Impact of Peanuts and Tree Nuts on Body Weight and Healthy Weight Loss in Adults[1,2]

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Abstract

Nuts (ground and tree) are rich sources of multiple nutrients and their consumption is associated with health benefits, including reduced cardiovascular disease risk. This has prompted recommendations to increase their consumption. However, they are also high in fat (albeit largely unsaturated) and are energy dense. The associations between these properties, positive energy balance, and body weight raise questions about such recommendations. This issue is addressed through a review of the literature pertaining to the association between nut consumption and energy balance. Epidemiological studies document an inverse association between the frequency of nut consumption and BMI. Clinical trials reveal little or no weight change with inclusion of various types of nuts in the diet over 1–6 mo. Mechanistic studies indicate this is largely attributable to the high satiety property of nuts, leading to compensatory responses that account for 65–75% of the energy they provide. Limited data suggest chronic consumption is associated with elevated resting energy expenditure resulting in dissipation of another portion of the energy they provide. Additionally, due to poor bioaccessibility, there is limited efficiency of energy absorption from nuts. Collectively, these mechanisms offset much of the energy provided by nuts. The few trials contrasting weight loss through regimens that include or exclude nuts indicate improved compliance and greater weight loss when nuts are permitted. This consistent literature suggests nuts may be included in the diet, in moderation, to enhance palatability and nutrient quality without posing a threat for weight gain. J. Nutr. 138: 1741S–1745S, 2008.

Introduction

There is increasing evidence of health benefits associated with ingestion of specific foods and food components. This has prompted recommendations to increase their consumption. However, given the high prevalence of overweight/obesity in the United States and globally (1), it is essential to communicate messages related to increasing intake of energy-yielding foods responsibly, i.e. in a manner that does not promote positive energy balance. This review focuses on ground nuts (i.e. peanuts, which are legumes) and tree nuts, which will hereafter be referred to as "nuts" unless otherwise stated. There is a qualified health claim stating consumption of most nuts may reduce the risk of heart disease. As interest in incorporating nuts into the diet grows, it is important that consumers understand how to include them into a healthy diet without promoting weight gain. They are a high-fat, energy-dense food and are therefore a potential threat for contributing to positive energy balance. However, evidence is accumulating to question this view and to update recommendations on nut use under conditions of weight loss and maintenance. The purpose of this article is to review evidence on nut consumption and body weight.

Based on the evidence from epidemiological and controlled clinical studies, nut consumption is not associated with higher body weight (2–7). In fact, the epidemiological evidence indicates consistently that nut consumers have a lower BMI than nonconsumers. With respect to clinical studies, the evidence is nearly uniform that their inclusion in the diet leads to little or no weight gain (8–12). Moreover, adherence to a moderate-fat, weight loss diet vs. a low-fat weight loss diet in free-living subjects is better and the nutrient profile of the moderate-fat diet is superior (13). Moderate-fat diets that contain nuts elicit a more

favorable lipid and lipoprotein profile after maintenance of weight loss as well (14). The evidence supporting these observations is outlined below.

Epidemiologic studies of nut consumption and BMI
In 1992, Fraser et al. (2) reported the results of the Adventist Health Study, which showed beneficial associations between nut consumption and coronary heart disease risk. The authors noted, "In our data set, we found a statistically significant negative association between consumption of nuts and the Quetelet index of obesity, showing that the higher nut consumers in this population were less obese." Since then, other U.S. epidemiological studies have reported similar results. In the Nurses Health Study, Hu et al. (3) reported a negative association between nut consumption and BMI. Sabate (6) also reported that adult nut eaters had a lower BMI than non-nut eaters using data from the Continuing Survey of Food Intakes by Individuals, 1994–1996. Likewise, in a study conducted by Griel et al. (7) using the same database, female peanut users had a significantly lower BMI than nonusers and for men, there was a trend for a lower BMI in peanut users. BMI was lower in children (both males and females) who were peanut consumers relative to nonusers. In the Iowa Women's Health Study (4), weight change over 16 y was not related to frequency of nut consumption. Albert et al. (5) reported no association between nut consumption and BMI in the Physicians' Health Study. No observational study has reported a higher BMI in nut and peanut consumers than in low consumers/nonusers.

Clinical studies of nut and peanut consumption and body weight
Controlled clinical trials have also documented a limited impact of nut consumption on body weight (8,9). Most had cardiovascular disease risk factors rather than body weight as primary end points, but the findings are strongly supported by the few studies that expressly focused on energy balance. The common design feature of these trials was that a portion of nuts was provided without dietary or other lifestyle advice, other than that the nuts were to be consumed daily. In 1 trial, 15 healthy, normal-weight adults ingested a mean of 2113 ± 494 kJ/d of peanuts for 8 wk (9). With no compensation, the predicted weight gain was 3.6 kg. The measured change was only 1 kg, ~28% of that predicted. Among a Ghanaian sample tested under the same conditions, weight gain was ~0.27 kg, only 8% of a predicted gain of ~3.4 kg. Another study provided a mean of 965 kJ (12% of calculated energy need) of walnuts to the diets of 90 healthy adults for 6 mo (10). The unadjusted weight gain was predicted to be 5.3 kg, but the measured increment was 0.4 ± 0.2 kg. Thus, the gain was ~7% of that predicted. Two trials have been reported using almonds. In one, 81 healthy adults were provided a mean of 1339 kJ (15% of calculated daily energy needs) of raw or dry-roasted almonds for 6 mo. The expected uncompensated weight gain was 6.4 kg (11). The observed values were 0.65 kg for males and 0.14 kg for females, indicating the change was only ~2–10% of that predicted. The most recent trial entailed providing 20 healthy, overweight, adult women with 1440 kJ/d of almonds for 1 10-wk period or no almonds for another 10 wk in a randomized, crossover design trial with a 3-wk washout period between the experimental periods (12). The theoretical weight gain was 3.4 kg, but the participant weights were 70.4 ± 9.0 kg at the start of the almond arm and 70.3 ± 9.3 kg at the end, indicating no change.

In only 1 study did body weight increase with increased nut consumption (15). In this trial, 20 normal weight (BMI, 23 ± 0.3 kg/m²) adults (mean age, 25 ± 1 y) were instructed to consume an extremely large portion (100 g) of almonds daily for 4 wk. This was accomplished by providing whole almonds or almonds in a variety of pastries. Subjects were advised of the energy content of their almond regimens and were asked to reduce energy consumed from other sources by an equivalent amount to maintain a steady diet. Weight increased slightly (0.9 kg in males and 0.3 kg in females) but significantly after 4 wk of the almond-enriched diet (P = 0.006). However, the almond-enriched diet also produced favorable reductions in total and LDL cholesterol (P = 0.0001). HDL cholesterol was lowered (P = 0.0002) with greater reductions in women than men (P = 0.0001). The ratios of total cholesterol:HDL cholesterol (P = 0.003) and LDL cholesterol: HDL cholesterol both decreased (P = 0.0005) and were differentially based on gender (P = 0.006 for women; P = 0.01 for men) after consumption of the almond-enriched diet. Insulin sensitivity and glucose response did not differ.

These clinical trials reveal a limited impact of nut consumption on body weight. To date, the interventions conducted have provided greater amounts than the recommended daily serving of nuts (i.e. 42 g). Future studies should explore the effects of smaller portions over longer time periods.

Clinical studies of nut consumption and weight loss
Numerous intervention studies have contrasted the effects of incorporating nuts in a weight management regimen on body weight. In 1 trial (16), an assessment was made of including almonds vs. complex carbohydrates in a weight reduction program in 65 adults. Thirty-two participants (mean age, 53 ± 2 y; mean BMI, 39 ± 1 kg/m²) were randomized to a formula-based diet enriched with almonds (84 g/d). Thirty-three adults (mean age, 57 ± 2 y; BMI, 37 ± 1 kg/m²) were randomized to a group allowed to self-select complex carbohydrates (53% of total energy) and safflower oil. Both diets were considered low energy (~4184 kJ/d) and were matched for protein (29% of energy), cholesterol (4%), and saturated fat (3%). The study lasted 24 wk with a 2-wk run-in period where no intervention was administered. Participants were advised to begin a walking program (20–30 min 3–5 times per week) starting in wk 5 of the dietary intervention. The attrition rate was 20%. The almond group had more favorable reductions in body weight and BMI (−18 vs. −11%; P < 0.0001), waist circumference (−14 vs. −9%), and fat mass (−30 vs. −20%) (P < 0.05) at the end of the 24 wk. HDL cholesterol increased in the carbohydrate group but not in the almond group (P < 0.05). Although total cholesterol, triglycerides, LDL cholesterol, and the LDL cholesterol:HDL cholesterol ratio decreased significantly over time, no differences between diet conditions were observed.

In another study (14), participants were fed a moderate-fat weight loss diet that provided 33% of energy from fat and purposefully included peanuts or a low-fat weight loss diet that provided 16% of energy from fat without peanuts. Both diets were designed to elicit ~1 kg/wk of weight loss. After 6 wk of weight loss, participants were fed the same diet designed to maintain body weight for 4 wk. As designed, similar weight loss was achieved with the 2 diets (7.2 kg for the moderate-fat group and 6.5 kg for the low-fat group at the end of the weight loss phase and ~8 kg for both groups at the end of the maintenance phase). Both diets reduced total and LDL cholesterol, but the low-fat diet decreased HDL cholesterol and increased triglycerides after maintenance of weight loss, unlike the moderate-fat diet. Thus, the moderate-fat weight loss diet that contained peanuts was preferable in terms of the effects on lipid cardiovascular disease risk factors.

Another trial (13) revealed a moderate-fat diet (35% of energy from fat) including nuts resulted in superior adherence and greater weight loss (4.1 vs. 2.9 kg) over 18 mo. Fifty-four percent of the subjects on the moderate-fat diet remained in the study, whereas only 20% of the subjects consuming the low-fat diet (20% of energy from fat) did so. Further, participants on the moderate-fat diet consumed 2 more servings per day of vegetables and more protein, fiber, and unsaturated fatty acids compared with the low-fat diet group. Consistent with these findings, a superior nutrient intake profile was noted among subjects consuming peanuts and peanut products compared with non-users based on 1994–1996 Continuing Survey of Food Intakes by Individuals data (7). Peanut users had higher intakes of vitamin A, vitamin E, folate, calcium, magnesium, zinc, iron, and dietary fiber, and a lower intake of saturated fat and cholesterol.

Taken together, these data indicate nuts can be included in a weight loss diet without compromising efficacy. Indeed, under certain circumstances, their addition may augment weight loss and improve diet quality.

Mechanisms for the less-than-predicted effects of nut consumption on body weight

Substantiation of the counterintuitive observations of little or no change of body weight with nut or peanut consumption requires identification of the mechanisms responsible. They would be quite uninteresting if simply due to poor dietary compliance, methodological artifacts, or changes of physical activity. However, these explanations are not viable. Based on changes of erythrocyte membrane fatty acid profiles (9–11) or plasma α-tocopherol concentration changes (12), the nuts were ingested during the trials. Participants were free-living, thereby minimizing novelty or context effects, and physical activity was not increased (9–12). However, accumulating evidence of other responses now provides a mechanistic basis for these findings.

Satiety. First, several lines of evidence demonstrate that nuts have high satiety properties. The provision of fixed energy loads of nuts (peanuts, almonds, and chestnuts have been tested to date) leads to sharp reductions in self-reported hunger on questionnaires (17). Loads matched on weight and volume to peanuts, but low in energy, were not as effective at suppressing hunger, indicating these are not the relevant properties. Foods varying in sensory attributes, energy density, and macronutrient composition were comparably effective, indicating these are not the key satiation determinants (17,18). Comparisons of peanut, olive, and safflower oils did not reveal marked differences (19), so fatty acid saturation does not exert a dominating role. The low glycemic index of nuts has also been proposed as a mechanism by which they modulate appetite (8). This assumes plasma insulin or glucose causally influence appetite. However, euglycemic clamp studies did not confirm this (20) and high concentrations of insulin in the brain suppress feeding (21). At this time, no single attribute accounts for the high, self-reported satiety value of nuts.

Dietary compensation. An alternate method for assessing satiety is to monitor dietary compensation, the spontaneous reduction of energy intake subsequent to nut consumption, relative to a no-load or alternate load condition. Reported values range from ~54 to >100% for nuts (9–12,15,17,22–24). That is, the majority, probably 65–75%, of the energy reportedly provided by nuts is offset by lower energy intake at later eating events. Again, no single component of nuts appears responsible for this effect. The fatty acid profile is often mentioned but does not appear to be responsible, because dietary compensation to walnuts, almonds,

and peanuts is comparable and they vary markedly in proportions of monounsaturated fatty acids and PUFA (25). In a 42-g portion, walnuts contain 19.5 g of PUFA, whereas almonds and peanuts contain 5.3 and 7.0 g, respectively. In contrast, almonds contain 13.7 g of monounsaturated fatty acid, whereas the content of walnuts contain 3.8 g and peanuts contain 10.4 g. Furthermore, energy compensation for chronic feeding of peanut and other oils is weaker than with whole nuts (by ~45–50%) (19).

Efficiency of energy absorption. Second, the efficiency of absorption of energy from nuts is limited. Studies exploring this property have used discrepant and rather extreme experimental conditions. However, they reveal consistently elevated fecal fat loss with nut consumption. In 1 early trial (26), peanuts provided 95% of the energy from fat to 10 participants adhering to a vegetarian diet for 6 d. With whole nuts, 17.8% of the lipid load was lost in the stool, whereas values were 7.0% and 4.5% for peanut butter and peanut oil loads, respectively. A more recent trial with 63 healthy adults compared fecal fat excretion to customary diets containing 70 g/d of whole peanuts, peanut butter, peanut flour, or peanut oil (27). Fecal fat content was significantly higher when whole peanuts were consumed (5.22 ± 0.59%) relative to the other forms (oil, 3.07 ± 0.36%; butter, 3.11 ± 0.31%; and flour, 3.75 ± 0.40%). Within-group comparisons revealed a significant increase in fecal fat only with whole peanuts. This corresponded to an energy loss of ~155 kJ/d or 9.4% of the load or 12% of the peanut lipid. In a trial where the fat content of the diet of 6 individuals was increased from 30% of energy to 43% of energy by the addition of pecans for 4 wk, the percentage of fecal fat increased from 2.9 to 8.3% (28). Several trials have been conducted with almonds. One short-term study progressively provided 3 individuals 100, 150, and 200 g/d over 3 d and observed an increase in fecal fat content from 3.5 ± 0.9% in the control diet to 9.9% ± 1.1% in the combined supplemented samples (29). A 10-wk trial providing 1255 kJ/d resulted in an increased fecal loss of ~6.7% of the almond energy (12). Several trials have demonstrated a dose-response relationship. One provided a 1770-kJ supplement to 27 hyperlipidemic adults for 3 1-mo periods. It contained 50–100 g of almonds, one-half the amount, or no almonds. An increase in fecal energy excretion of 247 ± 63 kJ/d and 113 ± 46 kJ/d above baseline was noted (30). In the other reported trial, fecal fat content following diets containing 0, 10, and 20% of total energy from almonds was 1.7, 3.2, and 4.1%, respectively (31). Although the data do not permit accurate calculation of the energy loss due to limited bioaccessibility, a working estimate may be 10–20% of the energy from the nuts. This inefficiency stems from resistance of the parenchymal cell walls of nuts to microbial and enzymatic degradation. Thus, cells that are not ruptured during mastication may pass through the gastrointestinal tract without releasing the oil they contain (29). This is supported by data demonstrating greater energy loss from whole nuts compared with their butter form (26,27), a higher energy requirement to maintain body weight during nut consumption (28), as well as microstructural analyses of fecal samples (29). One trial found that the energy from peanuts was substituted for other fat energy in the diet without weight loss (9). However, participants were free living, so the accuracy of the substitution cannot be determined.

Because lipid is the primary energy source in nuts, work on bioaccessibility has focused on this nutrient. However, the resistance of the cell walls of nuts to degradation in the gastrointestinal tract would also limit the bioaccessibility of other nutrients they contain, including protein, vitamins, minerals, and phytochemicals (32,33). A recent study with almonds

documented protein and vitamin E losses that paralleled those for lipid (34). With simulated gastric and duodenal digestion, the losses of lipid, protein, and vitamin E were 9.9 ± 0.71%, 13.9 ± 0.97% and 16.5 ± 0.06%, respectively, for 2-mm cubes of natural almonds and 34.0 ± 0.24%, 39.6 ± 0.23%, and 39.2 ± 0.32% for finely ground almonds. To the extent that these other nutrients contribute energy or influence metabolic processes, their limited bioaccessibility may also contribute to the less-than-predicted impact of nut ingestion on energy balance.

Increased energy expenditure. Third, chronic consumption of nuts may augment energy expenditure. This has not been reported for the thermogenic effect of feeding (9–12), nor is there evidence that nut consumption promotes physical activity (9–11). However, chronic consumption of peanuts has been associated with elevated resting energy expenditure (REE). In 1 trial, consumption of 2092 kJ/d of peanuts led to an 11% increment in REE over 19 wk (9). In another trial, peanut consumption for 8 wk was associated with a significant 5% increment in REE but only among obese individuals (the increment was 11% among males). This was not observed in 1 trial with almonds (12). Another study with almonds reported a ~209-kJ/d increment in REE that was corroborated by doubly-labeled water measurement (12). However, due to limited power (*n* = 20), neither of these changes, which would have accounted for roughly 14% of the almond energy, was significant. An explanation for the rise of REE is not obvious, but because the diets of nut consumers tend to reflect the nutrient contribution of the nuts, their high unsaturated fatty acid composition and protein concentration may be involved. Unsaturated fats are oxidized more readily than saturated fats (35,36) and protein has a high thermogenic effect (37). However, given the relatively short intervention period, in both instances, an effect on the thermogenic effect of feeding might have been expected rather than increased REE. Whether the data on peanuts and energy expenditure are valid and extend to other types of nuts remains to be verified. Assuming an REE of 6694 kJ/d, an increase of 5% in the REE with a peanut load of 42 g would amount to ~30% of the energy from the nuts.

Although there are insufficient data to definitively support each of these mechanisms for dissipation of the energy provided by nuts or for the estimated absolute values of their contributions, collectively they provide a basis for explaining the lack of effect of nut consumption on BMI identified in the epidemiological and clinical literature. Based on the totality of the literature, the current best estimates are that 55–75% of the energy contributed by nuts is offset by dietary compensation, another 10–15% by fecal loss, and an additional, less well-established estimate of 10% via increased energy expenditure (**Fig. 1**).

There are claims that energy-dense foods are especially problematic for weight loss and maintenance. Nuts are among the most energy-dense foods consumed, yet the literature consistently documents little impact of their ingestion on body weight. These data suggest that each food must be evaluated objectively for its impact on body weight and total diet quality to ensure recommendations about its use is sound and empirically based. Additional studies should be undertaken to quantify the contributions of the mechanisms involved in nut consumption and energy balance under controlled and free-living conditions with a range of intake levels.

Other articles in this supplement include references (38–43).

FIGURE 1 Estimated effects of dietary compensation, inefficient energy absorption, and increased energy expenditure on the percentage of energy contributed by nut ingestion. Dietary compensation is the percentage of energy contributed by the nut offset by a reduction of free-feeding energy intake. Fecal loss is the increment in fecal energy loss attributable to nut consumption. Energy expenditure is the increment of daily energy expenditure attributable to nut consumption calculated based on reported effects on REE measured by indirect calorimetry or total energy expenditure determined by doubly-labeled water. Values are only crude estimates, as many are based on assumptions such as mean values of energy intake are accurate and reflect individual behavior; energy loss is only due to fecal fat loss; and REE constitutes 75% of total daily energy expenditure.

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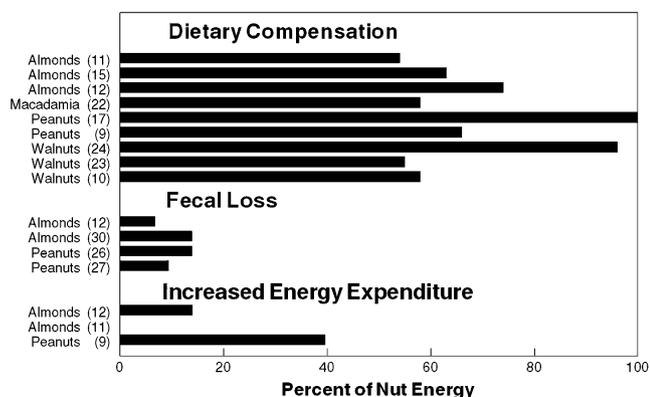15. Lovejoy JC, Most MM, Lefevre M, Greenway FL, Rood JC. Effect of diets enriched in almonds on insulin action and serum lipids in adults with normal glucose tolerance or type 2 diabetes. Am J Clin Nutr. 2002;76:1000–6.

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