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Effect of chronic consumption of almonds on body weight in healthy humans

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Small changes of diet may reduce CVD risk. One example is the inclusion of nuts. They are rich in fibre, unsaturated fatty acids and phytonutrients. However, their fat content and energy density raise concerns that chronic consumption will promote weight gain. Randomised intervention studies are required to evaluate whether this concern is well founded. This study's aim was to determine if the inclusion of a 1440 kJ serving of almonds in the daily diet results in positive energy balance, and body composition change. During a 23-week cross-over design study, participants were required to consume almonds for 10 weeks and were provided no advice on how to include them in their diet. For another 10 weeks (order counter-balanced), participants followed their customary diet and there was a 3-week washout between. The study group consisted of twenty women. Potential mechanisms of energy dissipation were measured. Ten weeks of daily almond consumption did not cause a change in body weight. This was predominantly due to compensation for the energy contained in the almonds through reduced food intake from other sources. Moreover, inefficiency in the absorption of energy from almonds was documented ($P<0{\cdot}05$). No changes in resting metabolic rate, thermic effect of food or total energy expenditure were noted. A daily 1440 kJ serving of almonds, sufficient to provide beneficial effects on cardiovascular risk factors, may be included in the diet with limited risk of weight gain. Whether this can be generalised to other high-fat energy dense foods warrants evaluation.

Nuts: Metabolic advantage: Body-weight: Energy expenditure: Appetite

Epidemiological studies indicate that the consumption of nuts is inversely related to risk of CHD[1,2]. Subsequent studies have provided a mechanistic basis for these observations and indicate that the regular consumption of nuts lowers plasma concentrations of LDL- and oxidised-LDL cholesterol while preserving HDL-cholesterol[3,4]. These data provide a rationale for encouraging nut consumption among the population and are the basis for an FDA (Food and Drug Administration)-approved health claim relating to risk of heart disease.

However, the habitual inclusion of nuts, a high-fat food group, in the diet may promote weight gain which would potentially negate the beneficial effects on blood lipid profiles. Epidemiological studies challenge this concern, revealing no or a negative association between nut consumption and body weight[1,2]. These findings are supported by intervention studies demonstrating that the inclusion of nuts in the diet poses limited risk for significant weight gain[4,5]. A mechanistic study exploring how the inclusion of nuts in the diet fails to promote positive energy balance and weight gain has not been conducted.

Three explanations for the lack of expected weight gain noted in nut consumers have been proposed. First, constituents of almonds such as fibre or protein are associated with increased sensations of satiety[6–8]. Therefore, the energy contained in the consumed nuts may be offset by reductions in energy intake from other foods so that overall energy intake remains unchanged. Secondly, previous research has suggested that the regular consumption of nuts may lead to increases in energy expenditure[9]. This would, in part, adjust for the energy contributed by nuts. Thirdly, the energy content of nuts that is bioaccessible may be less than predicted as faecal fat excretion increases with consumption[10]. One study using almonds demonstrated that this is due to the cell wall's resistance to enzymatic degradation in the gastrointestinal tract which results in the encapsulation of fat[11]. The present study explored these mechanisms by measuring the effect of consuming a 1440 kJ portion of almonds, each day for 10 weeks, on energy intake, diet composition, energy expenditure, faecal energy excretion, body weight and body composition.

Subjects and methods

Eligibility criteria included being of good health, a non-smoker, weight stable (deviation of $<2{\cdot}5$ kg over the previous 3 months), BMI of 23–30 kg/m^2, using no medications that would interfere with the outcome measures and obtaining less than 5 % of daily energy from tree or ground nuts. On being accepted into the study, participants were randomised to one of two conditions, almond or control, using a computerised random number generator. The almond group was required to eat a 1440 kJ portion of raw, unsalted almonds each day for 10 weeks whereas the control group followed their usual diet. After the first 10 weeks and a subsequent 3-week washout period, the two groups crossed over so that

Abbreviations: RMR, resting metabolic rate; TBW, total body water; TEF, thermic effect of food.

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the almond group became the control group and vice versa. The participants were not given advice about how to incorporate the almonds in the diet and were not instructed to reduce their food intake from other sources. During both conditions, all participants underwent the same measurement procedures. Data were collected between December 2003 and August 2005.

Body weight was measured on a random morning of each week of the study. For this measure, the overnight fasted participant voided and wore a surgical gown. All body weight measurements were made on the same calibrated electronic scale. Fat mass and fat free mass were determined by air displacement plethysmography (Bod-Pod, Life Measurement, Inc., Concord, CA, USA) during weeks 1 and 10 of each study arm. The bod-pod has been validated as a sensitive measure to detect small to moderate body composition changes[12].

Resting metabolic rate (RMR) and the thermic effect of food (TEF) were measured by indirect calorimetry using a SensorMedics Vmax 29 n metabolic cart (SensorMedics, Anaheim, CA, USA). The instrument was calibrated before each measurement using known concentrations of gas. RMR measurements were made during weeks 1 and 8 of each study arm. The TEF measure was made during week 8 of each study arm. Participants were required to fast for 12 h and avoid alcohol or heavy exercise in the 24 h prior to measurement. On the test morning, participants were asked to report to the laboratory as soon as possible after waking and by a method of minimal energy expenditure. Upon entering the thermo-neutral laboratory, participants were asked to lie supine and as still as possible on a bed for 20 min. After the rest period, a clear plastic ventilated hood was placed over the participant's head for a period of 45 min. The last 15 min of measurements were used to determine the RMR.

To determine the TEF, an RMR measurement was made before participants consumed a 1672 kJ portion of almonds and 250 ml of water within a 15 min period. TEF was estimated by energy expenditure measurements made for 15 min of each hour over the following 6 h. During this time, the participant was required to remain in a supine position and was asked to remain as stationary as possible.

Physical activity was measured using a three-axis accelerometer (Stayhealthy Inc., Monrovia, CA, USA), and a 3 d activity log[13]. The accelerometer was worn on the waist from the time of rising in the morning to going to bed at night, and has been validated as a reliable method for measuring physical activity[14]. The activity log was split into 15 min sections and covered the whole 24 h period. Participants recorded the type and intensity of activities they engaged in for each 15 min period. These measures were made on three random days (two weekdays and a weekend day) during weeks 4 and 8 of each study arm.

Total energy expenditure was measured using doubly labelled water administered during week 8 of each study arm. On the first day of each assessment period, a baseline urine sample was collected from the overnight fasted participant followed by oral dosing with doubly labelled water. The individualised dose was determined by the estimated total body water (TBW) pool size. $H_2^{18}O$ and D_2O were administered at 2·5 g/kg estimated TBW (10 atom % excess) and 0·1 g/kg estimated TBW (99·8 atom % excess), respectively. Participants were then instructed to refrain from eating or drinking for the next 4 h. Following this 4 h period, an additional urine sample was collected, with further urine collections at 24 h, 7 d and 14 d.

Energy intake was measured on three random days of week 4 and 8 with the stipulation that the recording include two weekdays and one weekend day. Participants were interviewed in person or over the telephone to determine the previous day's food intake. The standardised interviews were conducted using NDS multipass software (University of Minnesota, Minneapolis, MN, USA).

The metabolisable energy of the almonds was determined by a controlled feeding study. Participants were required to report to the laboratory, following a 12 h fast, to eat all their meals on four consecutive days during week 10 of each study arm. All meals were provided and they were consumed in the laboratory dining area. The same menu was used during both arms of the study. The baseline diet provided between 10 500 and 12 000 kJ, depending on the body size of the participant, and was comprised of approximately 55 % carbohydrate, 35 % fat and 15 % protein. The nutrient content of the supplied diet was calculated using USDA (United States Department of Agriculture) nutrient tables. During the almond arm, participants also consumed 1440 kJ of almonds each day.

With the first meal of the controlled feeding period, participants ingested a food colour marker (blue). They were then instructed to collect all faeces in separate containers until further notice. With the final meal of day 4, participants were provided with another food colour marker (red) and asked to monitor their collections for its appearance. Faecal composites were made and a sample was freeze-dried before analysis for gross energy by bomb calorimetry (Parr Instruments, Moline, IL, USA). The digestibility coefficient of the diet was calculated as:

$$(\text{energy intake} - \text{faecal energy excretion})/\text{energy intake} = \text{digestibility coefficient}$$

Compliance with the protocol was determined in several ways. First, regular contact (weekly) was made with the participants where an informal discussion relating to the almond consumption was conducted. A considerable effort was made to establish a relationship of trust with the participant and it was emphasised that it was vital that non-compliance, for whatever reason, was reported to the investigator. It was stressed that there were no negative consequences of such reports. Secondly, six diet dairies were completed during the study. Thirdly, fasting plasma α-tocopherol concentrations were measured during week 1 and week 10 of each study arm. Plasma α-tocopherol levels were assayed by reverse phase high-performance liquid chromatography. Participants were informed that the blood drawn contained a metabolite that would confirm long-term almond consumption.

All data are reported as means (SD). Mean values were compared using a Student's t test or a repeated measures ANOVA. Statistical significance was set at $P<0·05$, two-tailed. This study was approved by the Purdue University Institutional Review Board and all participants signed an informed consent form.

Results

Twenty-four individuals were randomised to treatment groups. Four individuals failed to finish the study. It was not possible to ascertain their reason. All subjects lost to the study withdrew within the first 4 weeks of the commencement of the study. The mean age of the study group was 24 (SD 9) years. Details of the study group's physical characteristics are provided in Table 1.

Plasma α-tocopherol concentrations increased significantly following the 10 week almond treatment period by 21·6 % ($P<0·05$). As almonds are a rich source of this vitamin[15], this was interpreted as evidence that the participants were compliant with the requirement to consume the almonds.

Data on body composition over the study are presented in Table 1. There were no significant changes in body weight ($F(3,95)$ 0·66, $P>0·05$), percent fat ($F(3,94)$ 0·310, $P>0·05$), fat mass ($F(3,94)$ 0·500, $P>0·05$), fat free mass ($F(3,93)$ 0·197, $P>0·05$) or TBW ($F(3,93)$ 0·256, $P<0·05$).

The inclusion of 1440 kJ of almonds in the daily diet did not lead to a statistically significant increase in food intake at any time point ($F(3,76)$ 0·311, $P>0·05$) (Table 2). Fat intake increased significantly during the almond treatment period ($F(3,76)$ 4·648, $P<0·05$). No difference in protein intake ($F(3,76)$ 0·206, $P>0·05$) was detected. The additional energy in the diet derived from fat was accounted for by a non-significant reduction in carbohydrate intake ($F(3,76)$ 0·586, $P>0·05$) and a non-significant increase in energy intake ($F(3,76)$ 0·311, $P>0·05$). While not significant, energy intake was greater by 322 kJ during the almond supplementation period. This suggests that dietary compensation for the energy contained in the almonds accounted for 74 % of the energy in the almonds.

Almond ingestion was associated with significant increases in the intake of PUFA ($F(3,77)$ 4·369, $P<0·05$) and MUFA ($F(3,77)$ 16·875, $P<0·05$) (Table 2). There was no significant change in the intake of saturated fatty acids. There were also significant increases of vitamin E ($F(3,77)$ 35·623, $P<0·05$) (Table 2), magnesium ($F(3,77)$ 18·311, $P<0·05$) and copper ($F(3,77)$ 9·365, $P<0·05$) Table 2).

No significant changes of RMR, TEF (Table 3) or physical activity as measured by the accelerometer or activity diary ($P>0·05$) were observed. However, there was a significant difference between the two methods for measuring physical activity, with the activity diary indicating a higher value. Total energy expenditure, as measured by doubly labelled water, did not differ between the two study periods.

Eating almonds led to a significant decrease in the digestibility coefficient of the diet (control = 0·96, almond = 0·95, $P<0·05$). This effect accounts for approximately 84 kJ/d of the energy contained in the daily almond portion. It was not possible to ascertain whether this decrease in digestibility was due to increased fat excretion.

Table 4 summarises the routes through which the energy in the 1440 kJ/d almond supplement was offset. Although the changes of energy expenditure were not statistically significant, based upon mean values, 95 % (based on doubly labelled water estimate of energy expenditure) to 98 % (based on the sum of the resting energy expenditure (REE), TEF and physical activity estimates) of the energy from the almond load was compensated.

Discussion

These data indicate that the consumption of a 1440 kJ serving of almonds each day for 10 weeks does not promote weight gain or changes in body composition. The increase in plasma α-tocopherol concentrations suggests that study participants consumed the almonds during the intervention study period.

The predominant mechanism through which the energy contained in the almonds was offset involved a spontaneous reduction of caloric intake from other dietary sources. The compensatory dietary response was 74 % of the energy contributed by the almonds. However, it is likely that the dietary recalls were subject to significant under-reporting as energy intake was significantly below the total energy expenditure as measured by doubly labelled water. Diet recalls are subject to several errors including under-reporting, due to forgotten foods or underestimated portion size, and altered eating behaviour due to participant's knowledge they are being observed. While this present study's use of a within-subjects design minimises these errors, they are still present and the diet recall data should be interpreted cautiously. One method to confirm the degree of dietary compensation would have been to include a laboratory-based test where the participant was given a 1440 kJ almond pre-load followed by an *ad libitum* buffet meal. However, due to the removal of the normal additional factors that influence food intake, the external validity of such results is uncertain.

Why almonds promote strong dietary compensation is still unclear. Almonds contain a significant amount of protein (21 g per 100 g of almonds), a macronutrient associated with increased satiation[6,16]. However, studies that demonstrate a satiating effect of protein use a far greater protein load than the 11 g supplied by the almonds in this study. Indeed, due to the compensatory dietary response to almonds, there was

Table 1. Changes in body composition due to the consumption of 1440 kJ/d of almonds

	Baseline	SD	Control week 1	SD	Control week 10	SD	Almond week 1	SD	Almond week 10	SD
Body weight (kg)	70·2	10·1	69·4	10·0	69·5	10·5	70·4	9·0	70·3	9·3
Height	1·6	0·1								
BMI	25·9	3·1								
% fat	34·0	6·2	33·6	7·2	35·4	8·4	35·8	8·0	36·7	7·8
FM (kg)			26·1	8·5	27·0	8·6	27·6	7·7	28·3	7·5
FFM (kg)			44·4	7·8	43·2	8·1	45·8	5·4	44·7	5·5
TBW (kg)			68·4	10·2	71·6	6·8	71·0	6·9	73·1	5·7

FM, fat mass; FFM, fat free mass; TBW, total body water.

Table 2. Energy, macronutrient and micronutrient intake measured during the control period and when 1440 kJ/d of almonds were consumed

	Control week 4	SD	Control week 8	SD	Mean	Almond week 4	SD	Almond week 8	SD	Mean
Energy (kcal/d)	1651	322	1700	400	1675	1746	397	1759	359	1752
Fat (g/d)	59·7	21·4	60·7	22·6	60·2	75·7*	16·8	78·0*	13·9	76·9
Protein (g/d)	65·9	15·7	65·9	17·4	65·9	70·0	29·9	69·0	15·7	69·5
CHO (g/d)	217·4	48·5	226·5	67·4	221	210·5	74·1	198·6	57·7	204
PUFA (g/d)	10·2	4·2	10·9	6·0		14·2*	3·5	13·9*	3·2	
MUFA (g/d)	22·5	9·3	22·8	8·7		35·0*	6·8	36·3*	6·3	
SFA (g/d)	21·3	7·5	22·0	8·4		19·4	7·2	22·8	6·9	
B_1	1·6	0·5	1·5	0·5		1·5	0·4	1·7	0·8	
B_2	2·0	0·9	1·8	0·5		1·9	0·5	2·2	0·8	
Niacin	21·0	6·8	19·4	6·1		18·2	5·4	22·9	8·4	
Pantenic acid	3·7	1·2	3·7	1·0		3·8	2·1	4·8	3·0	
B_6	1·7	0·7	1·6	0·6		1·4	0·5	1·7	0·8	
Folate	356	108	333	139		302	101	356	166	
B_{12}	4·0	2·6	4·1	2·1		3·2	1·6	4·0	4·0	
C	67·8	36·0	58·9	13·2		44·9	9·8	67·0	17·3	
D	3·2	1·8	3·6	1·9		4·9	8·0	4·1	3·1	
E	7·4	4·2	7·0	4·1		19·5*	6·4	20·4*	6·9	
Ca	710	239	758	294		834	302	831	357	
P	1012	185	1076	278		1160	243	1196	346	
Mg	212	55	219	74		326*	9	336*	96	
Fe	13·4	4·5	14·3	8·0		13·9	4·0	15·6	6·5	
Zn	8·7	3·0	9·1	3·1		9·3	3·1	10·7	3·7	
Cu	1·1	0·4	1·0	0·4		1·5*	0·3	1·6*	0·5	
Se	94·7	20·8	92·7	27·4		76·6	23·9	86·9	27·6	
Na	2866	730	2633	790		2461	770	2624	1029	
K	1923	498	1965	723		2206	546	2190	822	

* $P<0·05$.

only a net 5 g increase in protein intake. Such a small increment in protein intake is unlikely to have a marked effect on appetite. Almonds are a significant source of fibre, a food constituent with documented satiating properties[17]. However, it is also unclear that the 6·5 g of fibre contained in almonds could have a marked effect on appetitive sensations and food intake[18]. The crunchy textural property of almonds could also promote satiety[19]. It is possible a synergy between these, and possibly other, properties (e.g. micronutrients, flavour) of almonds account for their high satiety value.

Another possibility is that the participants cognitively adjusted their energy intake. While this cannot be dismissed, there are reasons to believe it was not the case. First, the participants were given the almonds in packets with no indication of their caloric value. Secondly, the study group consisted of predominantly overweight individuals who, by definition, fail to match energy intake to expenditure accurately.

The consumption of a 1440 kJ portion of almonds each day also had some beneficial effects on diet quality. During the almond-supplemented period, intake of MUFA and PUFA increased, which may have a positive effect on blood lipid profiles[20]. Saturated fatty acid concentrations remained unchanged. Other notable changes in diet quality were an increase in vitamin E, magnesium and copper. These changes in diet quality are noteworthy as mean vitamin E intake was only 7 mg/d, substantially lower than the RDA of 15 mg/d. Indeed, only one individual consumed the RDA. During the almond supplementation period, the mean intake of vitamin E was approximately 20 mg/d and every individual consumed more than the RDA. Average intakes of copper and magnesium were also below recommended levels. The addition of almonds to the diet brought the mean group intake above recommended levels.

This study did not confirm previous results in lean and obese individuals, using peanuts, that there is a significant increase in RMR following chronic nut consumption[9,21]. As body weight is the predominant determinant of RMR, it is unclear whether the previous results are anomalous or are

Table 3. Energy expenditure during the control period or when 1440 kJ/d almonds were being consumed

	Control week 1	SD	Control week 4	SD	Control week 8	SD	Almond week 1	SD	Almond week 4	SD	Almond week 8	SD
RMR (kcal/d)	1473	182			1446	236	1455	200			1499	195
TEF (% above RMR)					3·1	6·3					3·2	4·6
Accelerometer (kcal/d)			2305	340	2171	354			2269	337	2152	281
Activity diary (kcal/d)			2710	473	2724	403			2828	375	2692	460
DLW (kcal/d)					2207	916					2250	955

RMR, resting metabolic rate; TEF, thermic effect of food; DLW, doubly labelled water.

Table 4. Details regarding the routes through which energy from the almond supplement was dissipated; note these mean increments in energy expenditure were not statistically significant

	Component of energy balance	Energy dissipated (%)
Predicted body weight gain (kg)	3	
Actual body weight gained (kg)	0	
Dietary compensation (kJ)	802	74
Faecal excretion (kJ)	84	7
Energy expenditure (kJ)		
REE	297	24
TEF	13	0·01
Physical activity	−79	−6
Total energy expenditure (DLW)	180	14

TEF, thermic effect of food; DLW, doubly labelled water.

specific to peanuts, particularly as another study with almonds also did not observe changes of RMR[5]. Further research is required to clarify the effects of nuts on energy expenditure. In addition, the present study, consistent with others[9], did not find an effect of habitual almond consumption on TEF. Indeed, the measured TEF following almond consumption was lower than may have been predicted[22].

As reported previously[10], this study provides further evidence that the energy accessible from almonds may be lower than predicted. A decrease in the digestibility coefficient of the diet was noted during the almond ingestion period. This accounted for approximately 84 kJ of the 1440 kJ contained in the daily almond portion. This small amount of energy loss would have only a limited independent effect on body weight.

The 1440 kJ contained in the daily portion of almonds were almost fully offset by changes in energy intake, energy expenditure and faecal fat absorption. These indices accounted for approximately 95–98 % of the daily almond load. Given the measurement errors associated with each of these indices, this accounting must be viewed with some caution. However, it is consistent with the noted stability of body weight over the trial.

In summary, the present study demonstrates that the intake of 1440 kJ of almonds daily for 10 weeks does not promote weight gain. Indeed, other work suggests that almond consumption may aid weight loss[23], probably by increasing compliance with an energy-restricted diet. The effects of slightly lower levels of intake (i.e. 1045 kJ/d), such as proposed in the health claim for nuts, over an extended period of time remain to be evaluated. More broadly, these findings highlight the potential error of questioning the health effects of high-energy dense foods based on this property alone.

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