British Journal of Nutrition (2006), **96**, Suppl. 2, S79–S86
DOI: 10.1017/BJN20061867

Nuts, body weight and insulin resistance

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Traditionally, nuts have been considered a staple food, but because of their high energy and fat content are not considered good for body weight control or insulin sensitivity. Frequent consumption of nuts reduces the risk of coronary artery disease and type-2 diabetes and nut-enriched diets favourably alter blood lipids in normal and hypercholesterolemic individuals under controlled and free-living dietary conditions. However, whether or not frequent consumption of nuts can cause weight gain and impair insulin sensitivity is not fully understood. Review of the available data to date suggests that adding nuts to habitual diets of free-living individuals does not cause weight gain. In fact, nuts have a tendency to lower body weight and fat mass. In the context of calorie-restricted diets, adding nuts produces a more lasting and greater magnitude of weight loss among obese subjects while improving insulin sensitivity. Further studies are needed to clarify the effect of long-term (≥ year) consumption of nuts on body weight and their role in altering insulin sensitivity both in normal and type-2 diabetics. In the meantime, there is sufficient evidence to promote the inclusion of nuts as part of healthy diets.

Nuts: Body weight: Insulin resistance: Obesity: Body fat

Traditionally, nuts have been used as a staple food for providing energy, protein, essential fatty acids and a host of vitamins and minerals. At the same time, since nuts are energy dense high fat foods, they have not been considered good for controlling body weight and improving insulin sensitivity. While the research on the role of nuts in body weight control and insulin resistance is relatively new, their cardio-protective benefits have been well established. In fact, the current recommendations by the American Heart Association (Krauss *et al.* 2000) for a heart-healthy diet include nuts as one of the desirable foods to improve blood lipid profile. To date, four large epidemiological studies (Fraser *et al.* 1992; Kushi *et al.* 1996; Hu *et al.* 1998; Albert *et al.* 2002) have shown that frequent consumption of nuts lowers the risk of fatal and non-fatal coronary heart disease even after adjusting for known coronary risk factors such as age, smoking, sex, family history of hypertension, body weight, physical activity and the intake of other protective foods. More recently, the Nurses Health Study demonstrated that frequent consumption of nuts is inversely related to the risk of type-2 diabetes (Jiang *et al.* 2002). Besides population based observations, several intervention studies with different kinds of nuts in well-controlled dietary conditions (Sabaté *et al.* 1993, 2003; Curb *et al.* 2000; Rajaram *et al.* 2001; Iwamoto *et al.* 2002; Sabaté, 2003) or in free living individuals on self-selected diets (Spiller *et al.* 1992; Abbey *et al.* 1994; Colquhoun *et al.* 1996; Chisholm *et al.* 1998; Spiller *et al.* 1998; Durak *et al.* 1999; Edwards *et al.* 1999; Morgan & Clayshulte, 2000; Zambon *et al.* 2000; Almario *et al.* 2001; Hyson *et al.* 2002; Jenkins *et al.* 2002; Lovejoy *et al.* 2002; Morgan *et al.* 2002; Garg *et al.* 2003) have shown that nuts improve blood lipid parameters in both men and women with normal and hypercholesterolemia.

With the steady increase in the incidence of obesity and related chronic diseases, it becomes important to both scientists and health professionals to understand the role of nuts in body weight regulation (Garcia-Lorda *et al.* 2003; Sabaté, 2003; St-Onge, 2005) and insulin resistance. This review summarizes the data available to date by looking at three lines of evidence: (1) Data from population based studies on the association between nut intake and body weight and insulin resistance; (2) Data from nut intervention studies under controlled and free-living conditions with chronic disease risk factors as the primary outcome; and (3) Data from nut intervention trials with body weight outcome both in free-living healthy subjects and in the context of weight loss in obese individuals. Some of the potential mechanisms by which nuts may influence body weight and insulin resistance are also provided, followed by concluding remarks.

Nuts and body weight

Epidemiologic evidence

There is no epidemiological data to support the concern that nuts may cause weight gain. In Mediterranean countries where the per capita consumption of nuts is almost double that in the United States, the rate of obesity is significantly lower (Sabaté, 1993). A cross-sectional study of 800 schoolgirls in Spain found no differences in body weight in the various categories of nut intake ranging from zero to daily intake (Soriguer *et al.* 1995). In fact, Mediterranean dietary patterns, of which nuts are an integral part, have been inversely associated with BMI and obesity (Schroder *et al.* 2004). In the United States, data from the Continuing Survey of Food Intakes by Individuals (US Department of Agriculture & Agricultural Research Service, 2000) showed no association between nut intake and body mass index (BMI). In this

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survey, the nut-eater group consisted of individuals that reported consumption of tree nuts, peanuts or seeds on any of the 2 days in which the 24-hour recalls were collected. Data from the 1994–6 survey showed that young and adult nut eaters had lower BMI compared to non nut-eaters in spite of higher energy intake among nut eaters. Also, among the nut eaters, the amount of nuts and seeds in their diets did not relate to BMI (Sabaté, 2003).

Large cohort studies that reported a decrease in the risk of coronary artery disease (CAD) with frequent consumption of nuts all show an inverse or no relationship between frequency of nut intake and BMI. In the Adventist Health Study (Fraser *et al.* 1992), a statistically significant negative effect was seen between consumption of nuts and BMI in a cohort of 31 200 subjects, suggesting that those who ate nuts more often were leaner than those who ate nuts infrequently. In addition, a substantial reduction in CAD risk was observed among both lean and obese subjects in this prospective study, with a ~50 % risk reduction for individuals with either low or high BMI consuming nuts ≥5 times/wk compared to their counterparts consuming nuts ≤1 time/wk (Fig. 1). The Nurses Health Study (Hu *et al.* 1998) also observed a negative association between nut consumption and BMI among 86 000 female subjects and a similar cardio-protective effect of nuts in both obese and lean individuals. The Physicians' Health Study (Albert *et al.* 2002) did not report any association between BMI and nut intake and the BMI's by quartile of nut consumption were very similar. More recently, in a large cohort of women that were followed up for 16 years, the average weight change according to frequency of nut consumption (never to ≥5 times a week), after adjusting for potential confounders, was not significantly different (Kushi *et al.* 1996). The results of these epidemiological findings indicate an inverse or no relationship between frequent nut consumption and BMI.

Intervention studies with nuts

Nut feeding studies with CAD risk factors as primary outcome

Several nut-feeding studies have been conducted to date with the primary objective of measuring cardiovascular disease risk outcomes. These differ in methodological and dietary rigor,

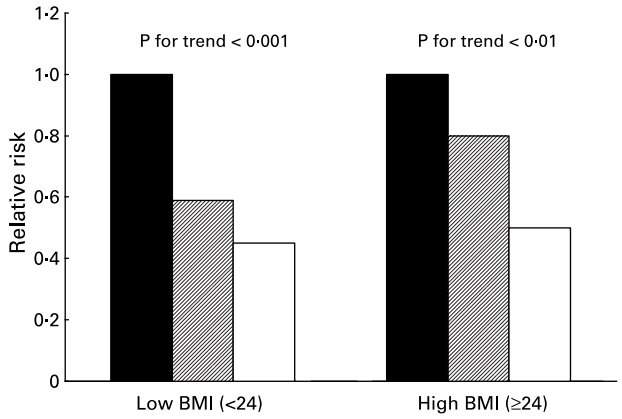

Fig. 1. Age- and sex-adjusted relative risks of coronary artery disease at three nut intakes. (■, <1 time/week; ▨, 1–4 times/week; □, ≥5 times/week) according to BMI (kg/m^2) in the Adventist Health Study. Reproduced with permission from Fraser *et al.* (1992).

macronutrient composition, especially with respect to the proportion of the different fatty acid in the diets, duration of intervention, subject characteristics and sample size. Collectively, these studies show that nuts lower cholesterol and are therefore recommended as part of heart-healthy diets. None of these studies were set up to look at differences in body weight, because by design the diets in many of the studies, especially the metabolic feeding studies, were controlled and adjusted according to body weight changes.

In the controlled crossover nut intervention studies conducted by our research group, with walnuts, pecans and almonds (Sabaté *et al.* 1993; Rajaram *et al.* 2001; Sabaté *et al.* 2003), two isoenergetic diets (nut diet and the control diet) were tested for their effects on blood lipids and lipoproteins. In these studies, subjects were weighed daily during the 2-week run-in period and once a week thereafter through the remainder of the study. Caloric intake of the subjects was adjusted to ensure that they neither gained nor lost weight. Based on our observations, when subjects were on the nut diet they tended to lose weight and some reported to being hungry. Hence, their caloric intake was increased in order to prevent weight loss. Using a similar study design, others have also shown that walnuts (Iwamoto *et al.* 2002) and macadamia nuts (Curb *et al.* 2000) do not cause changes in body weight when the total energy in the experimental diets are continually adjusted.

Most of the less controlled dietary intervention studies (Table 1) that involved self-selected diets to which a nut supplement was added, also do not report any weight gain with nut consumption. In fact, some of them observed a non-significant tendency to lower weight while subjects were on the nut supplement (Spiller *et al.* 1992; Abbey *et al.* 1994; Colquhoun *et al.* 1996; Zambon *et al.* 2000; Garg *et al.* 2003). Three of them observed no differences in body weight in spite of a total increase in energy intake in the nut diet. In one of these studies (Morgan & Clayshulte, 2000), adding a 68 g pecan supplement for 8 weeks increased the total energy intake by ~72 kJ (300 kcal) and yet both BMI and body weight remained unaffected except in two female subjects who showed a non-significant increase of less than 1 kg. In the other two studies (Almario *et al.* 2001; Morgan *et al.* 2002), adding 48 g or 64 g walnuts to the habitual diet of hypercholesterolemic patients for 6 weeks increased energy intake by ~86 kJ (360 kcal) and ~67 kJ (280 kcal), respectively, with no change in body weight. On the other hand, two studies noted an increase in body weight with nut consumption. The first one (Durak *et al.* 1999) showed that adding 1 g/kg body weight/d of hazelnuts to normocholesterolemic subjects increased the mean body weight by 0·5 kg. However, this study was not controlled and non-randomized with no report of energy intake in the nut group compared to baseline. The second (Lovejoy *et al.* 2002) was on normal weight subjects who added 100 g almonds to their daily diet for 4 weeks. An increase in body weight of 0·9 kg in men and 0·3 kg in women was noted. However, this study did not have a control group and there was an increase in energy intake by ~48 kJ when nuts were added to the diet. Overall, these studies demonstrate that when caloric intake remains the same or slightly increased by adding nuts to habitual diet in free-living conditions or replaces a portion of the calories of baseline diets under controlled conditions, there

Table 1. Body weight outcome from nut intervention studies conducted on free-living subjects

Reference	Subjects	Study design	Diet intervention	Results
Spiller *et al.* 1992	13 M, 13 F Hypercholesterolemic	Pre-post supplemental field study, dietary advice	100 g/d almonds supplemented to baseline diet	Energy intake: ↑ 81 kcal Body weight: No change
Abbey *et al.* 1994	16 M Healthy	Pre-post consecutive supplemental field study, dietary advice	84 g/d almonds or 68 g/d walnuts added to reference diet to replace 50 % fat energy, 3 weeks each diet	Energy intake: No change Body weight: No change
Colquohoun *et al.* 1996	7 M, 7 F Hypercholesterolemic	Randomized, Pre-post crossover field study, dietary advice	20 % energy of baseline diet replaced with macadamia, 4 weeks each diet	Energy intake: No change Body weight: No change
Spiller *et al.* 1998	12 M, 33 F Hypercholesterolemic	Randomized, parallel arm field study, dietary advice	100 g almonds added to baseline diet, 4 week duration	Energy intake: No change Body weight: No change
Chisholm *et al.* 1998	21 M Hypercholesterolemic	Randomized, crossover clinical study, dietary advice	20 % energy of low-fat diet replaced with walnuts, 4 weeks each diet	Energy intake: No change Body weight: No change
Edwards *et al.* 1999	4 M, 6 F Hypercholesterolemic	Randomized, crossover clinical study, dietary advice	20 % energy of habitual diet replaced with pistachio, 3 weeks each diet	Energy intake: No change Body weight: No change
Durak *et al.* 1999	18 M, 12 F, Healthy	Pre-post supplemental field study, dietary advice	1 g/kg body weight/d hazelnut added to habitual diet, 4 weeks	Energy intake: Not reported Body weight: ↑ 0·5 kg
Zambon *et al.* 2000	28 M, 27 F, Hypercholesterolemic	Randomized, crossover clinical study, dietary advice	18 % energy of habitual diet replaced with walnuts	Energy intake: No change Body weight: No change
Morgan *et al.* 2000	4 M, 15 FHealthy	Randomized, pre-post parallel arm supplemental field study	68 g pecans added to self-selected diets, 8 week duration	Energy intake: ↑ ~71 kJ in pecan group Body weight: No change
Almario *et al.* 2001	5 M, 13 F, Dyslipidemic	Pre-post consecutive supplemental field study, dietary advice	48 g walnuts added to either habitual diet or low-fat diet, 6 weeks each diet	Energy intake: ↑ ~86–90 kJ Body weight: No change
Hyson *et al.* 2002	10 M, 12 F Healthy	Randomized pre-post crossover field study, dietary advice	50 % of fat energy of habitual diet replaced with almonds or almond oil (~66 g/d), 6 weeks each diet	Energy intake: No change Body weight: No change
Morgan *et al.* 2002	42 M Hypercholesterolemic	Randomized, crossover clinical study, dietary advice	64 g walnuts added to low-fat diet, 6 weeks each diet	Energy intake: ↑ ~67 kJ Body weight: No change
Jenkins *et al.* 2002	15 M, 12 F, Hypercholesterolemic	Randomized crossover clinical study, dietary advice	73 g almonds supplement added to baseline diet, 4 weeks each diet	Energy intake: No change Body weight: No change
Lovejoy *et al.* 2002	10 M, 10 F, Healthy	Pre-post supplemental field study, dietary advice	100 g almonds/d added to habitual diet, 4 week	Energy intake: ↑ ~51 kJ Body weight: ↑ 0·9 kg (M), ↑ 0·3 kg (F)
Garg *et al.* 2003	17 M, Hypercholesterolemic	Pre-post supplemental field study, dietary advice	15 % energy of habitual diet replaced with macadamia (40–90 g/d), 4 week	Energy intake: No change Body weight: ↓ 0·52 kg

M–Males; F–Females

is no weight gain in healthy individuals and hypercholesterolemic patients. This is true either when the percent energy from fat after adding nuts remains the same (Sabaté *et al.* 1993) or is significantly increased (Rajaram *et al.* 2001; Sabaté *et al.* 2003).

Nut feeding studies with body weight as primary outcome

Three recent studies specifically investigated the effects on body weight of supplementing habitual diets with nuts in free-living subjects (Alper & Mattes, 2002; Fraser *et al.* 2002; Sabaté *et al.* 2005). One of these studies was on peanuts (Alper & Mattes, 2002), which although botanically classified as legumes, will be considered for discussion, since they are nutritionally very similar to tree nuts. In this study, 119 kJ/d (500 kcal/d) of peanut supplement was provided to fifteen adults with normal body weight. There were three phases to the study: the free-feeding phase in which subjects received peanuts without dietary guidance, an addition phase in which subjects were asked to add the peanuts to their habitual diet and a substitution phase in which they were asked to replace an equal amount of other fats in the diets for peanuts. During the substitution phase no weight gain was noted. In the free-feeding phase, subjects gained 1·0 kg during the 8-week intervention which is considerably lower than the predicted 3·6 kg based on the additional calories coming from the peanuts. Also, during the addition phase, subjects gained 0·6 kg as opposed to the 1·4 kg which was predicted. Part of this difference is attributed to the dietary compensation for a portion of the extra calories coming from peanuts. An increase in resting energy expenditure of 11 % was noted after peanut consumption despite no changes in physical activity level.

Fraser *et al.* (2002) reported that 6 mo of almond supplementation had minimal effects on body weight. Eighty-one subjects were provided with 42–70 g raw or dry-roasted almonds/d (averaging 76 kJ per person) with no instructions other than eating the almonds. Six months following nut supplementation, men gained 0·65 kg while women did not gain any significant weight (Table 2). Only lean subjects in the lowest tertile of baseline BMI gained weight and obese women (highest tertile of baseline BMI) actually lost weight during almond supplementation. The average body weight increase for subjects during almond supplementation was 0·4 kg while the predicted weight gain based on the extra calories consumed from almonds is 6·4 kg. The physical activity report showed no significant difference in frequency and vigour of activity during the two diet phases nor was there a difference in resting energy expenditure. However, food displacement was seen following almond supplementation, and approximately 54 % (by 24-hour recalls) or 78 % (from food diary) of the extra calories from almonds was displaced by a reduction of intake in other foods of the habitual diet (Jaceldo-Siegl *et al.* 2004).

Table 2. Body weight changes after 6 months almond supplementation in free-living healthy subjects (mean values)

Measurement	Control	Almonds	*P* Value*
Body weight (kg)			
Males	83·18	83·83	< 0·01
Females	69·13	69·40	0·79
Waist/hip ratio			
Males	0·943	0·955	< 0·01
Females	0·779	0·784	0·18

* Paired *t* test compares the means of the two diet periods. Adapted from Fraser *et al.* 2002.

In a recent walnut supplemented trial (Sabaté *et al.* 2005), ninety free-living men and women were assigned to either a walnut supplemented diet (28–56 g walnuts/d) or habitual diet for 6 months each in a crossover fashion. Walnuts were supplemented to the habitual diets with no specific instructions other than to eat the allotted amount. The walnut supplementation caused an increase in daily energy intake of 557 kJ (133 kcal). Although this should have led to a weight gain of 3·1 kg over the 6 months, the observed increases in body weight was 0·4 kg, in BMI 0·2 kg/m^2 and in fat mass 0·2 kg. In fact, this small increase in body weight with the walnut supplement was not significant when energy differences between the two diets were controlled for. These findings confirm that adding nuts to habitual diets does not adversely affect body weight.

Nut supplement in the context of weight loss diets

There have been two trials so far that have looked at the efficacy of nuts in the context of weight loss diets. In one study (McManus *et al.* 2001), 101 overweight subjects received either a low fat (20 % energy from fat) or a moderate fat diet (35 % energy from fat) containing several nuts, peanut butter and olive oil. Following an 18-month intervention, the moderate fat group experienced weight loss and decrease in waist circumference while the low fat group regained weight progressively. After $2\frac{1}{2}$ years, the moderate fat group still weighed significantly less than the low fat group.

In a weight reduction study (Wien *et al.* 2003), sixty-five overweight and obese individuals were randomized into either a complex carbohydrate enriched or an almond enriched low calorie diets for 24 weeks under free-living condition. Approximately 84 g/d of almonds was provided to the almond enriched diet group, which was equivalent to 39 % energy from fat as opposed to 18 % energy from fat in the carbohydrate enriched low calorie diet. As expected, both diet groups lost weight (Fig. 2), but the almond group experienced a sustained and 62 % greater weight reduction for the 24-week duration than the carbohydrate group. The almond group also had a 50 % greater decrease in waist circumference and a 62 % decrease in fat mass than the carbohydrate group. This study suggests that in the context of low caloric diets, including nuts may enhance weight loss and improve body composition indices. Additional benefits for nuts included in weight loss diets are that it may provide for variety in texture, contribute to satiety and improve long-term compliance.

Potential mechanisms

From the epidemiological studies we note an inverse relationship between nut consumption and body weight. Since nuts are

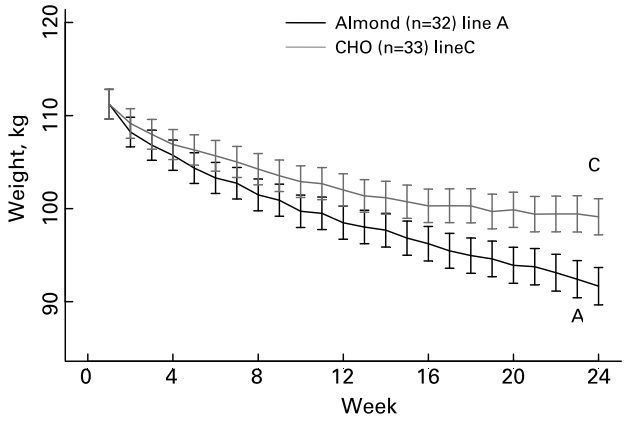

Fig. 2. Body weight loss on a calorie restricted diet with almond or carbohydrate supplement. Almond – Low calorie almond diet; CHO – Low calorie carbohydrate diet. Reproduced with permission from Wien *et al.* (2003).

a high fat, high energy food, it is possible that obese individuals avoid them while lean individuals include them more freely and this may result in the observed negative association between nut intake and body weight. It is also likely that those eating nuts frequently practice other healthy lifestyle habits, such as increased physical activity. In fact, nut consumption was associated with greater frequency of vigorous exercise among participants of the Nurses Health Study (Hu *et al.* 1998). The Physicians' Health Study also showed that men who ate nuts frequently were also more physically active (Albert *et al.* 2002). However, this was not substantiated in an intervention study where almonds were supplemented to the habitual diets of free-living subjects for 6 months. No change in exercise frequency and vigour was observed (US Department of Agriculture & Agricultural Research Service, 2000) in this study.

Several mechanisms can potentially explain why nuts do not cause weight gain. Nuts are energy dense foods with high fibre, protein and low glycaemic index, all of which are dietary factors that have been shown to increase satiety (Holt *et al.* 1995). In the 6-month almond supplemented study (Fraser *et al.* 2002), 54–78 % of the extra energy from almonds was displaced by reductions in other foods suggesting a satiety effect. Nuts are high in unsaturated fatty acids and hence may influence diet-induced thermogenesis by increasing resting energy expenditure. Both animal and human studies have shown that unsaturated fat increases fat oxidation because of higher diet-induced thermogenesis resulting in less body fat accumulation (van Marken Lichtenbelt *et al.* 1997). While peanut supplementation for 19 weeks resulted in an 11 % increase in resting energy expenditure (Alper & Mattes, 2002), daily almond supplementation for 6 months did not change resting energy expenditure (Fraser *et al.* 2002).

Given that nuts are a complex matrix of nutrients, it is likely that fatty acid availability from nuts is decreased from incomplete digestion and or absorption. An earlier study with peanuts showed that 76 g peanuts consumed for 4–6 days resulted in 17 % excretion of dietary fat in stools (Alper & Mattes, 2002). Visual inspection showed that stools contained intact portions of the nuts indicating that absorption was compromised. In an intervention feeding trial with pecans, we demonstrated that stool fat increased (25 g/d) when subjects were on the nut diet compared to the control diet (6 g/d). This represented 8 and 3 % of the dietary fat of the pecan and control diet, respectively (Zemaitis & Sabaté, 2001). A similar observation was made when the intervention food was almonds, although the percent fat loss in stools was much less (4 %) than on the pecan or peanut diets. Recently Ellis *et al.* (2004) systematically looked at the bioavailability of fatty acids from almonds by simulating chewing action and following up with a digestibility study. They demonstrated that the mechanical disruption of almond tissue or the chewing process damaged only the first layer of cells of the almond resulting in an incomplete release of the fatty acids. Further, consumption of 100–200 g/d of almonds by healthy volunteers caused a 3-fold increase in percent faecal fat excretion compared to when they ate a nut free diet. Electron microscopy revealed intact cell walls in the faecal samples that had resisted digestion and thus retained some of the intracellular fatty acids contained in the almonds.

Thus, the excretion of fat in the stools and the displacement of foods from habitual diets together may account for the lack of weight gain among nut eaters. Future studies need to further explore the mechanism(s) by which nuts prevent weight gain or in some cases induce weight loss.

Nuts and insulin resistance/type-2 diabetes

Epidemiological evidence

A prospective cohort study of women (Jiang *et al.* 2002) found that the frequency of nut consumption had a substantial and highly significant inverse association with risk of type-2 diabetes. The reduction in risk of developing type-2 diabetes was 30 % lower in those consuming nuts five or more times per week and 20 % lower in those consuming nuts 1–4 times a week compared to those that almost never ate nuts (Table 3). The inverse association persisted even after adjusting for other known risk factors including age, obesity, physical activity, smoking, family history and other dietary factors.

The protective effect of nut consumption on incidence of type-2 diabetes was consistent among various subgroups in women. The frequency of nut consumption was significantly and inversely related to the risk of type-2 diabetes in both multivitamin users and non-users, those above or below mean body mass index, and in those that consumed a high or low glycaemic load. Similar findings were also noted for the consumption of peanut butter. However, whether a similar relationship will be observed in men remains to be determined in future studies.

To date, this is the only study that has looked at the association between nut intake and risk of diabetes although previously, unsaturated fat intake has been shown to reduce the risk of type-2 diabetes and improve glycaemic control in diabetic patients (Hu *et al.* 2001).

Intervention studies

In a randomized, double-blind crossover study (Lovejoy *et al.* 2002), 34 men and women with type-2 diabetes were assigned

Table 3. Relative risks of type 2 diabetes in women according to frequency of nut consumption

	Frequency of nut consumption (28 g serving)				
	Never/almost never	<Once/week	1–4 times /week	≥5 times/week	*P* for trend
Cases, no.	1314	1133	644	115	–
Person-years	441.007	466·464	309·608	66·468	–
Age-adjusted RR (95 % CI)	1·00	0·82 (0·76–0·89)	0·69 (0·63–0·76)	0·55 (0·45–0·66)	<0·001
Age- and BMI-adjusted RR (95 % CI)	1·00	0·91 (0·84–0·99)	0·83 (0·75–0·91)	0·74 (0·61–0·89)	<0·001
Multivariate RR (95 % CI)*	1·00	0·92 (0·85–1·00)	0·84 (0·76–0·93)	0·73 (0·60–0·89)	<0·001

RR, Relative risk; BMI, Body mass index; CI, Confidence interval.
* Relative risk was adjusted for age (5-year categories), BMI (<21, 21·0–22·9, 23·0–24·9, 25·0–27·9, 28·0–29·9, 30·0–34·9, ≥35, and missing information), family history of diabetes in a first-degree relative (yes or no), moderate/vigorous exercise (<1, 1, 2–3, 4–6, ≥7 h/week), cigarette smoking (never, past, or current smoking of 1–14, 15–24 or ≥25 cigarettes/d), alcohol consumption (0, 0·1–5·0, 5·1–15·0 or >15 g/d), and total energy intake. Reproduced with permission from Jiang *et al.* 2002.

to one of four different diets: high fat-high almond diet, low fat-high almond diet, high fat-control diet and low fat-control diet for 4 weeks each. The fat source in the low fat and high fat control diets was olive or canola oil. The almond enriched diets used 10 % of energy from almonds (~85 g almonds/619 kJ diet). In this study no significant effect of fat amount or source was seen on plasma glucose and insulin levels during a 2-h oral glucose tolerance test. The almond enriched high fat diet had a favourable effect on serum total cholesterol without adversely affecting glycaemia. It is likely that nut intervention longer than 4 weeks and greater than 10 % of total energy intake is required to modify insulin sensitivity and glycaemic control. Similarly, 30 g a day of walnuts added to the low-fat diet of type-2 diabetic patients improved blood lipid profiles by specifically increasing HDL-C/total-C ratio without adversely affecting glycosylated haemoglobin levels and body weight (Tapsell *et al.* 2004).

More recently, sixty-five overweight subjects (37 women, 28 men) completed a randomized dietary intervention trial for 24 weeks to determine the efficacy of an almond-based hypocaloric diet on dyslipidemia, insulin resistance and weight changes (Wien *et al.* 2003). Seventy percent of the participants had type-2 diabetes and the remainder had insulin resistant syndrome. Upon being selected, all participants followed a 2-week run-in period with no intervention apart from a multivitamin/mineral supplement. At the end of the 2 weeks, participants were randomized based on gender and presence or absence of type-2 diabetes into a formula-based low caloric diet supplemented with either almonds (84 g/d per 238 kJ) or complex carbohydrates equivalent in calories for 24 weeks.

There was a 54 % reduction in fasting insulin levels in the almond supplemented group compared to the carbohydrate diet group (Table 4). Insulin resistance as measured by the homeostasis model analysis (HOMA-IR) was decreased significantly in both diet groups, but an improved beta cell function was only observed on the almond diet. Among subjects with type-2 diabetes, diabetes medication reduction was either sustained or reduced further in 96 % of the almond supplemented group compared to only 50 % among the carbohydrate supplemented group. There was also a significant increase in ketosis in the almond group indicating a greater fat oxidation which probably accounts for the greater magnitude of weight loss seen with almond supplementation. Thus, there is some evidence to suggest that almonds, and perhaps other nuts, may have a favourable effect on insulin sensitivity. More studies are needed to support these observations.

Potential mechanism of action

The fatty acid composition of nuts may play a role in modifying insulin resistance, and therefore, the risk for type-2 diabetes. In fact, specific types of fatty acids are better predictors of the risk of type-2 diabetes than total dietary fat intake (Hu *et al.* 2001). A higher intake of n-3 PUFA is associated with lower risk of type-2 diabetes whereas a higher intake of saturated and trans fatty acid intake adversely affects glycaemic control thereby increasing the risk of type-2 diabetes (Hu *et al.* 2001; Vessby *et al.* 2001). The mechanisms by which specific fatty acids affect insulin sensitivity are not clearly understood. However, it has been shown that the fatty acid composition of the phospholipids in the skeletal muscle cell membranes is directly related to insulin sensitivity in humans. A specific fatty acid in cell membranes could influence insulin action through altering insulin receptor binding or affinity and influencing ion permeability and cell signalling (Storlien *et al.* 1996). Monounsaturated fatty acids are also known to improve beta cell efficiency through enhanced

Table 4. Body weight and insulin sensitivity measures on two weight loss diets (least square mean values with their standard error)

Measurement	Week	Almond-LCD	Carbohydrate-LCD
Weight (kg)	0	111·3 (0·82)	111·2 (0·82)***
	24	201·7 (2·3)	218·1 (2·2)
BMI (kg/m^2)	0	38·3 (0·3)	38·4 (0·3)***
	24	31·6 (0·3)	34·2 (0·3)
Fat mass (kg)	0	46·6 (1·0)	46·4 (1·0)*
	24	32·5 (1·1)	37·3 (1·1)
Fat free mass (kg)	0	62·7 (0·63)	62·4 (0·63)
	24	57·6 (0·72)	60·0 (0·72)
Insulin (μU/ml)	0	46 (5)	47 (5)
	24	21 (5)	32 (5)
Glucose (mg/dL)	0	152 (11)	152 (11)
	24	128 (11)	127 (11)
HOMA-IR	0	20 (4)	17 (4)**
	24	7 (2)	11(2)
Ketone (mmol/L)	0	0·10 (0·04)	0·11 (0·03)**
	24	0·36 (0·05)	0·11 (0·05)

LCD, Low calorie diet. HOMA-IR, Homeostasis model analysis to measure insulin sensitivity. ***($P<0·0001$), ** ($P<0·02$) and * ($P<0·05$) Statistically significant differences between groups. Adapted and reproduced with permission from Wien *et al.* 2003.

Table 5. Nutritional composition (per 100 g edible portion) of some selected tree nuts*

Nutrient	Walnut	Almond	Pecan	Macadamia	Pistachio	Cashew	Hazelnut
Energy (kcal)	654	578	691	718	557	566	628
Protein (g)	15·2	21·3	9·2	7·9	20·6	18·2	15·0
Carbohydrate (g)	13·7	19·7	13·9	13·8	28·0	27·1	16·7
Fat (g)	65·	50·6	71·9	75·8	44·4	46·9	60·8
Saturated (g)	6·1	3·9	6·2	12·1	5·4	8·3	4·5
MUFA (g)	8·9	32·2	40·8	58·9	23·3	25·5	45·7
PUFA (g)	47·2	12·2	21·6	1·5	13·5	8·4	7·9
Fiber (g)	6·7	11·8	9·6	8·6	10·3	3·3	9·7
Magnesium (mg)	158	275	121	130	121	292	163
∝ -tocopherol (mg)	2·9[a]	26·2	4·1[a]	0·6	4·6	1·5	15·2
Lysine: Arginine	0·2	0·3	0·3	0·4	0·6	0·5	0·2
Copper (mg)	1·0	0·9	1·2	0·3	1·2	2·2	1·5

* Source: United States Department of Agriculture Nutrient Database for Standard Reference, Release August 2005. [a]γ-tocopherol in walnuts is 17·2 mg, in pecans is 19·1 mg.

secretion of glucagon-like peptide-1 that is known to help in regulating postprandial glucose clearance and insulin sensitivity (Rocca *et al.* 2001).

In addition to the type of fatty acid in nuts, the non-lipid components of nuts (Table 5) may also contribute towards lowering the risk of type-2 diabetes. Several prospective studies (Paolisso *et al.* 1990; Salmeron *et al.* 1997) have shown that the risk of type-2 diabetes is lowered with higher intakes of fibre and magnesium and the inclusion of low glycaemic index foods. As reviewed by Salas-Salvadó *et al.* (2006) in this supplement, nuts provide a significant amount of fibre (~4–12 g/100 g). They are also considered a good source of magnesium (~120–300 mg/100 g), and magnesium intake is inversely associated with diabetes risk (Lopez-Ridaura *et al.* 2004). Being a whole food, it is reasonable to expect synergistic interaction between many of the nutrients and non-nutrients in nuts as they work to influence insulin resistance and, therefore, the risk of type-2 diabetes.

Conclusions

Although nuts are high in fat, the available data demonstrate that adding nuts to habitual diets of free-living individuals does not cause weight gain. In fact, there may be a tendency for nuts to decrease fat mass. As expected, isoenergetic replacement of nuts for other foods does not result in changes in body weight. In the context of weight loss diets, adding nuts to a calorie-restricted diet in obese individuals produces a greater magnitude of weight loss. Studies over 1 year are required to prove the safety of consuming nuts under free-living conditions in the long-term.

New research is also pointing to a protective role for nuts in reducing the risk of type-2 diabetes and in improving insulin sensitivity in obese diabetics under weight loss situation. Clearly more research is needed in determining whether nuts do influence insulin sensitivity in both normal and type-2 diabetic individuals. Future studies on the role of nuts on body weight regulation and insulin sensitivity should attempt to understand the mechanism(s) by which nuts bring out the desirable effects. In the meanwhile, there is sufficient evidence to continue promoting the inclusion of nuts as part of healthy diets.

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