

Original Research

Effect on Body Weight of a Free 76 Kilojoule (320 Calorie) Daily Supplement of Almonds for Six Months

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Objective: Regular nut consumption is associated with lower rates of heart attack. However, as nuts are fatty foods, they may in theory lead to weight gain, although preliminary evidence has suggested otherwise. We tested the hypothesis that a free daily supplement (averaging 76 kJ) of almonds for six months, with no dietary advice, would not change body weight.

Methods: Eighty-one male and female subjects completed the randomized cross-over study. During two sequential six-month periods, diet, body weight and habitual exercise were evaluated repeatedly in each subject. Almonds were provided only during the second period. The design was balanced for seasonal and other calendar trends.

Results: During the almond feeding period, average body weight increased only 0.40 (kg) ($p \sim 0.09$). The weight change depended on baseline BMI ($p = 0.05$), and only those initially in the lower BMI tertiles experienced small and mainly unimportant weight gains with the almonds. We estimated that 54% (recalls) or 78% (diaries) of the extra energy from almonds was displaced by reductions in other foods. The ratio unsaturated/saturated dietary fat increased by 40% to 50% when almonds were included in the diet.

Conclusion: Incorporating a modest quantity (76 kJ) of almonds in the diet each day for six months did not lead on average to statistically or biologically significant changes in body weight and did increase the consumption of unsaturated fats. Further studies are necessary to evaluate longer term effects, especially in men.

INTRODUCTION

Dietary fat has a bad reputation among the public and health professions alike. Although this is partly deserved, a wide variety of evidence suggests that there is benefit in being more discriminating in this evaluation. Specifically, it has long been understood that although saturated fats raise blood cholesterol, unsaturated fats, by comparison, lower blood LDL cholesterol, and monounsaturates accomplish this with little or no reduction in HDL cholesterol [1]. However, comparative effects on body weight are unclear.

More recently, there have been a number of publications describing associations between the frequent consumption of small quantities of nuts, foods rich in unsaturated fats, and lower rates of coronary heart disease events. Several large

epidemiologic studies that recorded data on nut consumption consistently suggest such an effect [2].

A number of the nut feeding trials did not impose constraints on body weight, yet noted no significant changes in weight during the nut feeding, despite this being a fatty food [3–6]. As discussed more fully below, it is quite controversial whether fat as compared to other sources of calories is more likely to cause weight gain [7–11]. Nevertheless, a continuing concern regarding a recommendation to increase consumption of a fatty food, such as nuts, is that this may lead to weight gain in the population at large.

The purpose of this report is to describe a randomized cross-over experiment to test the null hypothesis that consuming approximately two ounces (76kJ) of almonds each day over a period of six months does not change body weight. The goal

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was to duplicate the setting of subjects responding to public health advice, or perhaps advertisement, by choosing to eat more nuts.

MATERIALS AND METHODS

Subjects for this study were between the ages of 25 and 70 years. They were respondents to radio, newspaper and notice board advertisements. After screening, we enrolled equal numbers of both men and women and equal numbers in the three age ranges 25–39, 40–54, and 55–70 years. As the main endpoint of this part of the study was weight change, we excluded the relatively atypical subjects who were higher than the 95th percentile of their age-gender body mass index distribution. Enrollment to the 1–50th, 50–85th, and 85–95th percentile groups was stratified proportionate to expectation. Other exclusion factors were cigarette smoking, drinking more than two glasses of alcoholic beverage per day, more than 9 kg weight change in the last six months, a very heavy exercise program, concurrent medical conditions that might affect body weight, pregnancy, a very atypical diet, allergy or aversion to nuts, and already eating nuts more than twice weekly (this would diminish the contrast between usual diet and nut supplementation).

As there may be seasonal trends in body weight, subjects were randomly allocated to four groups that were enrolled to the study at three-month intervals. The randomization to study group was stratified by age, gender and body mass index. This strategy ensured that in each quarter, similar subjects were assigned to intervention and non-intervention status and in equal numbers. Thus, any calendar time-related effects cancel when contrasting mean results from intervention and non-intervention periods.

Within each group there was no formal intervention during the first six months. However, data was collected, and this included seven telephone 24-hour recalls evenly spread throughout the period. These were unannounced, and were scheduled to include five weekdays and each weekend day. The Nutrition Data System interactive software [12] was used by the interviewing nutritionists who were registered dietitians or senior Masters students in nutrition. All had been trained in the use of this software. The 24-hour recalls were tape-recorded, later reviewed, and if necessary corrected by a second nutritionist.

A second means of dietary assessment was the collection of two one-day diet diaries, one on a Sunday and one on a Wednesday. The diary also included questions about subjective satiety at each meal or snack during the day (these results are not included in this report). Subjects attended clinic on four occasions throughout the control period when height, weight and hip circumference were measured. Blood was drawn at the third and fourth clinics in order to measure the fatty acid content of erythrocyte membranes. This was used as a baseline

to check compliance with the study requirement to consume almonds during the second six months of the study. For measurement of fatty acids, lipids were extracted from plasma using chloroform: methanol [13]. Individual lipid classes were separated by preparative thin-layer chromatography [14]. Fatty acid methyl esters were separated and quantified by capillary gas chromatography (Hewlett-Packard model 6890, Wilmington, DE) [14].

Weight while fasting was measured with Scale-Tronix electronic scales that were calibrated at the beginning of each early morning clinic and read to the nearest 0.05 kg. Waist and hip measurements were all conducted by four trained technical staff, according to a written protocol. Subjects stripped down to their underclothes. Waist measurements were taken at a level one inch above the navel. Hip measurements were made at the widest part between the waist and knees. All measurements were the average of three on each occasion.

An exercise questionnaire [15] was completed at each clinic visit. We measured the frequency of vigorous exercise by the question, "How many times per week do you engage in vigorous activities such as brisk walking, jogging, bicycling, etc., long enough or with enough intensity to work up a sweat, get your heart thumping or get out of breath?" A choice was made between eight different frequencies ranging between "never" to "six or more times per week."

Subjects also attended an early morning clinic where resting, fasting energy expenditure was obtained using the Sensor-medics 4400 metabolic unit [16]. This was to evaluate whether differences in metabolic rate during different periods of the study could help us understand any mismatch between energy intake, energy expenditure and change in body weight. Unfortunately a failure in the original equipment invalidated the data for groups 1 and 2. Hence we report results for groups 3 and 4 only, which were obtained using new equipment.

The formal intervention occupied the second six months of the study for each group. The intervention followed control periods for all subjects, as this would be the order of diets in the situation being modeled, i.e., incorporation of nuts in the diet as a response to public health advice or advertisements. During the intervention period, observations to collect data were identical to those described above for the first period, except that there were only three clinic visits, also at two month intervals. Blood was drawn at the first and third clinics (months 8 and 12 of the study). Fasting energy expenditure was again measured.

The intervention consisted of eating a small daily allowance of almonds that, based on each subject's first series of dietary recalls, was calculated to account for about 15% of daily energy for each individual. On average, this supplement was 54.3 grams or 76.4 kJ (319.8 calories) per day (about 40–50 nuts), and the range was 59.9 to 99.9 kJ per day. Subjects could choose either raw or dry-roasted nuts.

It is important to note that no dietary advice was given, and subjects were able to incorporate the nuts to their diet as they saw fit. This could be at one time or on several occasions

throughout the day. If subjects enquired further, only general suggestions were made that almonds could be added to cereals, salads, desserts, or used as snacks. No recipes were provided. Subjects were free to compensate for the nuts by changing consumption of other foods if they saw fit. Subjects were aware that we were checking the impact of eating the nuts on biochemical, physiologic and morphologic measures, but were unaware of the specific nature of any such hypotheses or of any particular interest in the effect on body weight.

The nuts were provided free of charge in daily packets, labeled with a particular day of the week and the weight of almonds in ounces. They were distributed at clinic visits as a two month supply, with sufficient extra packages to allow for any modest delay in attending the next clinic. There was also an excess designated for other family members in order to minimize the likelihood of their eating the nuts assigned for the study subject.

We made a considerable effort to establish a relationship of trust with the participants, by frequent telephone contact, conversation at clinic visits and recognition of special occasions. In particular, it was emphasized that if the almonds were not eaten, either by oversight or other circumstances, it was important that the investigators know and that there were no negative consequences. A requirement for inclusion in the study was that subjects must have no aversion to eating almonds.

Paired *t*-tests were used to compare results from intervention and control periods. Linear regression was used to test the effects of the intervention while adjusting for other factors. These analyses were carried out using the SAS [17] and S-PLUS [18] computer packages. The study had 80% power at $\alpha = 0.05$ to detect a difference in body weight between study periods of 0.65 kg. The design and ethical conduct of this study was reviewed and approved by Loma Linda University Human Subjects Committee before the study began.

RESULTS

One hundred subjects were initially enrolled, and 81 completed the one-year study. The 19 who dropped out did so for

the following reasons, only 5 of which could be related to the intervention: 4—inconvenience of attending the clinic for tests; 3—severe difficulty in contacting the subject to obtain the repeated telephone 24-hour recalls; 3—women became pregnant; 2—moved out of the area; 1—was prescribed a weight loss program by his physician; 1—died of unrelated causes; 4—possible gastrointestinal reactions to almonds; 1—developed an aversion to almonds. These dropouts included 13 women and 6 men. Their average age was 40.4 years, a little younger than the average subject. Their average BMI's were 24.32 kg/m² (SD = 4.05) in women and 27.97 kg/m² (SD = 4.43) in men.

Selected baseline characteristics of the subjects who completed the study are shown in Table 1. The average age is just below 50 years for men and women, and values of BMI and waist/hip ratio are close to community norms. Mean values for all variables were relatively similar between groups, the differences being easily compatible with random variation.

On average, there was a non-significant weight gain of less than a pound (0.40 kg) when comparing the average weight during the six months of almond feeding with the control period (Tables 2 and 3). The men gained a biologically insignificant 0.65 kg ($p < 0.01$) and the women 0.11 kg ($p = 0.79$). In both genders (Table 3), it was only those with initially lower or medium tertile BMI values that gained any weight, and women with initially higher BMI values actually lost weight. An inverse association between baseline BMI and change in weight was statistically significant in men, also when both genders were combined. However, all changes were small and probably not of biological significance. The distribution and range of weight changes is indicated by the following numbers of subjects who were in the given ranges of weight change; 3—greater than 2.0 kg weight loss, 8—1.01 to 2.0 kg weight loss; 20—0.01 to 1.0 kg weight loss, 20—0 to 1.0 kg weight gain, 18—1.01 to 2.0 kg weight gain, 8—2.01 to 3.0 kg weight gain, 4—greater than 3.0 kg weight gain. The maximum weight gain was 4.21 kg.

Similarly, the mean waist/hip ratios measured at each clinic during the study are shown in Table 2. Again, it was only in

Table 1. Baseline Characteristics of Subjects Completing the Study

	Group				All
	1	2	3	4	
Males					
Number of Subjects	11	11	11	10	43
Age (Years)*	49.8 (14.5)	47.7 (12.4)	52.0 (13.4)	47.0 (15.4)	49.2 (13.6)
BMI (kg/m ²)*	27.1 (2.80)	25.4 (3.47)	26.8 (2.44)	27.5 (5.16)	26.7 (3.56)
W/H ratio*	0.97 (0.06)	0.94 (0.19)	0.96 (0.03)	0.93 (0.09)	0.94 (0.07)
Females					
Number of Subjects	10	9	8	11	38
Age (Years)*	48.9 (13.4)	51.8 (11.7)	51.0 (15.3)	48.5 (14.9)	49.9 (13.4)
BMI (kg/m ²)*	25.9 (5.79)	28.1 (6.50)	25.3 (6.76)	24.8 (3.16)	25.9 (5.54)
W/H ratio*	0.78 (0.06)	0.79 (0.08)	0.79 (0.09)	0.79 (0.07)	0.78 (0.06)

* Standard error in parentheses.

Table 2. Mean Body Weight and Waist/Hip Measurement* by Period of the Study

Follow-up Month§	0	Control			Mean	Almonds			Mean	p†
		2	4	6		8	10	12		
Body Weight (kg)										
Males	83.2	83.5	82.8	83.3	83.18	83.5	83.9	84.0	83.83	<0.01
Females	69.3	69.2	69.0	69.1	69.13	69.1	69.2	69.4	69.24	0.79
Waist/Hip Ratio										
Males	0.94	0.95	0.94	0.95	0.943	0.95	0.96	0.95	0.955	<0.01
Females	0.78	0.78	0.77	0.79	0.779	0.78	0.79	0.78	0.784	0.18

* Mean of two at each measurement.

† Paired *t* test compares the means of the two periods.

§ Each of the four admission groups had an assessment at baseline and on three subsequent occasions during each of the control and almond periods. Thus data for a particular follow-up month refers to different dates for each group.

Table 3. Change in Body Weight (kg) Comparing Six Month Periods Without and With Almond Supplementation: Dependence on Baseline BMI

Subgroup	Tertile of Baseline BMI§	Without Almonds	With Almonds	Difference	p value
All		76.6	77.0	0.40	0.09†
	Low	64.3	65.1	0.82	
	Medium	74.8	75.5	0.61	0.05*
	High	90.1	89.8	-0.35	
Men		83.2	83.8	0.65	<0.01
	Low	72.3	73.8	1.44	
	Medium	84.2	84.7	0.55	<0.005*
	High	92.9	92.7	-0.05	
Women		69.1	69.2	0.11	0.79
	Low	56.3	56.5	0.19	
	Medium	65.5	66.2	0.77	0.49*
	High	87.0	86.4	-0.57	

* Test of linear trend in body weight change according to level of baseline BMI.

† 95 percent confidence interval (-0.06 to 0.86 kg).

§ Tertiles are for males ≤25.51, 25.52–28.22, ≥28.23; for females ≤22.74, 22.75–27.66, ≥27.67.

males that there was any indication of change, and this was slight, but statistically significant. Further analysis showed that when dividing the males to tertiles of baseline BMI, those with highest BMI had an increase of 0.010 in waist/hip ratio, and those with lowest baseline BMI had an increase of 0.015 in waist/hip ratio. In all baseline BMI tertiles in women the change in waist/hip ratio was less than 0.005. In both genders these changes are probably biologically insignificant.

During the almond feeding period, each subject was provided an average of 13,743 extra kJ (or 57,500 extra calories) from the almonds. If this energy was simply added to the diet and stored, then the predicted weight gain is 6.40 kg, whereas in fact there was no significant weight gain. We considered various possible explanations, and collected data to address the questions listed below.

- a) Did the subjects eat the almonds?
- b) Was there a reduction in energy eaten from other foods, that is displacement of other foods?
- c) Was the metabolic rate increased during the almond-feeding period?

d) Did the subjects increase energy expenditure by more exercise during the almond feeding period?

We have evidence that the almonds were indeed eaten. First, subjects openly discussed with us their occasional omissions. Second, of the 567 24-hour recalls that were conducted during the almond feeding period, the almond supplement was reported as eaten in 90.2% of these recalls. Of the 162 single day food diaries completed by the subjects without assistance or prompting, 89.2% included the almonds during the almond-feeding period. There were also days where double-almond intake was reported, an apparent make-up for an omission the day before. Third, as almonds are rich in oleic acid, we measured the oleic acid content of red blood cell membranes in 30 randomly selected study subjects. The oleic acid content of red blood cells reflects oleic acid metabolism over several months, although intrinsic production rather than dietary consumption keeps levels relatively constant. The two control period blood samples were mixed, as were those from the almond feeding period. During the control period, the oleic acid as a portion of total membrane fat was 11.53%, and this rose to 12.08% during

almond feeding. Although the magnitude of this change is relatively small, it is a highly significant difference ($p < 0.0002$) indicating that a real change had occurred. The displacement of calories from other foods (see below) is also evidence for an important change in dietary habits.

We have evidence that a portion of the energy from almonds was compensated for by displacement of energy previously consumed from other foods. Let i be a nutrient; C_i = energy from that nutrient eaten in the control phase; A_i = energy from that nutrient eaten in the almond feeding phase; S_i = energy from that nutrient contained in the almond supplement; $T_i = C_i + S_i$. Then displacement of nutrient i is given by $D_i = T_i - A_i$.

Estimates of percent displacement ($100 \times D_i/S_i$) using 24 hour recall data (Table 4) suggest that 53.6% of total energy from the almonds and 75% of that from the saturated fat was displaced, but that there was displacement of only 15.5% of energy from total fat and a small proportion of that from unsaturated fats. Although the percentage displacement of carbohydrate was very high (260%), in absolute terms this made only a modest contribution to displaced energy, as there was little carbohydrate in the almond supplement.

Displacement data from the diaries were often unstable with large standard errors, as there were only two days of data for each feeding period. Nevertheless, the estimated displacement (SE) for total energy was 78.2% (35.7), for carbohydrate 328.6% (136.0) and saturated fat 205.9% (92.6), again suggesting that much of the total energy and possibly all of carbohydrate and saturated fats were displaced. Both the recalls and diaries showed that the ratio of unsaturated/saturated fats was higher (by 42% in the recalls or 51% in the diaries), when eating the diet that incorporated almonds.

Table 5 shows estimates of resting, fasting energy expenditure. As can be seen, in the 41 subjects for which estimates were available, there was no evidence of any difference between the control and almond phases of the study. Indeed, estimated resting energy expenditure was a little less during the almond feeding period. We can be 95% certain that we did not miss an excess of energy expenditure of 33.6 kcal/day or more during the almond period. This is based, however, only on early morning fasting data, extrapolated to the whole day.

The mean frequency of vigorous physical activity sessions did not change during the study, as shown in Table 6. Men had slightly fewer such sessions during the almond feeding phase and women slightly more, but these differences are compatible with random variations in their habits. A similar question to evaluate activities of moderate intensity also showed no significant differences between the two feeding periods. If anything, there was a little less moderate intensity activity during the almond feeding phase for both men and women.

DISCUSSION

Our original motivation for this study was the observation that weight gain had never been a problem in several nut feeding studies using human subjects [3–6], despite very limited dietary advice. Our more formal investigation of this question finds that after six months of a free daily supplement of almonds, a food rich in monounsaturated fatty acids, average weight gain was only 0.40 kg, this being compatible with a chance fluctuation. There was also no significant overall change in waist/hip ratio. On average the almond supplement

Table 4. Displacement (D) of Nutrients During the Almond Period (kilojoules): Means of Seven 24-hour Recalls or Two One-Day Diaries for Each Feeding Period

	Dietary Method	Control Phase (C)	Almond Phase (A)	p^\dagger	Almond Supplement (S)*	$D = S + C - A$	%D = $100 \times D/S$ (SE)
Energy	Recalls§	486.8	522.0	0.0005	75.3	40.1	53.6 (17.5)
	Diaries§	475.8	516.2	0.02	73.1		
Protein	Recalls	71.3	76.7	0.001	9.6	4.2	39.8 (22.9)
	Diaries	69.8	74.8	0.12	9.3		
Fat	Recalls	157.6	204.0	0.0001	61.9	15.5	24.5 (9.3)
	Diaries	158.5	207.1	0.0001	60.0		
CHO	Recalls	265.9	252.7	0.02	9.2	22.5	259.8 (78.0)
	Diaries	255.2	246.2	0.25	9.0		
SFA	Recalls	51.8	53.1	0.43	5.8	4.5	75.0 (37.0)
	Diaries	53.3	53.1	0.96	5.6		
MFA	Recalls	60.3	94.0	0.0001	40.2	6.4	16.4 (6.1)
	Diaries	59.8	96.1	0.0001	39.0		
PFA	Recalls	33.7	43.1	0.0001	13.0	3.6	25.1 (9.8)
	Diaries	33.5	43.7	0.0001	12.6		

* Almond supplement column is the net difference in almond intake between the two study periods, and takes into account the occasional small quantities of almonds eaten in addition to the supplied supplement during both periods.

† t test comparing control and almond dietary phases.

§ 24-hour recall data are means of seven days, each day from a different week.

Diaries are means of two days, each day (a Wednesday or Sunday) from a different week.

Table 5. Metabolic Rate Measures at Rest and Fasting: Control and Almond Periods (N = 41 Subjects*)

	Control	Almonds	<i>p</i>	Least Detectable Differences (Power = 0.8, α = 0.05)
VO ₂ (mL)	183.6	178.8	0.29	12.7
VCO ₂ (mL)	161.5	156.1	0.40	17.6
RQ	0.88	0.87	0.60	0.05
Estimated Resting Energy Expenditure (kJ/day)	308.8	301.4	0.35†	22.1

* Subjects: 10 from Admission Group 2; 15 from Admission Group 3; 16 from Admission Group 4.

† 95% confidence interval of almond minus control data (-22.8, +8.0).

Table 6. Mean Weekly Frequency of Vigorous Physical Activity Sessions Reported at Clinic Visits During Each Study Period

Follow-up Month†	0	Control			Mean	Almonds			Mean	<i>p</i> *
		2	4	6		8	10	12		
Males	3.14	3.23	2.99	2.74	3.03	2.77	2.94	2.86	2.86	0.34
Females	2.91	2.83	3.35	3.05	3.04	3.57	3.41	3.26	3.41	0.07
Total	3.03	3.04	3.16	2.89	3.03	3.15	3.17	3.05	3.12	0.53

* Paired *t* test compares the means of the two periods.

† See footnote to Table 2.

was 76 kJ (320 calories) per day. When considering the genders separately, there was evidence of a minor weight gain in men, but not in women. However, gender-specific changes were not considered a priori when the study was designed. Moreover, it was only those in the lowest two tertiles of body mass index who gave any indication of weight gain. The more obese subjects of both genders actually lost small amounts of weight while taking the supplement.

This may seem a surprising result as it is generally believed that high fat diets promote weight gain. However, there is little published evidence that changes in the intake of a fatty food in non-obese free-living subjects will change their body weight. We note that although on average this study population was borderline overweight, this was by design, reflecting the U.S. population. However, those above age-gender specific-95th percentiles of body weight were excluded. Whether these results would apply to a study population of ideal weight is unknown.

A traditional study design may have included a control group that had no dietary intervention during the whole 12 months of the study. However, this would have eliminated the possibility of paired testing and importantly reduced study power. Further, the combined control periods of our study groups provided an appropriate six-month non-intervention control using the same subjects, covering all seasons of the year and equivalent to the intervention period except for a six-month shift in time. We do not know of any reason that a small secular difference would produce differences in the forces determining body weight, but the reader should be aware of this design feature. There is also no evidence that our dietary monitoring during the control phase promoted changes in body weight or waist/hip ratios during the six-month control periods. Repeated

assessments during the control phase (Table 2) give no suggestion of trends.

Did the subjects in our study eat the almonds? We selected subjects who stated that they enjoyed eating almonds and encouraged them to discuss with us any problems with compliance. Two different methods of dietary assessment, used on different days, were remarkably consistent in suggesting that less than 10% of the nuts were not eaten. The estimated displacement of 40% to 50% of the extra energy from almonds is also strong evidence of compliance to at least this extent, as it is unlikely that reported intake of energy from other sources would spontaneously decline.

The dietary intake of oleic acid changed from approximately 12.5% to 18% of energy during the almond supplement period. The increase of the erythrocyte membrane oleic acid content from 11.53% to 12.08% of all fats (<0.0002) during this period is consistent with good compliance and is a result that is similar to the findings of others when the oleic acid content of the diet changes moderately [19–22]. The fatty acid content of erythrocytes was chosen in view of their relatively long half-life. The small change in percent of erythrocyte membrane oleic acid was expected, as most oleic acid is manufactured endogenously and dietary oleate has a much smaller influence [23]. However, oleic acid seemed the most obvious biomarker of almond consumption given the present knowledge of almond chemistry. Thus, there is evidence that a high percentage of the almonds were eaten.

We estimated that 54% (recalls) or 78% (diaries) of the additional energy from almonds was displaced by a reduction of intake in other foods. However, this was an uneven displacement across nutrients resulting in a higher intake of both mono- and poly-unsaturated fats during the almond period and little

change in other macro-nutrients. There is very little known about the effect of a free fatty supplement on intake of other foods, and it is likely that the results may depend on the specific nutrient content of the supplement.

If subjects tended to report their usual diet rather than what they actually ate, our dietary assessment methods may have detected only part of a larger actual displacement. However, there is reasonable concordance between the two dietary methods, suggesting that much of the energy from almonds was displaced. Although fatty foods are often considered palatable but not satiating [24], almonds may be both palatable and satiating due to non-fatty components such as protein and dietary fiber. If satiating, this would lead to a reduction in intake of other foods, as we observed.

There was no evidence of any change in energy expenditure due to physical activity. We did not find any change in resting, fasting, early morning energy expenditure. There was also no obvious change in respiratory quotient that may have indicated a change in the source of energy being burned. However, if metabolic energy expenditure did increase, this may have largely followed meals, or the eating of almonds, which would not have been detected by our fasting tests.

It is probable that the absorption of energy from the nut fat was incomplete. Indeed there is data to support this conjecture for the consumption of pecans [25], almonds (Sabaté J, unpublished observations) and peanuts [26]. In that case, the combination of the observed displacement of energy from other foods plus incomplete absorption could largely explain the lack of weight gain.

Although it is generally held that a higher percentage of energy from fat in the diet promotes weight gain, the evidence for this is by no means secure. Ecologic evidence does not clearly support this conclusion. For instance, although the fat content of the U.S. diet has decreased modestly over recent decades, the increase in obesity continues unabated [7].

Cross-sectional epidemiologic studies have often reported a positive association between percent of energy from fat and body mass index [7, 8, 27]. The evaluation of this evidence must be tempered by the difficulty of establishing the temporal direction of any underlying causal connection. Prospectively designed studies provide stronger evidence in theory. However, the results from these studies are quite inconsistent [7, 8, 27–29].

Perhaps the strongest study design is the randomized trial. Trials have generally been either short-term controlled feeding trials of small numbers of subjects or long-term trials generally with less dietary control. Many of these trials used obese subjects, though some included persons of normal weight, often for reasons other than the evaluation of changes in their body weight.

Generally, trials have found that substantial reductions in fat content of the diet will lead to loss of body weight [7, 9]. The energy reduction associated with the reduced fat is usually not fully compensated by increased consumption of other nutrients [30–32]. Thus it is the reduced energy rather than specifically

reduced fat that is responsible for any weight loss in these studies. However, the effect of a fat-reduction strategy on body weight is usually small (1–2 kg), and often partially disappears over time [7, 33–35]. When energy is carefully controlled, or did not differ between diets, a reduction in either fat or carbohydrate content produces similar results on body weight [10, 11, 36–40]. This appears to be also true in diabetics [41].

There is incomplete, but intriguing evidence that just as all dietary fats are not equal in their effects on blood lipids, this may also be so for effects on body weight. At least two feeding studies of rats [42, 43] have shown that, as compared to dietary saturated fat, polyunsaturated fats led to much less weight gain and increased oxygen consumption. Another report found that, in obese human subjects only, whole-body postprandial burning or oxidation of dietary fat was greater when the dietary polyunsaturated to saturated ratio was higher [44].

CONCLUSIONS

This study documents a surprising absence of significant weight gain on average, when a free 76 kJ (320 calorie) supplement of almonds was supplied daily for a period of six months. A lack of weight gain during this period was especially evident in more obese subjects and such women as actually lost small amounts of weight. The explanation appears to be that the additional energy was largely displaced by reduced intake of other foods. Indeed it is possible that all of the extra absorbed energy was displaced, given the probable modest malabsorption of fat from almonds. Interestingly, very little of the additional oleic or linoleic acids from the almonds were displaced. Hence the fatty acid composition of the total diet changed favorably during the six month almond supplement period. Although there was no overall significant weight gain, men did gain 0.65 kg on average while on almonds for six months. If this were to continue, it would clearly become biologically significant, but only longer term studies can decide this question.

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REFERENCES

1. Denke MA: Cholesterol-lowering diets. A review of the evidence. *Arch Int. Med* 155:17–26, 1995.
2. Fraser GE: Nut consumption, lipids, and risk of a coronary event. *Clin Cardiol* 22 (Suppl III), S11–S15, 1999.
3. Spiller GA, Gates JE, Jenkins DAJ, Bosello O, Nichols SF, Cragen L: Effects of two foods high in monounsaturated fat on plasma

- cholesterol and lipoproteins in adult humans [Abstract]. *Am J Clin Nutr* 51:524, 1990.
4. Curb JD, Wergowski G, Abbott RD, Dobbs JC, Tung J, Austin MA, Marcovina S: High monounsaturated fat macadamia nut diets; effects on serum lipids and lipoproteins [Abstract]. *FASEB J Abstracts*, Part I, A506, 1998.
 5. Curb J, Wergowski G, Dobbs JC, Abbott RD, Huang B: Serum lipid effects of a high-monounsaturated fat diet based on macadamia nuts. *Arch Int Med* 160:1154–1158, 2000.
 6. Almario RU, Vonghavaravat V, Wong R, Kasim-Karakas SE: Effects of walnut consumption on plasma fatty acids and lipoproteins in combined hyperlipidemia. *Am J Clin Nutr* 74:72–79, 2001.
 7. Willett WC: Is dietary fat a major determinant of body fat? *Am J Clin Nutr* 67(Suppl): 556S–562S, 1998.
 8. Seidell JC: Dietary fat and obesity: an epidemiologic perspective. *Am J Clin Nutr* 67(Suppl):546S–550S, 1998.
 9. Bray GA, Popkin BM: Dietary fat intake does affect obesity! *Am J Clin Nutr* 68:1157–1173, 1998.
 10. Golay A, Allaz AF, Morel Y, de Tonnac N, Tankova S, Reaven G: Similar weight loss with low- or high-carbohydrate diets. *Am J Clin Nutr* 63:174–178, 1996.
 11. Lehmann T, Golay A, James RW, Pometta D: Effects of two hypocaloric diets, fat restricted or rich in monounsaturated fat, on body weight loss and plasma lipoprotein distribution. *Nutr Metab Cardiovasc Dis* 5:290–296, 1995.
 12. Nutrition Data System: Nutrition Coordinating Center (NCC), University of Minnesota, 1993.
 13. Folch J, Lee M, Sloane-Stanley GH: A simple method for the isolation and purification of total lipids from animal tissues. *J Biol Chem* 226:497–509, 1957.
 14. Watkins SM, Lin TY, Davis RM, Ching JR, DePeters EJ, Halpern GM, Walzem RL, German JB: Unique phospholipid metabolism in mouse heart in response to dietary docosahexaenoic or α -linolenic acids. *Lipids* 36:247–254, 2001.
 15. Singh PN, Tonstad S, Abbey DE, Fraser GE: Validity of selected physical activity questions in white Seventh-Day Adventists and non-Adventists. *Med Sci Sports Exerc* 28:1026–1036, 1996.
 16. Sensormedics Corp., Yorba Linda, CA, 1983–90.
 17. SAS/STAT Software. SAS Institute Inc., Cary, NC, 1997.
 18. S-PLUS 4. Math Soft Inc., Cambridge, MA, 1999.
 19. Sarkkinen ES, Agren JJ, Ahola I, Ovaskainen M-L, Uusitupa MIJ: Fatty acid composition of serum cholesterol esters, and erythrocyte and platelet membranes as indicators of long-term adherence to fat-modified diets. *Am J Clin Nutr* 59:364–370, 1994.
 20. Berry EM, Eisenberg S, Friedlander Y, Harats D, Kaufmann NA, Norman Y, Stein Y: Effects of diets rich in monounsaturated fatty acids on plasma lipoproteins—the Jerusalem Nutrition Study. II. Monounsaturated fatty acids vs. carbohydrates. *Am J Clin Nutr* 56:394–403, 1992.
 21. Tynan MB, Nicholls DP, Maguire SM, Steele IC, McMaster C, Moore R, Trimble ER, Pearce J: Erythrocyte membrane fatty acid composition as a marker of dietary compliance in hyperlipidemic subjects. *Atherosclerosis* 117:245–252, 1995.
 22. Romon M, Nuttens M-C, Theret N, Delbart C, Lecerf J-M, Fruchart J-C, Salomez J-L: Comparison between fat intake assessed by 3-day food record and phospholipid fatty acid composition of red blood cells: Results from the Monitoring of Cardiovascular Disease—Lille Study. *Metabolism* 44:1139–1145, 1995.
 23. Lands WEM: Long-term fat intake and biomarkers. *Am J Clin Nutr* 61 (Suppl):721S–725S, 1995.
 24. Drewnowski A: Energy density, palatability, and satiety: implications for weight control. *Nutr Rev* 56:347–353, 1998.
 25. Haddad EH, Sabaté J: Effect of pecan consumption on stool fat. *FASEB J (Abstracts)* 14(4):A294, 2000.
 26. Levine AS, Silvis SE: Absorption of whole peanuts, peanut oil and peanut butter. *New Engl J Med* 303:917–918, 1980.
 27. Lissner L, Heitmann BL: Dietary fat and obesity: evidence from epidemiology. *Euro J Clin Nutr* 49:79–90, 1995.
 28. Kant AK, Graubard BI, Schatzkin A, Ballard-Barbash R: Proportion of energy intake from fat and subsequent weight change in the NHANES I Epidemiologic Follow-up Study. *Am J Clin Nutr* 61:11–17, 1995.
 29. Colditz GA, Willett WC, Stampfer MJ, London SJ, Segal MR, Speizer FE: Patterns of weight change and their relation to diet in a cohort of healthy women. *Am J Clin Nutr* 51:1100–1105, 1990.
 30. Lissner L, Levitsky DA, Strupp BJ, Kalkwarf HJ, Roe DA: Dietary fat and the regulation of energy intake in human subjects. *Am J Clin Nutr* 46:886–892, 1987.
 31. Kendall A, Levitsky DA, Strupp B, Lissner L: Weight loss on a low fat diet: consequence of the imprecision of the control of food intake in humans. *Am J Clin Nutr* 53:1124–1129, 1991.
 32. Hunninghake DB, Stein EA, Dujovne CA, Harris WS, Feldman EB, Miller VT, Tobert JA, Laskarzewski PM, Quiter E, Held J: The efficacy of intensive dietary therapy alone or combined with lovastatin in outpatients with hypercholesterolemia. *New Engl J Med* 328:1213–1219, 1993.
 33. Lee-Han H, Cousins M, Beaton M, McGuire V, Kriukow V, Chipman M, Boyd N: Compliance in a randomized clinical trial of dietary fat reduction in patients with breast dysplasia. *Am J Clin Nutr* 48:575–586, 1988.
 34. Boyd NF, Cousins M, Beaton M, Kriukov V, Lockwood G, Trichter D: Quantitative changes in dietary fat intake and serum cholesterol in women: results from a randomized, controlled trial. *Am J Clin Nutr* 52:470–476, 1990.
 35. Kasim SE, Martino S, Kim P, Khilnani S, Boomer A, Depper J, Reading BA, Heilbrun LK: Dietary and anthropometric determinants of plasma lipoproteins during a long-term low-fat diet in healthy women. *Am J Clin Nutr* 57:146–153, 1993.
 36. Alford BB, Blankenship AC, Hagen RD: The effects of variations in carbohydrate, protein and fat content of the diet upon weight loss, blood values, and nutrient intake of adult obese women. *J Am Diet Assoc* 90:534–540, 1990.
 37. Prewitt TE, Schmeisser D, Bowen PE, Aye P, Dolecek TA, Langenberg P, Cole T, Brace L: Changes in body weight, body composition, and energy intake of women fed high and low fat diets. *Am J Clin Nutr* 54:304–310, 1991.
 38. Rumpler WV, Seale JL, Miles CW, Bodwell CE: Energy-intake restriction and diet composition effects on energy expenditure in men. *Am J Clin Nutr* 53:430–436, 1991.
 39. Powell JJ, Tucker L, Fisher AG, Wilcox K: The effects of different percentages of dietary fat intake, exercise, and caloric restriction on body composition and body weight in obese females. *Am J Health Promot* 8:442–448, 1994.
 40. Jeffery RW, Hellerstedt WL, French SA, Baxter JE: A randomized

- trial of counselling for fat restriction versus calorie restriction in the treatment of obesity. *Int J Obes* 19:132–137, 1995.
41. Garg A: High-monounsaturated-fat diets for patients with diabetes mellitus: a meta-analysis. *Am J Clin Nutr* 67(Suppl):577S–582S, 1998.
42. Shimomura Y, Tamura T, Suzuki M: Less body fat accumulation in rats fed safflower oil diet than in rats fed a beef tallow diet. *J Nutr* 120:1291–1296, 1990.
43. Loh MY, Flatt WP, Martin RJ, Hausman DB: Dietary fat type and level influence adiposity development in obese but not lean Zucker rats. *Proc Soc Exp Biol Med* 218:38–44, 1998.
44. Jones PJH, Ridgen JE, Phang T, Birmingham CL: Influence of dietary fat polyunsaturated to saturated ratio on energy substrate utilization in obesity. *Metabolism* 41:396–401, 1991.

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