyunsaturated fatty acids, linoleic acid and $\alpha$-linolenic acid. They also contain bioactive components such as vegetable protein, fiber, calcium, magnesium, tocopherols, phytoesterols, phenolic compounds, arginine and other bioactive compounds that collectively contribute to their cardio-protective qualities [5].

The influence of nut consumption on cardiovascular risk factors has been addressed in a few review papers, which in general provide evidence of a cardio-protective effect. For example, observations from clinical studies suggest that nut consumption has beneficial effects on serum lipid profiles [5,6], as well as neutral or beneficial effects on biomarkers of lipid oxidation [7—9] and inflammation [10]. Additionally, findings from one meta-analysis and two systematic reviews of primarily short-term clinical trials have shown that nut consumption overall decreases total cholesterol (TC) and LDL-cholesterol (LDL) [4,6,11]. Alternatively, a meta-analysis of five 4-week randomized trials concluded that almonds have a neutral effect on serum lipid profiles [12].

The majority of the aforementioned studies used a randomized, crossover clinical trial whereby the duration of the nut intervention ranged from 3 to 9 weeks. Few studies used a sequential intervention, or a parallel design, and much less were interventions $>$12 weeks in free-living conditions. In the present study we sought to examine the effect of a 24-week almond supplementation on serum lipid concentrations of individuals in a free-living setting using data from a previously published study [13]. Specifically, we hypothesized that body mass index, and serum concentrations of LDL, TC and triglycerides at baseline would modify the cholesterol-lowering effect of almonds.

## Methods

### Study design

We performed secondary analysis using data from a non-randomized sequential intervention trial to examine the effect of a 24-week almond supplementation on serum lipids of adults. The study was conducted at Loma Linda University, Loma Linda, CA. The strategy used to enroll participants and details of the study protocol have been described previously [13]. Briefly, participants were allocated to four groups stratified by age, gender, and BMI. Participants entered the study in staggered 3-month intervals, each period representing a particular season of the year to offset any seasonal effects on diet. In each quarter, equal numbers of subjects, with similar attributes (age, sex, BMI), were enrolled. All participants followed their habitual diet during the first 6 months, and then consumed an almond-supplemented diet (habitual diet plus almonds) for an additional 6 months. Clinics were conducted at baseline (0 month), and then at 2, 4, 6, 8, 10, and 12 months, where anthropometric measures were obtained. Blood samples were collected at 4, 6, 8, and 12 months to assess serum lipids. During each diet period, 7 unannounced random order telephone 24-h dietary recalls (5 weekdays and each weekend day) were obtained, thus a total of 14 diet recalls were collected from each participant.

### Eligibility criteria

Eligible individuals were below the 95th percentile of their age-sex body mass index (BMI) distribution, did not have more than 9 kg weight change during the past six months, ate nuts less than twice a week or ate less than 7 g of nuts weekly, had no allergy or aversion to nuts, did not smoke, had no concurrent medical conditions that might affect body weight, drank no more than two glasses of alcoholic beverage per day, were not pregnant, did not consume an atypical diet or follow a rigorous exercise program.

### Almond-supplemented diet

The almond supplement was equivalent to 15% of each participant's mean energy intake during the habitual diet period. Participants had a choice of either dry roasted or raw almonds, or both. All participants were provided packaged daily portions of the almond supplement. No dietary advice was given, thus participants ate the almonds according to their individual preferences.

### Laboratory procedures

Blood was drawn from each participant after a 12-h fast into a vacutainer tube containing sodium ethylenediaminetetraacetic acid (EDTA). Serum concentrations of triglycerides (TG), total cholesterol (TC), LDL-cholesterol (LDL), and HDL-cholesterol (HDL) were assessed using standardized enzymatic procedures.

### Outcomes

Outcomes included serum total cholesterol, LDL-cholesterol, HDL-cholesterol, triglycerides, and the ratios TC:HDL and LDL:HDL.

## Statistical analysis

Univariate analyses were performed to determine the distribution of selected demographic and baseline variables. To test the effect of diet on serum lipids, analyses were conducted using repeated measures mixed models that accounted for the within-subjects variation in serum lipids and included age, gender, and change in body weight as covariates, and diet as a fixed-effect term. To assess the assumptions of the repeated measures model, univariate and sensitivity analyses were conducted on the demographic characteristics, covariates and outcome measures.

To test the possible effect modification by BMI, TC, LDL, and TG at baseline, we included a term in the model which represented their interaction with diet. Each outcome variable (TC, HDL, LDL, TG, and the ratios TC:HDL and LDL:HDL) was analyzed separately as absolute change (almond-habitual) and percent change (absolute change/habitual $\times$ 100). For each of the six outcome variables, formal tests of interaction were repeated using a different stratification variable in the interaction term. These included: 1) baseline BMI (BMI $<$ 25.0 kg/m$^2$ versus BMI $\geq$ 25.0 kg/m$^2$); 2) baseline TC (TC $<$ 5.20 mmol/L versus TC $\geq$ 5.20 mmol/L); 3) baseline LDL (LDL $<$ 3.30 mmol/L versus LDL $\geq$ 3.30 mmol/L); 4) baseline TG (TG $<$ 1.70 mmol/L versus TG $\geq$ 1.70 mmol/L). Cut points for BMI and blood lipids were defined according to The World Health Organization [14] and the Third Report of the National Cholesterol Education Program [15], respectively. All analyses were performed using SAS version 9.2 (SAS Institute Inc, Cary, North Carolina).

**Table 1** Selected Baseline Characteristics of Study Participants by Gender.

| Baseline Characteristic | Males $n$ = 43 | Females $n$ = 38 | Combined $n$ = 81 |
|---|---|---|---|
| Age (years) | % | % | % |
| 25–34 | 20.93 | 15.79 | 18.52 |
| 35–44 | 13.95 | 21.05 | 17.28 |
| 45–54 | 27.91 | 23.68 | 25.93 |
| 55–64 | 16.28 | 26.32 | 20.99 |
| 65+ | 20.93 | 13.16 | 17.28 |
| Mean Age ± Std. Dev | 49.63 ± 13.94 | 49.21 ± 13.30 | 49.43 ± 13.56 |
| Body Mass Index (kg/m$^2$)[a] | % | % | % |
| Normal, <25 | 41.86 | 63.16 | 51.85 |
| Overweight, 25–<30 | 53.49 | 21.05 | 38.27 |
| Obese, ≥30 | 4.65 | 15.79 | 9.88 |
| Mean BMI ± Std. Dev. | 25.66 ± 3.45 | 24.61 ± 4.74 | 25.17 ± 4.12 |
| LDL-Cholesterol (mmol/L)[b] | % | % | % |
| <2.60 | 31.71 | 23.68 | 27.85 |
| 2.60–3.29 | 31.71 | 44.74 | 37.97 |
| 3.30–4.09 | 24.39 | 18.42 | 21.52 |
| 4.10–4.90 | 9.76 | 13.16 | 11.39 |
| >4.90 | 2.44 | – | 1.27 |
| Mean LDL ± Std. Dev. | 3.08 ± 0.86 | 3.08 ± 0.72 | 3.08 ± 0.79 |
| HDL-Cholesterol (mmol/L)[b]* | % | % | % |
| <1.03 | 39.53 | 5.26 | 23.46 |
| 1.03–1.55 | 53.49 | 52.63 | 53.09 |
| >1.55 | 6.98 | 42.11 | 23.46 |
| Mean HDL ± Std. Dev. | 1.12 ± 0.21 | 1.53 ± 0.30 | 1.31 ± 0.33 |
| Total Cholesterol (mmol/L)[b] | % | % | % |
| <4.20 | 25.58 | 10.53 | 18.52 |
| 4.20–5.19 | 39.53 | 47.37 | 43.21 |
| 5.20–6.19 | 20.93 | 26.32 | 23.46 |
| 6.20–7.19 | 13.95 | 13.16 | 13.58 |
| >7.20 | – | 2.63 | 1.23 |
| Mean Cholesterol ± Std. Dev. | 4.93 ± 1.06 | 5.19 ± 0.93 | 5.05 ± 1.00 |
| Triglycerides (mmol/L)[b] | % | % | % |
| <1.69 | 69.77 | 84.21 | 76.54 |
| 1.69–2.25 | 11.63 | 7.89 | 9.88 |
| 2.26–5.64 | 13.95 | 7.89 | 11.11 |
| >5.65 | 4.65 | – | 2.47 |
| Mean Triglycerides ± Std. Dev. | 1.71 ± 1.34 | 1.27 ± 0.71 | 1.51 ± 1.11 |

*$p \leq 0.05$ based on chi-square.
[a] BMI classified according to the World Health Organization [14].
[b] Serum lipids classified according to the Third Report of the National Cholesterol Education Program [15].

**Table 2** Nutrient Profile, Body Weight and Body Mass Index During Habitual and Almond-Supplemented Diets.

| | Habitual Diet | Almond Diet | Difference | 95% CI of the Difference | % Difference | *p*-value |
|---|---|---|---|---|---|---|
| Energy Intake (kJ/day) | 7214.18 | 7880.59 | 666.41 | (504.31, 828.52) | 9.24 | <.0001 |
| Total Fat Intake (%energy) | 31.96 | 38.98 | 7.02 | (6.46, 7.59) | 21.98 | <.0001 |
| Saturated Fat Intake (%energy) | 10.35 | 9.71 | −0.65 | (−0.87, −0.43) | −6.26 | <.0001 |
| Monounsaturated Fat Intake (%energy) | 11.73 | 17.53 | 5.80 | (5.51, 6.10) | 49.48 | <.0001 |
| Polyunsaturated Fat Intake (%energy) | 7.43 | 9.10 | 1.67 | (1.46, 1.88) | 22.51 | <.0001 |
| Body weight (kg)$^2$ | 76.12 | 75.88 | −0.24 | (−0.55, 0.06) | −0.32 | 0.116 |
| Body Mass Index (kg/m$^2$) | 26.30 | 26.22 | −0.09 | (−0.20, 0.02) | −0.33 | 0.123 |

The design and ethical conduct of this study was approved by Loma Linda University Human Subjects Committee before the study began.

## Results

One hundred men and women were enrolled; 81 completed the study. The dropouts included 13 women (Mean BMI = 24.32 kg/m$^2$, SD = 4.05) and 6 men (Mean BMI = 27.97 kg/m$^2$, SD = 4.43) with average age 40.4 years. Participants who completed the study were men ($n$ = 43) and women ($n$ = 38) aged 25–70 years. Their mean (SD) age was 49.4 (13.6) years (Table 1). Approximately 52% had normal BMI (<25 kg/m$^2$), 38% were overweight (BMI 25 to < 30 kg/m$^2$) and 10% were obese (BMI ≥ 30 kg/m$^2$). The proportion of participants with normal levels of LDL (<3.30 mmol/L), TC (<5.20 mmol/L) and TG (<1.70 mmol/L) was 66%, 62%, and 77%, respectively, and 76% of the participants had HDL ≥ 1.03 mmol/L.

On average, almond supplementation by participants was 52 g. Compliance was 90% according to reported intake of almonds from individual 24-h dietary recalls [16]. Intake of energy, total fat, monounsaturated fat, and polyunsaturated fat significantly increased, and saturated fat intake significantly decreased during the almond-supplemented diet (Table 2). No significant change in body weight or BMI was observed between the diet periods.

We observed non-significant changes in TC, HDL, LDL, and TG, and small but statistically significant reductions in the ratios TC:HDL and LDL:HDL, after controlling for age, gender and change in body weight (Table 3).

When testing for possible effect modification, we found that during almond supplementation, estimated reductions in the ratios TC:HDL and LDL:HDL were statistically significant in individuals with BMI < 25 kg/m$^2$, but not in heavier subjects (Table 4). Results of formal interaction tests between diet and BMI, however, did not reach statistical significance. Serum concentrations of TC and LDL, and the ratios TC:HDL and LDL:HDL, in terms of absolute change (% change), significantly decreased by 0.23 mmol/L (3.9%), 0.21 mmol/L (5.5%), 0.35 mmol/L (7.3%), and 0.28 mmol/L (8.8%), respectively, in subjects with TC ≥ 5.20 mmol/L. Neither absolute nor percent change were significant among those with TC < 5.20 mmol/L. Tests of interaction between diet and TC were significant for the absolute change in these outcomes: $P$ = 0.004 for TC, $P$ = 0.04 for LDL, $P$ = 0.0009 for TC:HDL, and $P$ = 0.01 for LDL:HDL. Similarly, TC, LDL, TC:HDL and LDL:HDL significantly dropped among subjects with high LDL (≥3.30 mmol/L), by nearly the same magnitude as those with high TC, and no change among those with LDL < 3.30 mmol/L. Again, interaction tests between diet and LDL were significant for the absolute change in TC ($P$ = 0.01), LDL ($P$ = 0.05), TC:HDL ($P$ = 0.0003) and LDL:HDL ($P$ = 0.01). On stratification by baseline TG, estimated change in TG was −0.21 mmol/L among subjects with TG ≥ 1.70 mmol/L, and no significant change among those in the lower strata. Formal test of interaction between diet and TG was significant for TG ($P$ = 0.03). Individual levels of BMI and blood lipids did not modify HDL.

## Discussion

The favorable effect of tree nuts on serum lipids has been demonstrated in a number of studies with almonds [17–20], walnuts [21–25], pecans [26,27], macadamias [28,29], and pistachios [30]. In clinical trials with almonds in particular, hyperlipidemic adults consistently experienced reductions

**Table 3** Changes (Δ)[a] in Serum Lipid and Lipoprotein Levels After 24 Weeks of Almond Supplementation in Free-Living Individuals.

| | Habitual Diet | Almond Diet | Difference | 95% CI of the Difference | % Difference | *p*-value |
|---|---|---|---|---|---|---|
| Total Cholesterol (mmol/L)[b] | 5.13 | 5.05 | −0.08 | (−0.17, 0.02) | −1.48 | 0.124 |
| HDL-Cholesterol (mmol/L)[b] | 1.30 | 1.32 | 0.02 | (−0.01, 0.04) | 1.38 | 0.171 |
| LDL-Cholesterol (mmol/L)[b] | 3.13 | 3.04 | −0.09 | (−0.19, 0.01) | −2.94 | 0.074 |
| Total Cholesterol:HDL | 4.17 | 4.01 | −0.16 | (−0.26, −0.06) | −3.78 | 0.003 |
| LDL:HDL | 2.56 | 2.42 | −0.14 | (−0.24, −0.04) | −5.53 | 0.006 |
| Triglycerides (mmol/L)[c] | 1.48 | 1.46 | −0.02 | (−0.13, 0.10) | −1.03 | 0.794 |

[a] Mean change (almond–habitual) in the outcome measures were determined using age-, gender-, and weight- adjusted repeated measures multivariable regression analysis.
[b] To convert to mg/dL, multiply by 38.67.
[c] To convert to mg/dL, multiply by 88.57.

**Table 4** Absolute Changes (Δ)[a] in Blood Lipid and Lipoprotein Concentration According to Baseline Body Mass Index and Blood Lipids[b] after 24 Weeks of Almond Supplementation in Free-Living Individuals.

| | Total Cholesterol[c] (mmol/L) | HDL-Cholesterol[c] (mmol/L) | LDL-Cholesterol[c] (mmol/L) | Triglycerides[d] (mmol/L) | Total Cholesterol:HDL | LDL:HDL |
|---|---|---|---|---|---|---|
| Body Mass Index (kg/m$^2$) | | | | | | |
| <25.0 | −0.11 (−0.25, 0.02) | 0.02 (−0.02, 0.05) | −0.13 (−0.27, 0.01) | 0.03 (−0.13, 0.19) | −0.17 (−0.32, −0.03) | −0.17 (−0.31, −0.03) |
| ≥25.0 | −0.04 (−0.18, 0.10) | 0.02 (−0.02, 0.06) | −0.05 (−0.19, 0.10) | −0.06 (−0.23, 0.10) | −0.14 (−0.29, 0.01) | −0.11 (−0.25, 0.04) |
| Total Cholesterol (mmol/L) | | | | | | |
| <5.20 | 0.05 (−0.08, 0.18) | 0.00 (−0.03, 0.03) | 0.01 (−0.13, 0.14) | 0.08 (−0.08, 0.23) | 0.00 (−0.14, 0.13) | −0.03 (−0.16, 0.11) |
| ≥5.20 | −0.23 (−0.37, −0.09)[e] | 0.04 (0.00, 0.08) | −0.21 (−0.36, −0.06)[e] | −0.13 (−0.30, 0.04) | −0.35 (−0.50, −0.20)[f] | −0.28 (−0.43, −0.13)[e] |
| LDL-Cholesterol (mmol/L) | | | | | | |
| <3.30 | 0.03 (−0.10, 0.15) | 0.00 (−0.04, 0.03) | −0.01 (−0.14, 0.12) | 0.10 (−0.04, 0.24) | 0.01 (−0.13, 0.14) | −0.03 (−0.16, 0.10) |
| ≥3.30 | −0.22 (−0.37, −0.07)[g] | 0.04 (0.00, 0.08) | −0.22 (−0.37, −0.06)[g] | −0.13 (−0.30, 0.03)[g] | −0.37 (−0.52, −0.21)[h] | −0.31 (−0.47, −0.15)[g] |
| Triglycerides (mmol/L) | | | | | | |
| <1.70 | −0.04 (−0.16, 0.07) | 0.01 (−0.02, 0.04) | −0.08 (−0.20, 0.04) | 0.06 (−0.07, 0.20) | −0.11 (−0.24, 0.01) | −0.13 (−0.25, −0.02) |
| ≥1.70 | −0.16 (−0.34, 0.02) | 0.04 (−0.01, 0.09) | −0.12 (−0.32, 0.07) | −0.21 (−0.43, 0.00)[i] | −0.27 (−0.46, −0.08) | −0.17 (−0.36, 0.03) |

Data are reported as means (95% confidence intervals).

[a] Mean change (almond diet - habitual diet) in the outcome measures were determined using age-, gender-, and Δweight-adjusted repeated measures multivariable regression analysis.

[b] BMI cutoffs defined according to the World Health Organization [14], and serum lipid and lipoprotein cutoffs according to the Third Report of the National Cholesterol Education Program [15].

[c] To convert to mg/dL, multiply by 38.67.

[d] To convert to mg/dL, multiply by 88.57.

[e] $P < 0.05$.

[f] $P < 0.001$ for diet × total cholesterol interaction.

[g] $P < 0.05$.

[h] $P < 0.001$ for diet × LDL-cholesterol interaction.

[i] $P < 0.05$ for diet × triglycerides interaction.

in LDL; however, the effect on total cholesterol, HDL and LDL:HDL varied. For example, TC and LDL significantly dropped while HDL was preserved in two randomized crossover parallel studies in free-living adults [20,31]. The intervention in these two studies was 4 weeks and the almond supplement was either 50 g or 100 g. In two randomized crossover studies also in hyperlipidemic and normolipidemic participants, significant reductions in TC, LDL, LDL:HDL, and a significant elevation in HDL were observed following the experimental diets which contained the highest amount of almonds compared to control diets in both the 4-week controlled feeding trial [19], and the 1-month intervention in a free-living setting [32]. In feeding trials of 6—9 weeks duration among normolipidemic individuals, investigators reported significant reductions in TC and LDL [17,33], and in one of these, there was a significant increase in HDL [33]. Clearly, these human feeding trials provide some evidence of the beneficial effect of almond consumption on serum levels of TC and LDL in the short term.

Our results suggest that 24 weeks of almond supplementation can induce significant improvements on TC:HDL and LDL:HDL. On examination of the interaction between diet and specific factors, however, we found that the responsiveness to long-term almond supplementation varied according to individual levels of total cholesterol, LDL and triglycerides. That we observed a reduction in TG among subjects with hypertriglyceridemia, and improvements in TC, LDL, TC:HDL, and LDL:HDL among those with high baseline LDL ($\geq$3.30 mmol/L), but not in individuals with lower LDL, agree with the findings of a recent pooled analyses of 25 nut intervention trials [34].

In a meta-analysis of five randomized, 4-week intervention trials, investigators reported a trend toward a 0.05 mmol/L reduction in HDL associated with almond consumption; however, no consideration was given to baseline BMI or lipid levels [12]. In the present study, the change in HDL tended to be in a positive direction with a magnitude of 0.4 mmol/L among hypercholesterolemic individuals, however, this did not reach significance. These as well as our findings of improved TC, LDL, TC:HDL, and LDL:HDL among hypercholesterolemic subjects further confirm what Keys and Anderson [35] claimed long ago: "Men who are intrinsically hypercholeremic exhibit greater cholesterol response to dietary changes, while hypocholesteremic men are less responsive, than reference men."

There was a tendency in our study for those with normal BMI to improve the ratios TC:HDL and LDL:HDL; however, our non-significant results on the interaction between BMI and diet do not corroborate the findings of a pooled analysis [34], which found BMI as a significant effect modifier of the cholesterol-lowering property of nuts.

We reported previously an overall improvement in the nutrient profile [16], and a small but non-significant weight gain of 0.4 kg [13] in our participants during the almond-supplemented diet. This report extends our findings to include blood lipids. The majority of studies on nut consumption and serum lipids are randomized, crossover trials testing 2 or more dietary interventions and a background diet under metabolic or *ad libitum* dietary conditions, with each dietary period lasting 4—8 weeks. Although the metabolic study approach is preferred, it has limited generalizability, as by design such a study requires rigorous dietary intervention, which not only introduces nuts into the diet as the primary test variable, but also imposes a prescribed background diet that departs from the participant's customary eating patterns. The value of the present study is the longer-term duration (24 weeks) of the nut intervention under free-living conditions. Using the habitual diet as control and providing no dietary advice also were important features of the study design as our goal was to duplicate the setting of a public health advice, or consumer advertisement, to consume more nuts than individuals would customarily eat. This approach provided an opportunity to observe changes in lipids in the context of any natural dietary modification or changes in body weight that may have occurred as a consequence of adding a single test variable, in our case, almonds.

The limitations of this investigation relate primarily to the study design. We controlled for seasonal trends in body weight; however, we could not control for training effect and secular trends in the economy and the environment, which may have affected dietary behaviors, and subsequently serum lipids. All participants started with the habitual diet, and then crossed over to the almond diet. The lack of randomization in the diet order may have resulted in biased estimates in the observed differences in serum lipids.

We demonstrated in this study that daily consumption of 52 g (1.8 oz) of almonds for 24 weeks without dietary advice can improve TC:HDL and LDL:HDL ratios. Changes in total cholesterol, LDL, and the ratios TC:HDL and LDL:HDL induced by nut supplementation may be more responsive in persons with hypercholesterolemia. We provide strong evidence that the cholesterol-lowering effect of almonds is modifiable by individual cholesterol levels; weak and no evidence that BMI and TG, respectively, modify the effect of almond supplementation on serum lipids. Findings from this study may provide evidence-based support for the Qualified Health Claim on nuts and coronary heart disease issued by the U.S. Food and Drug Administration in 2003 [36].

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The Almond Board of California had no role in the study design; the collection, the analysis and interpretation of data; the writing of the report; and the decision to submit the paper for publication.

## Conflict of interest

A grant from the Almond Board of California provided support for this work. Joan Sabaté has served on the scientific advisory boards for the California Walnut Commission and Paramount Farms. All other authors report no conflict of interest.

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