A review of the effects of nuts on appetite, food intake, metabolism, and body weight[1–3]

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ABSTRACT

Tree nuts and peanuts are good sources of many nutrients and antioxidants, but they are also energy dense. The latter often limits intake because of concerns about their possible contribution to positive energy balance. However, evidence to date suggests that nuts are not associated with predicted weight gain. This is largely due to their high satiety value, leading to strong compensatory dietary responses, inefficiency in absorption of the energy they contain, a possible increment in resting energy expenditure, and an augmentation of fat oxidation. Preliminary evidence suggests that these properties are especially evident when they are consumed as snacks. *Am J Clin Nutr* 2014;100(suppl):412S–22S.

INTRODUCTION

The long-standing pillars of nutrition advice to optimize health are to practice balance, moderation, and variety. If followed, no food must be excluded from the diet and each food can make some useful contribution, the value of which is determined by the health status and needs of the individual. In an era in which positive energy balance has dominated health concerns in Western nations and is a growing problem globally, high-fat, energy-dense foods have often been identified as especially problematic. Nuts are an example, and one in which the science does not support this perspective. This review focuses on the role of nut consumption on appetite, energy intake, energy metabolism, and body weight. Recent studies that report the effects of nuts on various aspects of human energy balance are summarized in **Table 1**.

NUTS IN THE CONTEXT OF A WHOLE DIET

Nuts contribute to energy and nutrient intake directly and indirectly via multiple mechanisms. First, nuts themselves are rich sources of energy, various nutrients (eg, tocopherols, magnesium, potassium), and antioxidants (1, 2). Each form of nut has its own inherent sensory profile that is more or less appealing to individual consumers and so will influence their ingestive decisions. However, the sensory profile of the raw nut is commonly modified through processing. Roasting and frying darken the color, increase brittleness, and develop new flavor compounds (3–5). Changes in physical properties are of particular importance for the acceptability of nuts (6). A wide array of flavor compounds (eg, salt, sugar, cinnamon, and capsaicin) is also added directly to the surface of nuts to enhance their appeal. Broadly, such modifications increase sensory variety and, by ameliorating monotony effects (7), may facilitate regular nut consumption and intake of the nutrients they contain. Sensory properties are among the strongest determinants of ingestive decisions (8, 9).

There are also indirect effects of nut consumption on total energy and nutrient intake. The sensory, nutrient, and/or physical properties of nuts alter gut hormone secretion (10, 11) and appetitive responses by consumers (12). In addition, nuts are frequently incorporated into the matrix of other foods (eg, confections, baked goods, ice cream), changing the flavor profile of both and creating a unique new unified sensory stimulus (13) that may guide intake of that item or influence the acceptability and selection of other items in the broader diet (14, 15). Whether this promotes greater energy intake remains to be determined.

APPETITE AND ENERGY INTAKE

With a few exceptions (16, 17), human feeding trials have shown that nut ingestion moderates appetite postprandially. Specifically, the inclusion of almonds and peanuts suppresses hunger (18, 19) and desire to eat (19) and increases fullness ratings after ingestion (17). Daily consumption of peanuts for 4 d also increased fasting satiety and fullness levels (20). These are important properties in weight management because a postprandial reduction in hunger may prolong meal latency; a decrease in desire to eat may prevent eating in the absence of hunger, and higher fasting satiety and fullness levels may translate into smaller meal sizes.

The satiating effects of nuts depend on 2 important factors. First, the form of nuts exerts differential effects on appetitive sensations. Smaller hunger suppression and greater hunger rebound (180 min postingestion) were observed when peanuts were consumed in the form of butter compared with whole nuts (18). However, whole almonds reportedly induce fullness levels comparable to those of almond butter (17). Second, the timing of nut consumption can affect appetite. The consumption of almonds together with a meal does little to augment the

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TABLE 1
A summary of recent trials on human energy balance using a variety of nuts[1]

Type of nut and first author (ref)	Year	Design	Length	*n*	Treatments	EI	Appetite	Fat absorption	Diet compensation	EE	Fat oxidation	Weight	Body fat
Almonds													
Kirkmeyer (18)	2000	Randomized, crossover	180 min and 24 h	24	500 kcal almonds vs no load condition and preloads matched on weight and volume	—	↓	—	57%	—	—	—	—
Fraser (31)	2002	Randomized, crossover	6 mo	81	320 kcal almonds/d vs no almonds	↑	—	—	54–78%	↔	↔	↔	—
Jenkins (32)	2002	Randomized, crossover	4 wk	27	22.2%E from almonds or 11.1%E from almonds + 11.1%E from muffins vs 22.2%E from muffins	↑	—	—	—	—	—	↔	—
Wien (115)	2003	RCT weight loss	24 wk	65	84 g almonds/d vs isocaloric complex carbs	—	—	—	—	—	—	↓	↓
Hollis (33)	2007	Randomized, crossover	10 wk	20	344 kcal almonds/d vs no almonds	↓	—	—	74%	↔	—	↔	↔
Mori (17)	2011	Randomized, crossover	490 min	14	43 g almonds vs no almonds, almond flour, and almond oil groups, all matched on 75 g available carbohydrate in breakfast meals	—	↑ Fullness	—	—	—	—	—	—
Novotny (61)	2012	Randomized, crossover	18 d	18	42 and 84 g almonds/d vs no almonds	—	—	−4.5 g (42 g) −9.1 g (84 g)	—	—	—	—	—
Foster (125)	2012	RCT weight loss	18 mo	123	56 g/d vs no almonds	—	—	—	—	—	—	↓	—
Tan (19, 21)	2012	RCT (suppl)	4 wk	137	43 g/d vs no almonds	↔	↓	—	—	—	—	↔	↔
Hazelnuts													
Mercanligil (34)	2007	Randomized, crossover	4 wk	15	40 g hazelnuts/d vs low-fat diet (no hazelnuts)	↑	—	—	—	—	—	↔	—

(Continued)

TABLE 1 (*Continued*)

Type of nut and first author (ref)	Year	Design	Length	*n*	Treatments	EI	Appetite	Fat absorption	Diet compensation	EE	Fat oxidation	Weight	Body fat
Yücesan (35)	2010	Prospective (suppl)	4 wk	21	1 g hazelnuts/kg per day	↑	—	—	—	—	—	↔	—
Macadamia nuts													
Garg (36)	2003	Prospective (suppl)	4 wk	17	40–90 g/d (15%E from macadamia nuts)	—	—	—	—	—	—	↓	—
Peanuts													
Kirkmeyer (18)	2000	Randomized, crossover	180 min and 24 h	24	500 kcal peanuts and PB vs no load condition and preloads matched on weight and volume		↓	—	Peanuts: 104%; PB: 151%	—	—	—	—
Alper (37)	2002	Crossover	30 wk	15	~500 kcal peanuts/d: FF vs ADD vs SUB	↔	↔	—	66% (FF)	↑	—	↔	↔ (SUB, FF) ↑ (ADD)
Griel (14)	2004	Epidemiology	NS	14,262	Users vs nonusers		—	—	—	—	—	Negative assoc	—
Traoret (64)	2008	Randomized, crossover	7–9 d	16	70 g peanuts/d vs no peanuts	↑	—	↓	—	—	—	—	—
Devitt (22)	2011	Randomized, crossover	300 min	66	300 kcal peanuts vs isocaloric load	↔	↔	—	↑ As snacks	—	—	—	—
Pecans													
Morgan (39)	2000	Prospective (suppl)	8 wk	19	68 g pecans/d	↑	—	—	—	—	—	↔	—
Rajaram (40)	2001	Randomized, crossover	4 wk	23	20%E from pecans vs no pecans, both treatments as part of Step 1 diet	↔	—	—	—	—	—	↔	—
Pistachios													
Kocyigit (41)	2006	Prospective (suppl)	3 wk	44	20%E from pistachios	—	—	—	—	—	—	↔	—
Sheridan (42)	2007	Randomized, crossover	4 wk	15	15%E from pistachios vs no pistachios	↔	—	—	—	—	—	↔	—
Li (123)	2010	RCT weight loss	12 wk	59	53 g pistachios/d vs 56 g pretzels/d	—	—	—	—	—	—	↓	—
Baer (69)	2012	Randomized, crossover	3 wk	18	42 and 84 g pistachios/d vs no pistachios	—	—	−4.5 g (42 g) −6.7 g (84 g)	—	—	—	—	—

(*Continued*)

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Type of nut and first author (ref)	Year	Design	Length	*n*	Treatments	EI	Appetite	Fat absorption	Diet compensation	EE	Fat oxidation	Weight	Body fat
Almario (43)	2001	Crossover	6 wk	18	48 g walnuts/d vs no walnuts	↑	—	—	—	—	—	↔	—
Tapsell (89)	2004	RCT weight maintenance	6 mo	58	30 g walnuts/d vs no walnuts	↔	—	—	—	—	—	↔	↓ (NS)
Sabaté (120)	2005	Randomized, crossover	6 mo	90	28–56 g/d (12%E from walnuts) vs no walnuts	↑	—	—	—	—	—	↑	↑
Walnuts													
Casas-Augustench (16)	2009	Randomized, crossover	5 h	29	33%E PUFAs from walnut-enriched meal vs olive oil (MUFAs)– and dairy product (saturated fat)–enriched isocaloric meals	—	↔	—	—	↑	↑ (NS)	—	—
Tapsell (85)	2009	Randomized, crossover	8 h	16	30 g walnut-enriched meal vs olive oil–enriched isocaloric meal	—	—	—	—	↔	↑	—	—
Tapsell (79)	2009	RCT weight maintenance	12 mo	50	30 g walnuts/d vs lower-fat diet (no walnuts)	↓	—	—	—	↔	—	↓	↓ (NS)
Brennan (20)	2010	Randomized, crossover	4 d	20	48 g walnuts/d vs isocaloric placebo	—	↓	—	—	↔	—	—	—
Pine nuts													
Pasman (11)	2008	Randomized, crossover	240 min	18	3 g pine nut FFA or 3 g TGs vs 3 g placebo (olive oil), all in combination with breakfast	—	↓	—	—	—	—	—	—
Nut not specified													
Ellsworth (108)	2001	Epidemiology	12-y follow-up	34,111 women	Eating frequency per week	—	—	—	—	—	—	Negative assoc (NS)	—
Albert (106)	2002	Epidemiology	12-mo follow-up	21,454 men	Eating frequency per week	—	—	—	—	—	—	No assoc	—
Bes-Rastrollo (107)	2007	Epidemiology	Median 28 mo	8865	Eating frequency per week	—	—	—	—	—	—	Negative assoc	—
Mozaffarian (111)	2011	Epidemiology	NS	120,877	Serving size	—	—	—	—	—	—	Negative assoc	—

[1] ADD, added to diet; assoc, association; EE, energy expenditure; EI, energy intake; FF, free feeding; FFA, free fatty acids; PB, peanut butter; RCT, randomized controlled trial; ref, reference; SUB, replaced equal amount of fat in diet; suppl, supplement; TG, triglyceride; ↑, significantly higher; ↓, significantly lower, ↔, no change; %E, percentage of energy; —, did not measure.

appetite-modulating effects of that meal, whereas consuming almonds alone as snacks blunts hunger and desire-to-eat ratings compared with individuals who received no nuts (19, 21). Other work noted that the ingestion of peanuts or peanut-containing snacks (300 kcal/d) tended to induce greater energy compensation when ingested as snacks relative to when they were consumed as part of a lunch meal (22). Hence, the form and timing of nut consumption may modulate appetitive sensations, with suggestive evidence that satiation/satiety effects may be greater for whole nuts consumed as snacks.

The underlying mechanisms for the appetitive effects of nut consumption are not well understood due to a paucity of studies on the issue. However, the available evidence indicates that the satiating effects of nuts are not likely to be mediated by delayed gastric emptying (23) or the release of selected appetite-regulating gut peptides including glucagon-like peptide 1 (GLP-1), glucose-dependent insulinotropic polypeptide (GIP), or ghrelin (10, 17, 20). However, the effect of nut consumption on protein YY (PYY) secretion is mixed (10, 20). Furthermore, feeding 3 g of pine nut oil (in the form of fatty acids or triglycerides) reportedly decreased prospective food-intake ratings and increased the secretion of cholecystokinin (CCK) (11). Together, these studies suggest that the satiating effects of nuts may be mediated by CCK and/or PYY secretion and that this effect may stem from the dietary protein or fat content of nuts. Their high unsaturated fat content has been proposed as the primary driver of satiety (24, 25). This hypothesis was based on evidence that unsaturated fat is oxidized more readily than saturated fats (26) and so would generate a more rapid and stronger satiety cue. However, several trials have directly tested this hypothesis and have not provided experimental support (27–30).

An expectation of appetite modulation by nuts is that it will translate into reduced energy intake from the balance of the diet by evoking a strong compensatory dietary response. Compensation data from trials that used almonds (31–33), hazelnuts (34, 35), macadamia nuts (36), peanuts (37, 38), pecans (39, 40), pistachios (41, 42), and walnuts (43) suggest that values range from 54% to 104% (18, 31, 33, 37). Thus, the majority of the energy provided by nuts is offset by spontaneous adjustments in the total diet. Dietary compensation may depend on the form of nuts consumed. Peanuts, in the form of peanut butter, produced higher dietary compensation than whole peanuts (104% compared with 151%) (18), despite evoking a weaker satiety effect (44). In summary, nut ingestion suppresses hunger and desire to eat and promotes fullness. These sensations may aid dietary compensation that offsets much of the energy contributed by nuts. However, strong compensation can also occur independently of reported appetitive effects. This may reflect imprecision in appetite measurement or a truly independent uncharacterized mechanism.

MASTICATION AND THE EFFICIENCY OF NUTRIENT ABSORPTION

Although the nutritive value of nuts is well documented, a growing body of evidence indicates that the published values may be substantively modified by the nut's physical properties (45). In particular, the structure and high fiber content of nuts modify the bioaccessibility and bioavailability of the nutrients they contain. To access the nutrients in nuts, their parenchymal cell walls must be disrupted. This may occur by enzymatic or microbial degradation or mechanical processing in the mouth (chewing) and stomach. The fiber nuts provide may bind with food constituents such as fatty acids, reducing the efficiency of their absorption (46). Fiber may also alter gastric emptying and gastrointestinal transit times and gut hormone secretion, with implications for appetite and energy intake (47). In addition, the cell walls may serve as a source of fermentable fiber in the colon, affecting energy balance and gut health (48, 49). Because these dynamic processes will largely determine the nutritional impact of nut consumption, they are attracting increasing research attention (50).

Nuts require considerable oral processing effort and this may, in part, account for the often-noted less-than-predicted effect of their consumption on body weight (31, 35, 39, 43). The mechanical act of chewing reportedly generates satiation signals through cognitive (51), neural (52), endocrine (12, 53), and physical (eg, gastric emptying) (54) mechanisms; augments cephalic phase responses linked to appetite (55–58); influences digestion efficiency (12, 59–61); modestly increases energy expenditure (62); and elicits dietary compensation (63).

A number of studies have evaluated the efficiency of energy absorption from ground and tree nuts through feeding trials. All showed substantive increases in fecal fat loss with nut consumption, although the values ranged widely from ~5% to >20% (12, 61, 64–70). One early trial, in which peanut products constituted 95% of daily fat intake, reported the percentage of dietary fat excreted was 17.8% for whole peanuts, 7.0% for peanut butter, and 4.5% for peanut oil (68). These results documented a food form effect. However, there was also an effect of background diet because these fecal fat-loss values were observed when participants were consuming 20 g crude fiber daily, but decreased to 16.8%, 4.2%, and 1.8% for the 3 peanut forms when the background dietary fiber content was reduced to 5 g/d. Presumably, there was greater binding of energy-yielding nutrients, especially fatty acids, to the fiber in the high-fiber condition, leading to greater fecal excretion. The contribution of background diet to treatment effects may account, in part, for differences in absorption efficiency across studies. Subsequent work showed elevated fecal fat excretion with nut consumption across different types of nuts, including almonds (12, 65, 67, 70), pecans (66), pistachios (69), and peanuts (64). In some (67, 70), but not all (61, 69), studies, the increment in fecal fat followed a dose-response pattern. In the latest studies conducted in the same laboratory under comparable conditions, fecal fat losses associated with 42 g almond and pistachio loads/d were comparable at ~4.5 g/d (61, 69). However, they were higher with almonds (~9.1 g/d) compared with pistachios (~6.7 g/d) at higher nut intakes (84 g/d). This suggests relative consistency across nuts for effects at suggested levels of consumption and possibly a nonlinear dose-response relation for some types of nuts.

These trials raise a number of nutritional issues. First, the inefficiency of absorption is a double-edged sword. Whereas this may be beneficial with respect to moderating energy intake and the possible contribution of nuts to positive energy balance and weight gain, it also likely reduces the absorption efficiency of lipid-soluble nutrients and other macronutrients. Fat accounts for ~55% of the increment in fecal energy loss when nuts are included in the diet (64), so other energy-yielding substances

must also be affected. Vitamin E extraction is lower in whole compared with finely ground and defatted almonds with added almond oil (45). The availability of all nutrients is increased with longer gastrointestinal residence time due, in part, to swelling of cell walls and leakage of nutrients out of the parenchymal cells (45). Consequently, nut form can be used for selective purposes: that is, consumption of whole nuts when moderating energy intake is of primary concern; consumption of chopped, sliced, or finely ground nuts or butter or oils when maximization of nutrient intake is the priority. The delayed absorption of lipid from whole nuts may also moderate postprandial lipemia (71) and glycemia (17). Processing of nuts is an additional factor that influences nutrient bioaccessibility (4). Roasting of almonds leads to smaller fragments with greater bioaccessibility of cell contents when chewing. Mastication studies of peanuts confirm that the physical properties of nuts influence their disintegration characteristics (13). They also show that the matrix in which peanuts are embedded modifies chewing behavior but without modifying the final nut particle size. Simulated gastric processing of peanuts shows graded disintegration rates, from the fastest to the slowest in the following order: frying > roasting > boiling > raw (5).

Second, the data on energy bioaccessibility raise questions about the use of Atwater conversion factors for nutrient labeling. A goal of labeling based on standardized servings is to permit consumers to make informed choices about the energy content of different foods to meet their nutritional goals. The structure of nuts may be different enough from other foods to render labels sufficiently inaccurate to warrant another basis for determination of their energy contribution to the diet (61, 69, 72).

Third, with the introduction of flavorings to nuts to enhance their appeal, questions have been raised as to whether flavors modify chewing and health outcomes. Because flavors are applied to the outer surface of nuts, they exert a strong sensory impact (73) and the actual amount added is limited. Consequently, they would not be expected to directly influence health risks associated with the flavor principle (eg, exacerbation of hypertension by sodium or hyperglycemia by sugar). However, they could theoretically alter oral processing and modify nutrient availability from the nuts themselves. The limited available data suggest that among the varieties tested (ie, raw, roasted unsalted, roasted salted, and honey roasted), no effects of flavor were reported on masticatory outcomes including particle size distribution (74, 75).

Issues of bioaccessibility raise questions about nutrient delivery between types of nuts. Almonds and peanuts have markedly different hardness. The initial break forces for almonds and peanuts are reported as follows: raw nut (7442 ± 332 compared with 3046 ± 380 *g*), honey roasted (5981 ± 172 compared with 1834 ± 232 *g*), roasted unsalted (5004 ± 209 compared with 1545 ± 337 *g*), and roasted salted (4940 ± 267 compared with 1195 ± 289 *g*) (74, 75). These differences in hardness lead to variation across forms and between nut types for indexes of oral processing effort such as number of chews, chewing rate, and time spent chewing, but the end result is a strikingly similar profile of particle sizes (**Figure 1**). No differences related to BMI have been reported, but processing effort is stronger in the fasted compared with sated states (74, 75). Thus, to the extent that particle size proxies for nutrient availability, the present literature suggests more commonalities across nuts than differences. This coincides with a large body of literature showing similar effects of different nuts on cardiovascular disease risk, postprandial glycemia, and body weight (15, 76–78).

ENERGY EXPENDITURE

A limited number of trials have explored the effects of nut consumption on thermogenesis, either postprandially (also known as diet-induced thermogenesis) or on resting energy expenditure (REE). The fatty acid composition of nuts has been the target of much of this work. In an acute-feeding setting, the consumption of a meal containing walnuts (33% of energy from PUFAs) increased diet-induced thermogenesis significantly when compared with a dairy-containing meal (32% of energy from saturated fat) (16). An isoenergetic meal containing olive oil (31% of energy from MUFAs) yielded comparable results to the walnut meal. The test diets were not perfectly matched, so the authors' attribution of the elevation of energy expenditure and fat oxidation to the higher PUFA content of the walnut and the higher MUFA content in the olive oil meals cannot be verified. In addition, the inclusion of almonds [60 g (74) or 54.3 g (31)] and peanuts [52.5 g (37)], both rich sources of unsaturated fats, into meals has not led to elevated thermogenesis. The acute postprandial thermogenic effect of nuts is yet to be confirmed.

Mixed findings on thermogenesis have been reported from short- and longer-term (ranging from 4 d to 12 mo) trials of nut consumption. No effects have been documented with walnuts (20, 79). One study with almonds reported no effect, whereas another observed an increase that accounted for ~14% of the energy contributed by the almonds (31, 33). Several studies with peanuts have noted an increase in REE. In one trial, there was an 11% increase after 8 wk of peanut consumption (52.5 g/d). When lean and overweight adults were supplemented with peanut oil (as 30% of REE) for 8 wk in another study, REE was elevated by 5%, but only in overweight individuals (80). Collectively, there is some evidence that nut consumption increases thermogenesis, but the data are not robust and there is no clear mechanism. One possibility is that the lipid from nuts is absorbed over a prolonged period of time, leading to a small but sustained source of substrate that fuels thermogenesis and could appear as an increase in REE.

ADIPOSE TISSUE AND FAT METABOLISM

It has been proposed that nut consumption elevates fat oxidation and preferentially reduces body fat mass, especially in the viscera. These actions are attributed to their high unsaturated fat content. If true, their inclusion in the diet could help to prevent or mitigate the effects of metabolic syndrome. Animal studies have shown that higher PUFA intakes suppress adipocyte differentiation and downregulate adipocyte P2 and adipsin genes (81). However, PUFAs did not have an effect on adipose tissue size (82). In rodent retroperitoneal (but not subcutaneous) adipose tissues, fatty acid synthase (FAS), hormone-sensitive lipase (HSL), phosphoenolpyruvate carboxykinase (PEPCK), lipoprotein lipase (LPL), CCAAT/enhancer binding protein α (C/EBPα), and leptin mRNA concentrations decrease with higher PUFA intake, suggesting targeting of the visceral fat pool (82). There are also indications that fat metabolism is enhanced by higher PUFA intake (83). Mitochondrial protein gene expression

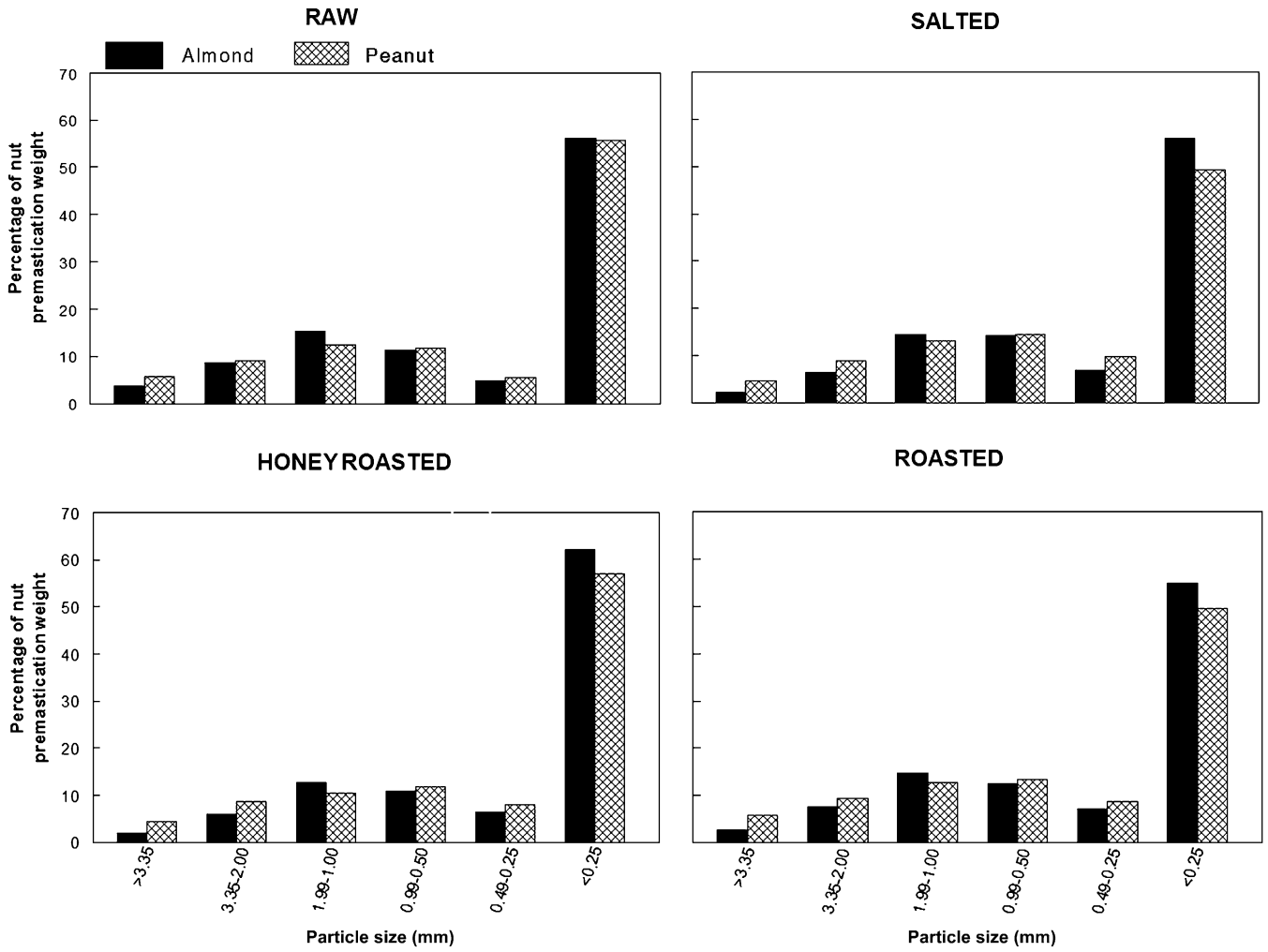

FIGURE 1. Particle sizes (in mm) of raw, salted, roasted, and honey roasted almonds and peanuts after mastication. The *x* axis indicates the size of particles after mastication before swallowing.

is upregulated in the epididymal fat of mice fed PUFAs (84). The expression of genes that regulate oxidative metabolism [eg, peroxisome proliferator-activated receptor α (*PPARα*), peroxisome proliferator-activated receptor γ coactivator 1α (*Ppargc1a/Pgc1α*), and nuclear respiratory factor 1 (*Nrf1*)] are also elevated (84).

In one acute-feeding study, a high-PUFA diet (33% of energy) enriched with walnuts increased fat oxidation in humans (albeit not significantly) (16). In another study, fat oxidation was significantly elevated ($\sim$50% higher compared with a control diet) when 30–35 g of walnuts were ingested by overweight and obese adults (85). The stronger effects noted in this trial may reflect the difference in participant BMI status. Blunted fat oxidation has been reported in adults with high body weight (86), but the inclusion of walnuts appears to reverse or normalize impaired fat oxidation. Notably, in the latter study, the dietary fat concentration and the composition of fat subtypes were matched, suggesting that the fat-oxidizing property of walnuts was not limited to their PUFA content alone. There are no human studies of fat oxidation with other nuts, which limits extrapolation of the walnut findings. Other nuts are richer sources of MUFAs compared with the high PUFA content of walnuts, and MUFAs are reported to induce comparable or higher fat oxidation rates (87, 88). This suggests that equal or greater effects on fat oxidation may be expected with other nuts.

Several clinical trials have examined whether elevated fat oxidation induced by walnuts translates into fat mass loss over time. In one trial (89), the inclusion of walnuts in a weight-maintenance diet of type 2 diabetic adults led to a small reduction in body fat, although body weight remained stable during the 6-mo study period. Body fat increased in individuals who adhered to the control diet (low-PUFA), and the difference between the groups approached significance ($P = 0.057$). The trend persisted when the intervention period was extended to 1 year, where the inclusion of walnuts produced greater fat mass loss relative to the control group despite comparable weight in the groups (79). Early evidence suggests that the loss of fat mass derived predominantly from the subcutaneous and less from the visceral fat pools, although the size of both fat depots decreased over time in the walnut group (79). The effects of PUFAs on different body fat pools have been studied more intensively with the use of animal models (81, 90–95). This work consistently shows that higher PUFA intake reduces visceral fat. However, findings in humans, to date, do not replicate these results (79). Indeed, just the opposite has been reported, in which PUFAs appeared to

preferentially reduce subcutaneous fat. No explanation for the inconsistent outcomes between human and rodent studies is apparent. Because subcutaneous and visceral fats are part of total body fat, evidence has shown that visceral fat loss is primarily determined by total fat mass loss (96). Human studies incorporating different nuts into the diet at realistic doses are needed to determine the effect of nut consumption on body composition. Findings may yield insights for management of body fatness and risk of metabolic syndrome.

NUT CONSUMPTION AND BODY WEIGHT

The literature on nut consumption and body weight has been the topic of several reviews (76, 97–104). Generally, epidemiologic studies indicate that incorporating nuts into diets on a regular basis does not compromise, and may aid, weight maintenance (14, 105–111). Because energy balance is the ultimate determinant of body weight, not surprisingly, controlled feeding studies using almonds, walnuts, pecans, and macadamia nuts all indicate that nut consumption does not cause changes in body weight when energy intake is continually adjusted (14, 40, 112–115). More important, when total energy intake is less controlled, studies that involve the inclusion of nuts in habitual diets of free-living individuals have also shown that nut consumption does not lead to weight gain (36, 116–119). However, it must be noted that these were relatively short-term trials with limited power to detect small changes in body weight. Although there are reports of small, but significant increases in body weight with nut consumption (30, 120–122), the preponderance of evidence indicates that under controlled or free-living situations, nut consumption does not promote weight gain.

Several studies assessing the role of nut consumption in weight-maintenance programs have noted a decrease in body weight from baseline (32, 36, 40, 79, 113, 114). Whether this is due to a greater thermic effect of food or REE effect of the nuts compared with the foods they displaced in the diet has not been established. Nevertheless, current data indicate that the inclusion of nuts in a weight-maintenance program will not lead to weight gain and may aid weight loss.

The inclusion of nuts in energy-restriction regimens does not impede weight loss (115, 123–126). In several trials in which nuts did not augment weight loss (125, 126), there was a reduction in cardiovascular disease risk indexes in the nut-consuming groups, suggesting that such benefits derive from properties of the nuts rather than just weight change. There is a need for long-term randomized intervention studies with body weight as a primary outcome to establish the effect of nuts consumed daily in realistic quantities on maximal and sustainable weight loss.

CONCLUSIONS

It is now well established that body weight and fatness are functions of energy balance rather than the macronutrient content of the diet (125, 127, 128). Nuts are a high-fat, energy-dense food, but the evidence indicates that they pose little challenge to and may even aid weight management. This is attributable to the strong dietary compensation effects they elicit, inefficiency in the absorption of the energy they provide, and possibly an elevation of energy expenditure and fat oxidation. Although energy is the determinant of body weight and composition, the greater health effects of diets will be determined by their macronutrient content and other constituents. Nuts are rich sources of unsaturated fats, minerals, vitamins, antioxidants, and fiber, which can contribute to a healthful diet.

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