British Journal of Nutrition (2005), **94**, 859–864
DOI: 10.1079/BJN20051567

Does regular walnut consumption lead to weight gain?

Joan Sabaté*, Zaida Cordero-MacIntyre, Gina Siapco, Setareh Torabian and Ella Haddad

Department of Nutrition, School of Public Health, Loma Linda University, Loma Linda, CA 92350, USA

(Received 8 November 2004 – Revised 10 May 2005 – Accepted 30 June 2005)

Studies consistently show the beneficial effects of eating nuts, but as high-energy foods, their regular consumption may lead to weight gain. We tested if daily consumption of walnuts (approximately 12 % energy intake) for 6 months would modify body weight and body composition in free-living subjects. Ninety participants in a 12-month randomized cross-over trial were instructed to eat an allotted amount of walnuts (28–56 g) during the walnut-supplemented diet and not to eat them during the control diet, with no further instruction. Subjects were unaware that body weight was the main outcome. Dietary compliance was about 95 % and mean daily walnut consumption was 35 g during the walnut-supplemented diet. The walnut-supplemented diet resulted in greater daily energy intake (557 kJ (133 kcal)), which should theoretically have led to a weight gain of 3·1 kg over the 6-month period. For all participants, walnut supplementation increased weight (0·4 (SE 0·1) kg), BMI (0·2 (SE 0·1) kg/m^2), fat mass (0·2 (SE 0·1) kg) and lean mass (0·2 (SE 0·1) kg). But, after adjusting for energy differences between the control and walnut-supplemented diets, no significant differences were observed in body weight or body composition parameters, except for BMI (0·1 (SE 0·1) kg/m^2). The weight gain from incorporating walnuts into the diet (control → walnut sequence) was less than the weight loss from withdrawing walnuts from the diet (walnut → control sequence). Our findings show that regular walnut intake resulted in weight gain much lower than expected and which became non-significant after controlling for differences in energy intake.

Walnuts: Body weight: Body mass index: Body composition: Weight change: Cross-over design

Studies have consistently shown that nut consumption is associated with reduced risk of CVD (Sabaté & Fraser, 1994; Hu & Stampfer, 1999; Sabaté, 1999) and improved serum lipid and lipoprotein profiles (Sabaté & Fraser, 1994; Kris-Etherton *et al.* 1999*b*; Sabaté *et al.* 2001), which are important biomarkers of disease risk. Because of its cardio-protective effects, incorporating nuts into the daily diet is now recommended by the American Heart Association (Krauss *et al.* 2000). Nuts are fat-rich foods but most of the fats are unsaturated (US Department of Agriculture, 2001), which could partly explain their beneficial effects. However, as an energy-dense food, nut consumption without regard to other factors that affect energy balance, e.g. reduction in energy intake from certain other foods or physical activity, may potentially lead to weight gain. Nevertheless, preliminary evidence suggests otherwise (Sabaté, 2003).

All large epidemiological studies show that nut consumption is either inversely or not associated with BMI (Fraser *et al.* 1992; Hu *et al.* 1998; Albert *et al.* 2002). Feeding trials where nuts isoenergetically replaced other foods in the diet indicate that short-term intake of moderate to large amounts of nuts results in either a non-significant weight loss or an absence of weight change (Sabaté *et al.* 1993; Abbey *et al.* 1994; Colquhoun *et al.* 1996; Spiller *et al.* 1998; Kris-Etherton *et al.* 1999*a*; Zambon *et al.* 2000; Rajaram *et al.* 2001; Iwamoto *et al.* 2002; Sabaté *et al.* 2003). Conversely, free-feeding studies wherein subjects consumed nuts in addition to their usual diet show minimal weight gain that is both statistically and clinically non-significant. Moreover, the reported increases in weight are much less than the predicted levels when the accumulated excess energy from nut intake is considered (Alper & Mattes, 2002; Fraser *et al.* 2002; St-Onge, 2005).

While regular nut-eating has a host of health benefits, its subsequent effects on weight may appear to be a deterrent in promoting such dietary advice (Sabaté, 2003). Potential effects on weight may also be coupled with changes in body composition and as such have been reported in only two studies of regular nut consumption (Alper & Mattes, 2002; Wien *et al.* 2003). In the present study, we aimed to determine the potential changes in body weight and body composition when free-living subjects who are not given additional dietary advice incorporate moderate amounts of walnuts (28–56 g, approximately 12 % of daily energy intake) into their diet for 6 months.

Subjects and methods

Subjects

Individuals from various Southeast California communities who responded to recruitment advertisements underwent a selection process which included two telephone screenings, an informational meeting and a personal interview. Eligibility criteria included weight change <1 kg during the previous 6 months, BMI <35 kg/m^2, and habitual diet including nuts less than once a week. A diagnosed metabolic disorder that can affect weight, i.e. diabetes, hypothyroidism, or aversion or known allergy to nuts, excluded an individual from the study. Of the ninety-four subjects enrolled in the study, two dropped out due to compliance difficulty and two were withdrawn when diagnosed with

* **Corresponding author:** Dr Joan Sabaté, fax +1 909 558 4095, email jsabate@llu.edu

a metabolic disorder at the time of the study. Thus, fifty females and forty males aged 30 to 72 years (mean 54·3 (SD 10·6) years) made up the analytic study population.

Study design

The study was a randomized cross-over field trial which included two 6-month diet periods, a control diet and a walnut-supplemented diet. Subjects underwent one diet for six consecutive months and then switched to the other diet for the next six consecutive months. At baseline, we randomly assigned participants to two treatment groups: the walnut-supplemented-to-control (walnut → control) and the control-to-walnut-supplemented (control → walnut). Based on our previous report (Fraser *et al.* 2002), a sample size of 80 would allow detection of a 0·5 kg weight change significant at α of 0·05 with 80 % power.

Diet

Participants were asked to follow their usual diet. While on the walnut-supplemented diet, we provided participants with walnuts that corresponded to approximately 12 % of their daily energy intake. When on the control diet, we asked them to refrain from eating walnuts and substantial amounts of any other nuts. We instructed the participants not to change their physical activity habits, and not to attempt to lose weight while in the study. To keep the study as free-living as possible no other guidance was given. Participants were unaware that body weight and body composition were the focus of the study.

Initial allotment of walnuts was based on basic energy expenditure computed using the WHO equations presented in the *Recommended Dietary Allowances* (National Research Council, 1989). In subsequent clinic visits, walnut allotment was adjusted based on the daily energy intake reported in the 24 h dietary recalls. For subjects with average reported daily energy intake of up to 7535 kJ (1800 kcal), the daily supplement of walnuts was 28 g; 37 g was allotted for those with intake of 7535–9628 kJ (1800–2300 kcal), 46 g for 9628–11 721 kJ (2300–2800 kcal), and 56 g for >11 721 kJ (2800 kcal). The walnuts were provided free of charge and in individually labelled packets with amounts in grams, one for each day of the week. An additional large pack of walnuts was given for the consumption of family members to make sure they would not consume the subject's walnut allocation. Walnuts were distributed at each clinic visit every 2 months. Subjects were requested to return to the investigators any unconsumed portion. Because walnuts are a particularly rich source of α-linolenic acid (9·1 %), its erythrocyte membrane concentration at the end of each diet period was measured as a biological marker of adherence to the intervention.

Dietary recalls

To assess dietary compliance as well as measure total energy intake, research nutritionists collected 24 h dietary recalls through telephone interviews with the participants. The research nutritionists were Master's level nutrition students who were trained on the use of Nutrition Data Systems for Research (Nutrition Coordinating Center, University of Minnesota, Minneapolis, MN, USA, 1998–2000). Telephone interviews were unannounced and administered non-consecutively to reduce the possibility that subjects would change their intake. To reduce inter-interviewer biases, the interviewers followed protocols.

We collected seven recalls from each participant during each diet period (a total of fourteen recalls for both diet periods). To capture daily variations in intake, all days of the week (two weekend days and five weekdays) were covered by the seven recalls.

Anthropometric measures

All anthropometric measurements were taken at each clinic visit: at baseline and every 2 months up to 12 months. Body weight and body composition were taken using the Tanita® TBF 300A Bioelectrical Impedance Analysis BIA scale (Tanita Corporation of America, Arlington Heights, IL, USA). Measurements were taken early in the morning, without shoes or heavy clothing and with empty pockets, and recorded to 0·1 kg. Subjects were weighed twice and the mean was used for data analysis (Lohman *et al.* 1991).

Height was measured using a wall-mounted stadiometer (Haltain Ltd, Crymych, Dyfed, UK) with subjects standing in an upright position without shoes. Two measurements, each to 0·1 cm, were taken and the mean was used for data analysis (Lohman *et al.* 1991).

Physical activity

An exercise questionnaire (Singh *et al.* 2001), previously used in other nut studies conducted by our institution, was completed by the participants at each clinic visit. Two sections in the questionnaire asked for the frequency of exercise sessions per week and the amount of time spent per session on the following physical activities: vigorous walking or hiking, running or jogging, aerobics or dancing, vigorous bicycling, stationary cycling or rowing, lap-swimming, tennis and other vigorous sports, moderate/heavy labour at work, vigorous yard work/gardening, resistance training, and other vigorous exercise. We computed the total exercise time per week by multiplying the frequency of exercise sessions per week with time spent per session. Total exercise time per week was averaged for each diet period and then compared.

Statistical analysis

Descriptive statistics (means and their standard errors) were calculated for subject characteristics and outcome variables at baseline. Tests for significant differences between treatment sequences were conducted by using two-sample *t* tests, except for gender (female %) for which χ^2 test was used. Outcome variables included in this analysis were body weight, BMI, fat mass, percentage body fat, fat-free mass and total body water. Tests for significant differences in outcome variables for each treatment sequence and for a significant sequence effect were conducted by using mixed linear models that included a random term for subjects and fixed terms for diet, period and their interaction. Paired *t* tests to compare within-subject differences in walnut intake, total energy and energy from walnut intakes, percentage dietary compliance and physical exercise were performed for both treatment sequence groups and all participants. All analyses were done using SAS System for Windows version 8.0 (SAS Institute, Cary, NC, USA).

Results

Table 1 presents selected subject characteristics at baseline according to treatment sequence. The two treatment sequence groups were similar (all $P > 0{\cdot}05$) in mean age, height, body weight, BMI, body composition parameters and gender distribution.

As shown in Table 2, walnut intake for all participants averaged 35·2 g (range 17·7–56·0 g) during the walnut-supplemented diet period and 0·5 g (range, 0·0–11·1 g) during the control diet period. We prescribed walnuts to account for approximately 12 % of total energy intake during the walnut supplementation; this percentage is reflected in the subjects' actual energy intake from walnuts. Across the treatment sequence groups and the diet periods, degree of dietary compliance was excellent at about 95 %. Compliance is defined as non-intake of walnuts (intake < 2 g) during the control period and intake of the allotted amounts of walnuts (intake ≥ 28 g) during the walnut-supplemented period.

We also assessed walnut intake compliance by measuring changes of α-linolenic acid concentration in the erythrocyte membrane at the end of each dietary period. Of the eighty-six subjects for whom we had fatty acid data, seventy-seven (89·5 %) had an increase of α-linolenic acid on the walnut diet compared with the control diet. Mean concentration of α-linolenic acid increased significantly ($P < 0{\cdot}001$) by 38·9 % from 0·185 (SE 0·006) mol% on the control diet to 0·257 (SE 0·006) mol% on the walnut diet.

By incorporating an average of 35 g of walnuts daily for 6 months, the theoretical weight gain, i.e. without dietary compensation, was 5·3 kg. Subjects while on the walnut-supplemented period had a higher total energy consumption, 8171 kJ (1952 kcal), than during the control period, 7614 kJ (1819 kcal). The mean difference between daily total energy intake during the two diet periods (557 (SE 142) kJ (133 (SE 34) kcal)) is less than the actual energy intake from walnuts (967 kJ (231 kcal)), which suggests partial substitution of other foods in the walnut-supplemented diet. Nevertheless, this greater daily energy intake (557 kJ (133 kcal)) during the walnut-supplemented diet should theoretically have led to a weight gain of 3·1 kg over the 6-month period (National Institutes of Health & National Heart, Lung and Blood Institute, 1998).

There were no significant changes in the amount of time spent on physical exercise between the two diet periods for both

Table 1. Subject characteristics at baseline according to dietary treatment sequence

(Mean values and standard deviations)

	Dietary treatment sequence			
	Control → walnut (n 41)		Walnut → control (n 49)	
	Mean	SD	Mean	SD
Body weight (kg)	78·5	15·0	73·3	13·1
Height (cm)	170·4	11·9	167·4	9·0
BMI (kg/m^2)	26·9	3·3	26·1	3·5
Fat mass (kg)	24·1	5·8	22·8	6·5
Body fat (%)	31·1	7·1	31·1	7·1
Fat-free mass (kg)	54·4	13·4	50·6	10·9
Total body water (kg)	39·8	9·8	37·0	7·9
Age (years)	53·1	11·4	55·5	9·9
Female (%)	59·2		53·7	

Table 2. Daily energy and walnut intakes, dietary compliance and physical activity assessment

(Mean values with their standard error)

	Control → walnut (n 41)				Walnut → control (n 49)				All subjects (n 90)			
	Control diet		Walnut diet		Walnut diet		Control diet		Control diet		Walnut diet	
	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE
Walnut intake (g)	0·5	0·2	34·0	1·4	36·3	1·1	0·6	0·3	0·5	0·2	35·2	0·87
Energy from walnuts (kJ) [kcal]	13·8 [3·3]	5·0 [1·2]	930·5 [222·3]	38·5 [9·2]	993·3 [237·3]	28·9 [6·9]	15·9 [3·8]	7·1 [1·7]	15·1 [3·6]	4·6 [1·1]	965 [230·5]	23·9 [5·7]
Total energy intake (kJ) [kcal]	7757 [1853]	427 [102]	8288 [1980]	481 [115]	8071 [1928]	318 [76]	7493 [1790]	360 [86]	7614 [1819]	276 [66]	8171 [1952]	280 [67]
% energy from walnuts	0·2	0·1	11·8	0·4	12·9	0·5	0·2	0·11	0·2	0·1	12·4	0·3
Dietary compliance* (%)	95·6		95·2		95·3		95·6		95·6		95·2	
Physical exercise (min/week)	262·1	34·2	247·2	32·8	227·4	26·8	216·0	24·1	237·3	20·5	236·3	20·8

*Dietary compliance is calculated as follows: for the control diet, number of recall days with walnut intake $< 2{\cdot}5$ g/total number of recalls during control diet; for the walnut diet, number of recall days with walnut intake ≥ 28 g/total number of recalls during walnut diet.

treatment sequence groups and the whole group (Table 2). Moreover, the treatment sequence groups did not differ in the amount of time spent on exercise. We also compared the amount of exercise time between the two diet periods according to categories of weight change (weight lost, no change or weight gained) and found no significant differences (data not shown). Thus, all subjects maintained their exercise levels throughout the study as directed.

Table 3 summarizes the results for each outcome variable by treatment sequence and for the whole group. Data are presented segregated by sequence because of our *a priori* hypothesis that body weight change would be different according to sequence. A formal test of the time–diet interaction (sequence effect) showed a difference of effect in body weight of 0·15 kg ($P=0·09$). No significant sequence effect was found for the rest of the outcome variables, thus the presentation also of results combining both sequences.

When the control → walnut sequence group incorporated walnuts in their usual diet for 6 months, there was a modest non-significant increase in body weight (0·4 (SE 0·2) kg) and BMI (0·1 (SE 0·1) kg/m^2). However, fat mass and percentage body fat decreased significantly by 0·4 (SE 0·2) kg ($P=0·04$) and 0·6 (SE 0·2) % ($P<0·0001$), respectively. Fat-free mass and total body water increased significantly ($P<0·0001$) by 0·8 (SE 0·1) kg and 0·6 (SE 0·1) kg, respectively. Energy adjustment (i.e. taking into account differences in energy intake between and within subjects) halved the non-significant difference in body weight between the diet periods (0·2 (SE 0·2) kg) and did not considerably change the results for body composition parameters.

When the walnut → control sequence group stopped consuming walnuts after 6 months, body weight, BMI, fat mass and percentage body fat decreased significantly by 0·5 (SE 0·2) kg ($P=0·004$), 0·2 (SE 0·1) kg/m^2 ($P<0·001$), 0·8 (SE 0·2) kg ($P<0·001$) and 1 (SE 0·2) % ($P<0001$), respectively. Fat-free mass and total body water increased significantly by 0·4 (SE 0·1) kg and 0·3 (SE 0·1) kg, respectively (both $P<0·001$). No significant difference in body weight was observed after adjusting for energy while differences in body composition parameters did not materially change with energy adjustment.

Results for analysis of the combined data showed that except for BMI, energy-adjusted changes in body weight, fat mass, percentage body fat, fat-free mass and total body water were minimal and not significant. Since participants did not spontaneously fully displace the extra energy provided by walnuts during the walnut-supplemented diet (control → walnut sequence), nor did they fully replace the walnut energy during the control diet (walnut → control sequence), energy-adjusted results are the best possible estimate of the outcome if walnuts were isoenergetically incorporated in the diet.

Discussion

We determined the effects of regular incorporation or removal of moderate amounts of walnuts in the diet on body weight and body composition and found a minimal body weight change that is much less than predicted. The differences in energy intake brought about by the addition or taking away of walnuts in the diet accounted for this body weight change. When energy intake differences are controlled for, this body weight effect disappears.

We found that the weight increase due to the addition of walnuts in the diet and the weight loss attributed to the withdrawal of walnuts from the diet are much lower than theoretical values. On the basis of the difference in daily energy intake, a theoretical weight change of 3·1 kg would have been expected over the 6-month period (National Institutes of Health & National Heart, Lung and Blood Institute, 1998). For all participants, daily walnut intake ranged between 17 and 56 g (mean 35·2 g) during the walnut diet period. If they had not partially compensated for the energy provided by walnuts, a weight gain of 5·3 kg would have been expected. Our results show an average body weight gain of only about one-tenth (0·4 kg) and a statistically non-significant body fat gain of 0·2 kg.

Table 3. Body weight and body composition changes by dietary treatment sequence and for all subjects

(Mean values with their standard error)

	Body weight (kg)		BMI (kg/m^2)		Fat mass (kg)		Body fat (%)		Fat-free mass (kg)		Total body water (kg)	
	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE
Sequence control → walnut (*n* 41)												
Control diet	78·3	2·2	26·8	0·5	24·5	1·0	31·7	1·1	53·9	1·9	39·5	1·4
Walnut diet	78·7	2·2	26·9	0·5	24·1	1·0	31·0	1·1	54·6	1·9	40	1·4
Raw difference†	0·4	0·2	0·1	0·1	−0·4*	0·2	−0·6**	0·2	0·8**	0·1	0·6**	0·1
Energy-adjusted difference	0·2	0·2	0·1	0·1	−0·5*	0·2	−0·7**	0·2	0·7**	0·1	0·5**	0·1
Sequence walnut → control (*n* 49)												
Control diet	73·7	2·0	26·2	0·5	23·7	0·9	32·2	1·0	50·1	1·8	36·7	1·3
Walnut diet	73·2	2·0	26·0	0·49	22·8	0·89	31·2	1·0	50·5	1·8	36·9	1·3
Raw difference	−0·5*	0·2	−0·2**	0·1	−0·8**	0·2	−1·0**	0·2	0·4**	0·1	0·3**	0·1
Energy-adjusted difference	−0·3	−0·2	−0·2*	0·1	−0·8**	0·2	−0·9**	0·2	0·4**	0·1	0·3**	0·1
All subjects (*n* 90)												
Control diet	75·6	1·5	26·4	0·4	23·6	0·7	31·4	0·8	52·0	1·3	38·1	1·0
Walnut diet	76·0	1·5	26·5	0·4	23·8	0·7	31·6	0·8	52·2	1·3	38·2	1·0
Raw difference	0·4**	0·1	0·2**	0·1	0·2*	0·1	0·2	0·1	0·2*	0·1	0·1*	0·1
Energy-adjusted difference	0·2	0·1	0·1*	0·1	0·1	0·1	0·1	0·1	0·2	0·1	0·1	0·1

Mean values were significantly different: *$P<0.05$, **$P<0.01$.

†The calculation of differences was done as follows: for the control → walnut sequence, the difference between walnut diet and control diet values; for the walnut → control sequence, the difference between control diet and walnut diet values; for all subjects combined, the difference between walnut diet and control diet values.

The effect on weight was greater when walnuts were withdrawn from the diet (−0·52 kg) than when they were added (+0·37 kg). While a training effect due to increased awareness of one's diet might have contributed to this difference, the possibility that subjects partially displaced certain other foods when they were on the walnut diet cannot be discounted (Fraser *et al.* 2002). The different effects on weight between the two diet sequences illustrate the potential limitations of cross-over designs on behavioural treatments such as diet modification in free-living conditions.

Results of the present study on body weight are in agreement with other reports on previous work that involved adding nuts into the diet of free-living subjects. Fraser *et al.* (2002) reported that daily consumption of 54·3 g of almonds for 6 months modestly increased weight by 0·40 kg. Alper & Mattes (2002) found a 1 kg body weight increase among free-living subjects who consumed 90 g of peanuts daily for 8 weeks. As in our study, reported actual weight changes for both these studies are lower than the expected values.

Several mechanisms, such as increased satiety levels, increased resting energy expenditure or energy malabsorption, can potentially explain the lower-than-expected weight gain due to walnut consumption among our subjects (Sabaté, 2003; St-Onge, 2005). Walnuts have a high ratio of polyunsaturated to saturated fatty acids, and in the human diet a similarly high ratio can increase diet-induced thermogenesis (Jones & Schoeller, 1988). Resting energy expenditure was greater among subjects after a 19-week peanut-supplemented diet (Alper & Mattes, 2002). Decreased fat absorption may be another explanation for the minimal, and much less than expected, weight gain. Decreased fat absorption from nuts may be due to the fibre content of nuts or to the structure of lipid-storing granules in the nuts (Ellis *et al.* 2004). It has been reported that consuming whole nuts may lead to a certain degree of poor fat absorption due to incomplete mastication. As such, some of the fat contained in the nuts becomes unavailable, decreasing the total energy value of what is consumed. Levine & Silvis (1980) reported that, regardless of the fibre content in a diet, subjects consuming whole peanuts excreted a higher amount of fat in their faeces than when the same subjects consumed peanut butter or peanut oil. A controlled feeding study with pecans conducted in our laboratory showed that subjects who consumed a pecan-rich diet (20 % of energy from pecans) for 4 weeks had significantly higher amounts of fat excreted in the faeces than the pecan-free diet (control) group (Haddad & Sabaté, 2000). In a feeding study on almonds, subjects consuming almond-containing diets had significantly higher faecal fat than on the free-nut (control) diet (Zemaitis & Sabaté, 2001).

Removing walnuts from the diet of the walnut → control sequence group resulted in a weight loss that paralleled a decrease in fat mass and percentage body fat, but increases in fat-free mass and total body water were almost half those values for the control → walnut sequence group. Total body water is affected by hydration status, which may change from day to day in most individuals. We checked if there had been differences in water intake between the two diet periods and found that the walnut → control treatment sequence group had a significantly lower water intake during the walnut-supplemented diet (4·7 (SE 0·4) *v.* 5·3 (SE 0·3) cups, $P=0·01$). Water intake of the control → walnut treatment sequence group remained the same during both diet periods. Body composition could also be affected by physical activity, but we ascertained that physical exercise did not change between the two diet periods. Since our subjects had been instructed to fast for 10–12 h the night before body measurements were taken except for water, between-subject differences in water intake before the clinics may have affected the bioelectrical impedance analysis readings for body composition. Thus, the increments in total body water and fat-free mass during the walnut-supplemented diet could be artifactual.

The limited number of nut studies that specifically looked at the impact of nut consumption on body weight and body composition changes all point out the fact that daily nut supplementation poses no risk of significant weight gain. Our findings indicate that although eating moderate amounts of walnuts daily for 6 months could lead to very minimal weight gain, such increase is much less than what is expected from the increment in energy intake due to walnuts. Further research is needed on potential mechanisms to explain the lower-than-expected weight gain due to nuts consumption found in this and other studies. In consideration of the present obesity epidemic situation (Mokdad *et al.* 2003) and the health benefits of walnut consumption, any recommendations for their regular intake should be coupled with suggestions to maintain energy intake, i.e. substituting walnuts for other foods, and energy expenditure through physical exercise.

References

Abbey M, Noakes M, Belling GB & Nestel PJ (1994) Partial replacement of saturated fatty acids with almonds or walnuts lowers total plasma cholesterol and low-density-lipoprotein cholesterol. *Am J Clin Nutr* **59**, 995–999.

Albert CM, Gaziano JM, Willett WC, Manson JE & Hennekens CH (2002) Nut consumption and decreased risk of sudden cardiac death in the Physician's Health Study. *Arch Intern Med* **162**, 1382–1387.

Alper CM & Mattes RD (2002) Effects of chronic peanut consumption on energy balance and hedonics. *Int J Obes Relat Metab Disord* **26**, 1129–1137.

Colquhoun DM, Humphries JA, Moores D & Somerset SM (1996) Effects of a macadamia nut enriched diet on serum lipids and lipoproteins compared to a low fat diet. *Food Aust: Off J Counc Aust Food Technol Assoc Aust Inst Food Sci Technol* **48**, 216–222.

Ellis PR, Kendall CW, Ren Y, Parker C, Pacy JF, Waldron KW & Jenkins DJ (2004) Role of cell walls in the bioaccessibility of lipids in almond seeds. *Am J Clin Nutr* **80**, 604–613.

Fraser GE, Sabaté J, Beeson WL & Strahan M (1992) A possible protective effect of nut consumption on risk of coronary heart disease. *Arch Intern Med* **152**, 1416–1424.

Fraser GE, Bennett HW, Jaceldo KB & Sabaté J (2002) Effect on body weight of a free 76 kilojoule (320 calorie) daily supplement of almonds for six months. *J Am Coll Nutr* **21**, 275–283.

Haddad E & Sabaté J (2000) Effect of pecan consumption on stool fat. *FASEB J* **14**, A294.

Hu FB & Stampfer MJ (1999) Nut consumption and risk of coronary heart disease: a review of epidemiologic evidence. *Curr Atheroscler Rep* **1**, 205–210.

Hu FB, Stampfer MJ, Manson JE, Rimm EB, Colditz GA, Rosner BA, Speizer FE, Hennekens CH & Willett WC (1998) Frequent nut consumption and risk of coronary heart disease in women: prospective cohort study. *BMJ* **317**, 1341–1345.

Iwamoto M, Imaizumi K, Sato M, Hirooka Y, Sakai K, Takeshita A & Kono M (2002) Serum lipid profiles in Japanese women and men during consumption of walnuts. *Eur J Clin Nutr* **56**, 629–637.

Jones PJ & Schoeller DA (1988) Polyunsaturated:saturated ratio of fat influences energy substrate utilization in the human. *Metabolism* **37**, 145–151.

Krauss RM, Eckel RH, Howard B, *et al.* (2000) AHA dietary guidelines revision 2000: a statement for healthcare professionals from the Nutrition Committee of the American Heart Association. *Circulation* **102**, 2284–2299.

Kris-Etherton PM, Pearson TA, Wan Y, Hargrove RL, Moriarty K, Fishell V & Etherton TD (1999*a*) High-monounsaturated fatty acid diets lower both plasma cholesterol and triacylglycerol concentrations. *Am J Clin Nutr* **70**, 1009–1015.

Kris-Etherton PM, Yu-Poth S, Sabaté J, Ratcliffe HE, Guixiang Z & Etherton TD (1999*b*) Nuts and their bioactive constituents: effects on serum lipids and other factors that affect disease risk. *Am J Clin Nutr* **70**, 504S–511S.

Levine AS & Silvis SE (1980) Absorption of whole peanuts, peanut oil, and peanut butter. *New Engl J Med* **303**, 917–918.

Lohman TG, Roche AF & Martorell R (1991) *Anthropometric Standardization Reference Manual*. abridged ed. Champaign, IL: Human Kinetics.

Mokdad AH, Ford ES & Bowman BA (2003) Prevalence of obesity, diabetes, and obesity-related health risk factors. *JAMA* **289**, 76–79.

National Institutes of Health & National Heart, Lung, and Blood Institute (1998) *Clinical Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults. The Evidence Report,* NIH Publication no. 98-4083, p. 74. Bethesda, MD: US Department of Health and Human Services, Public Health Service.

National Research Council, Subcommittee on the Tenth Edition of the RDAs (1989) *Recommended Dietary Allowances*, 10th ed., p. 24. Washington, DC: National Academy Press.

Rajaram S, Burke K, Connell B, Myint T & Sabaté J (2001) A monounsaturated fatty acid-rich pecan-enriched diet favorably alters the serum lipid profile of healthy men and women. *J Nutr* **131**, 2275–2279.

Sabaté J (1999) Nut consumption, vegetarian diets, ischemic heart disease risk, and all-cause mortality: evidence from epidemiologic studies. *Am J Clin Nutr* **70**, 500S–503S.

Sabaté J (2003) Nut consumption and body weight. *Am J Clin Nutr* **78**, 647S–659S.

Sabaté J & Fraser GE (1994) Nuts: a new protective food against coronary heart disease. *Curr Opin Lipidol* **5**, 11–16.

Sabaté J, Fraser GE, Burke K, Knutsen S, Bennett H & Lindsted KD (1993) Effect of walnuts on serum lipid levels and blood pressure in normal men. *N Engl J Med* **328**, 603–607.

Sabaté J, Haddad E, Tanzman JS, Jambazian P & Rajaram S (2003) Serum lipid response to the graduate enrichment of a Step I diet with almonds: a randomized feeding trial. *Am J Clin Nutr* **77**, 1379–1384.

Sabaté J, Radak T & Brown J (2001) The role of nuts in cardiovascular disease prevention. In *Handbook of Nutraceuticals and Functional Foods*, pp. 477–495 [REC Wildman, editor]. Boca Raton, FL: CRC Press.

Singh PN, Fraser GE, Knutsen SF, Lindsted KD & Bennett HW (2001) Validity of a physical activity questionnaire among African-American Seventh-day Adventists. *Med Sci Sports Exerc* **33**, 468–475.

Spiller GA, Jenkins DJA, Bosello O, Gates JE, Cragen LN & Bruce B (1998) Nuts and plasma lipids: an almond-based diet lowers LDL-C while preserving HDL-C. *J Am Coll Nutr* **328**, 603–607.

St-Onge MP (2005) Dietary fats, teas, dairy, and nuts: potential functional foods for weight control? *Am J Clin Nutr* **81**, 7–15.

US Department of Agriculture, Agricultural Research Service (2001) USDA Nutrient Database for Standard Reference, Release 14. http://nal.usda.gov/fnic/foodcomp.

Wien MA, Sabaté J, Ikle DN, Cole SE & Kandeel FR (2003) Almonds vs. complex carbohydrates in a weight reduction program. *Int J Obes Relat Metab Disord* **27**, 1365–1372.

Zambon D, Sabaté J, Munoz S, Campero B, Casals E, Merlos M, Laguna JC & Ros E (2000) Substituting walnuts for monounsaturated fat improves the serum lipid profile of hypercholesterolemic men and women, A randomized crossover trial. *Ann Intern Med* **132**, 538–546.

Zemaitis J & Sabaté J (2001) Effect of almond consumption on stool weight and stool fat. *FASEB J* **15**, A602.