# Nut consumption and body weight[1–3]

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**ABSTRACT** Frequent nut consumption is associated with lower rates of coronary artery disease (CAD). Also, nut-rich diets improve the serum lipid profile of participants in dietary intervention trials. However, nuts are fatty foods, and in theory their regular consumption may lead to body weight gain. Because obesity is a major public health problem and a risk factor for CAD, clinicians and policy makers ponder several questions. Will hypercholesterolemic patients advised to consume nuts gain weight? Is recommending increased nut consumption to the general population for CAD prevention sound public health advice? Epidemiologic studies indicate an inverse association between frequency of nut consumption and body mass index. In well-controlled nut-feeding trials, no changes in body weight were observed. Some studies on free-living subjects in which no constraints on body weight are imposed show a nonsignificant tendency to lower weight while subjects are on the nut diets. In another line of evidence, preliminary data indicate that subjects on nut-rich diets excrete more fat in stools. Further research is needed to study the effects of nut consumption on energy balance and body weight. In the meantime, the available cumulative data do not indicate that free-living people on self-selected diets including nuts frequently have a higher body mass index or a tendency to gain weight. *Am J Clin Nutr* 2003;78(suppl):647S–50S.

**KEY WORDS** Nuts, body weight, obesity, dietary recommendations

## INTRODUCTION

Over the past decade, it has become evident that nut consumption has a beneficial effect on coronary artery disease (CAD) risk (1–5). To date, 4 large prospective cohort studies have shown that frequent nut consumption is associated with lower rates of CAD (6–9). Also, a number of nut-feeding studies either in well-controlled dietary conditions or with free-living subjects consuming self-selected diets have demonstrated beneficial effects of nuts on serum lipids and lipoproteins, important markers for CAD risk (4, 5).

All nuts but chestnuts are high in fat. By weight, the total fat content ranges from 45 to 75%, but this fat is largely unsaturated (10). The fat contents of several nuts are summarized in **Table 1**. Traditionally, nuts have been perceived by the general public as fattening because of their high fat content. The evidence that a higher percentage of fat in the diet results in increased body weight is controversial (11). Yet health professionals are concerned whether hypercholesterolemic patients advised to eat nuts will gain weight (12). Additionally, obesity is a growing public health problem and a risk factor for CAD. Of importance for policy makers is whether the promotion of nut consumption for primary prevention of CAD may result in higher prevalence of obesity among the general population. In short, is advocating increased consumption of nuts good clinical advice and/or sound public health policy?

These important questions cannot yet be directly addressed with data. So far, no well-controlled feeding study on nuts that has body weight as the main outcome has been conducted. Indirect evidence, however, does not suggest that long-term nut consumption leads to weight gain. This paper reviews available data from consumer surveys, epidemiologic studies, and dietary intervention nut studies for which weight or body mass index (BMI) data are also reported.

## EPIDEMIOLOGIC EVIDENCE

Ecological data do not relate long-term nut consumption with obesity. For instance, per capita nut consumption in Mediterranean populations is about double that in the United States (1), and yet these populations' obesity rates are much lower. A cross-sectional study of 800 schoolgirls in Spain found no relationship between frequency of nut consumption (ranging from never to daily) and body weight (13).

Data from the 1994–1996 Continuing Survey of Food Intakes by Individuals conducted by the US Department of Agriculture has been used to compare BMI and total energy intake of nut eaters with that of non-nut eaters (14). Dietary intake data from a nationally representative sample were collected on 2 nonconsecutive 24-h recalls. Those reporting consumption of tree nuts, peanuts, or seeds on any of the 2 d were included in the nut eater group. Data in **Table 2** show that young and adult nut eaters had a lower BMI compared with non-nut eaters. Interestingly, energy intake was higher among nut consumers. Also, among nut eaters, the amount of nuts and seeds in their diets did not relate to BMI ($r = 0.04$, $P = 0.11$). The results of this analysis indicate an inverse relationship or no relationship between intake of nuts and BMI in the US population.

An inverse or no relationship between frequency of nut intake and BMI has also been observed in all large cohort studies from which these data were reported. In the Adventist Health Study, Fraser et al (6) reported a statistically significant negative association between consumption of nuts and BMI in a cohort of 31 200 California subjects, showing that those who ate nuts more frequently were leaner than the infrequent nut eaters. Hu et al (8) also

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**TABLE 1**
Fat composition of nuts[1]

| | Total fat | SFAs | MUFAs | PUFAs | UFAs:SFAs |
|---|---|---|---|---|---|
| | *% of weight* | *% of total fat* | *% of total fat* | *% of total fat* | |
| Almonds | 52 | 10 | 68 | 22 | 9.0 |
| Brazil nuts | 66 | 26 | 36 | 38 | 2.8 |
| Cashews | 46 | 20 | 62 | 18 | 3.9 |
| Hazelnuts | 63 | 8 | 82 | 10 | 11.9 |
| Macadamia nuts | 74 | 16 | 82 | 2 | 5.4 |
| Peanuts | 49 | 15 | 51 | 34 | 5.7 |
| Pecans | 68 | 8 | 66 | 26 | 10.9 |
| Pine nuts | 61 | 15 | 40 | 45 | 5.4 |
| Pistachios | 48 | 13 | 72 | 15 | 6.6 |
| Walnuts | 62 | 10 | 24 | 66 | 9.0 |

[1] Data from reference 10. MUFAs, monounsaturated fatty acids; PUFAs, polyunsaturated fatty acids; SFAs, saturated fatty acids; UFAs, unsaturated fatty acids (MUFAs plus PUFAs).

reported a negative association between nut consumption and BMI among 86 000 females in the Nurses Health Study. There was no apparent association between BMI and nut consumption in the Physicians' Health Study (9). BMIs by quartile of nut consumption (ranging from never to 2 times/wk) were 24.9, 24.9, 25.0, and 24.7 among this cohort of 21 500 males. Prospective cohort studies also demonstrated that nut consumption protects against CAD in both lean and obese people (6, 8). There was a substantial and similar reduction in CAD risk of ≈50% for individuals with either low or high BMI consuming nuts ≥5 times/wk compared with their counterparts consuming nuts <1 time/wk in the Adventist Health Study (15; **Figure 1**). Similar trends were also reported in the Nurses Health Study (8). Because obese people are at higher risk for CAD, it is particularly relevant that nut consumption may actually reduce their risk.

## NUT-FEEDING STUDIES

A number of dietary intervention studies on different types of nuts have been conducted (4, 5). Although differing in methodology and dietary control, collectively these investigations provide (substantial) evidence that short-term consumption of moderate to large amounts of nuts does not increase body weight. None of the well-controlled metabolic-type feeding studies show significant changes in body weight comparing the nut diet and non-nut control diet (16–20). This is expected in these types of studies where investigators adjust energy intake of participants to maintain body weight stability. In the controlled cross-over feeding studies (16, 19) conducted at Loma Linda University with walnuts, pecans, and almonds, subjects, while on the nut diets tended to be hungrier and to require more energy intake to maintain body weight than while on the nut-free control diets. Less controlled dietary intervention studies on nut-prescribed but self-prepared diets do not report weight gain either. To the contrary, most studies observed a nonsignificant tendency to lower weight while subjects were on the nut diets (21–27). One study reported a significant weight loss of ≈3 kg over 6 mo in the group fed nuts (28). Thus, these studies demonstrate that the isocaloric replacement of nuts for other foods in the diet does not lead to weight gain.

Two recent studies investigated the effects on body weight when the habitual diet of free-living subjects is supplemented with nuts. We recently reported that 6 mo of almond supplementation had minimal effects on body weight (29). Eighty-one subjects were provided with 42–70 g raw or dry-roasted almonds/d but were given no specific dietary instructions other than eating the nuts. After 6 mo of consuming the almond supplement, men gained 0.65 kg ($P < 0.01$) while women did not gain any significant weight (0.11 kg; $P = 0.79$). Only lean subjects (in the lowest tertile of baseline BMI) gained weight during the almond phase of the study, and women in the highest-baseline-BMI tertile actually lost weight with almond supplementation.

Alper and Mattes (30) reported the effect of peanut supplementation on weight gain in 15 adults with normal weight. Subjects were provided with 500 kcal/d peanuts (89 g) under 3 experimental conditions: a "free-feeding" phase in which participants were given peanuts without dietary guidance; an "addition" phase in which subjects were asked to add peanuts to their habitual diet; and a "substitution" phase during which peanuts replaced an equal amount of other fats in the diet. During the substitution period, no weight gain was observed. In the free-feeding phase, subjects gained 1.0 kg during the 8-wk intervention, considerably less than the predicted 3.6 kg based on the additional calories provided. Similarly, during the addition period, subjects gained only 0.6 kg, while 1.4 kg had been predicted. Part of this difference was attributable

**TABLE 2**
BMI and energy intake of nut eaters and non-nut eaters: Continuing Survey of Food Intakes by Individuals (CSFII), 1994–1996[1]

| | All | | Young persons (6–20 y of age) | | Adults (≥21 y of age) | |
|---|---|---|---|---|---|---|
| | Nut eaters ($n$ = 1993) | Non-nut eaters ($n$ = 10 095) | Nut eaters ($n$ = 649) | Non-nut eaters ($n$ = 2459) | Nut eaters ($n$ = 1344) | Non-nut eaters ($n$ = 7636) |
| BMI (kg/m$^2$) | 23.8 ± 0.1[2] | 25.0 ± 0.1 | 19.6 ± 0.2[2] | 21.2 ± 0.1 | 25.6 ± 0.1[2] | 26.1 ± 0.1 |
| Energy (kcal/d) | 2191 ± 20[2] | 1997 ± 9 | 2194 ± 40[3] | 2110 ± 18 | 2189 ± 24[2] | 1965 ± 10 |

[1] $\bar{x}$ ± SE. Means are sample weighted to provide population estimates.
[2,3] Significantly different from non-nut eaters ($t$ test): [2] $P < 0.001$, [3] $P < 0.05$.

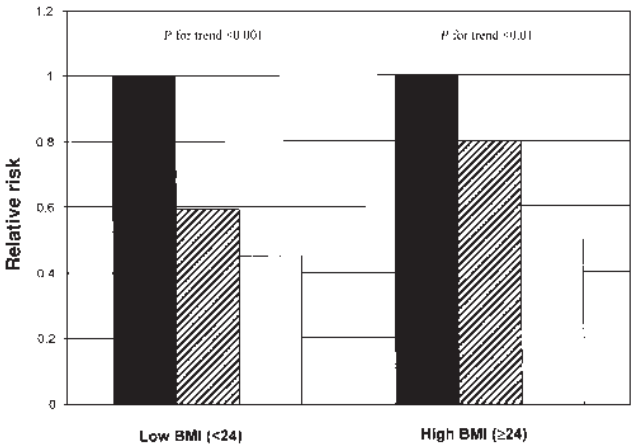


**FIGURE 1.** Age- and sex-adjusted relative risks of coronary artery disease at 3 nut intakes (■, < 1 time/wk; ▨, 1–4 times/wk; □, ≥ 5 times/wk) according to BMI (in kg/m$^2$) in the Adventist Health Study. Adapted from data from reference 15.

to dietary compensation for a portion of the extra calories provided by peanuts. In addition, resting energy expenditure was significantly higher after consumption of peanuts, although physical activity levels did not change.

Interestingly, nuts may have a role in the context of supervised weight-reduction programs. McManus et al (31) reported that obese subjects prescribed a moderate-fat weight-reduction diet containing several nuts, peanut butter, and olive oil experienced greater and more sustained weight loss than obese subjects prescribed a low-fat diet. Also, obese subjects on the nut-containing diet were significantly more likely to remain compliant at 18 mo. This increased adherence to a weight-loss regimen is attributed largely to increased palatability, flavor, and texture of the foods incorporated in the moderate-fat diet.

## POSSIBLE REASONS

Suggested reasons why nut consumption is not associated with increased BMI in free-living individuals include reverse causation, higher energy expenditure through physical activity or increased resting metabolic rate, enhanced satiety and corresponding decreased intake of other foods, and incomplete absorption of energy from nuts.

Reverse causation may explain to some extent the reported inverse relation between nut consumption and BMI found in consumer surveys and baseline data of cohort studies. Obese people may tend to avoid nuts because of their high fat and energy content, while lean individuals may have fewer reservations about consuming them.

People who eat nuts may tend to engage in higher levels of physical activity than non-nut eaters. Hu et al (8) reported that nut consumption was associated with greater frequency of vigorous exercise among the Nurses Health Study participants. The Physicians' Health Study (9) also noted that men who ate nuts more frequently were more physically active. However, in a study supplementing the habitual diet of free-living subjects with almonds for 6 mo, no changes in vigorous exercise were noted (29).

Nuts may increase resting energy expenditure because of their high-protein and unsaturated fat content (32, 33), and this may result in less fat deposition. In rats, a diet high in unsaturated fats led to much less weight gain and more oxygen consumption than a high-fat diet (34, 35). In humans, a high polyunsaturated-to-saturated-fatty-acid ratio in the diet can increase resting energy expenditure and diet-induced thermogenesis (33, 36). Alper and Mattes (30) reported an 11% greater resting energy expenditure in 15 subjects after peanut supplementation for 19 wk. However, Fraser et al (29) reported no changes in resting energy expenditure after daily almond supplementation for 6 mo.

Nuts may enhance satiety. They are energy-dense and good sources of fiber and protein, dietary factors that increase satiety ratings (37, 38). Peanuts exercised a strong suppression of hunger and affected subsequent food intake in a preload study (39). Dietary compensation seems to be a major reason for the lack of predicted weight gain in long-term nut-supplemented diets. On a 6-mo almond supplement study, 54–78% of the extra energy from almonds was displaced by reductions in other foods (29). Similar observations were reported on a peanut study of shorter duration (30).

Fecal fat loss because of incomplete mastication of nuts or other factors may result in a loss of available energy. Earlier work has shown that whole nuts are inefficiently absorbed (40). Subjects fed whole peanuts excreted 17% of dietary fat in the stool; only 4–7% of dietary fat was excreted when the rats were fed peanut butter (40). On a well-controlled feeding trial with pecans (19), there was increased excretion of fat in the stools of subjects while on the nut diet (25 g/d) compared with the control diet (6 g/d). This represented 8% and 3%, respectively, of the dietary fat of the pecan and control diets (41). Increased stool fat was also noted on a high almond diet (42), but the increment of percent fat in stools (4%) was less than on the pecan (41) and peanut (40) diets. Nevertheless, the losses of fat in stools of nut eaters combined with the observed food displacement would largely explain the lack of weight gain.

## CONCLUSIONS

The available data demonstrate that nut consumption among free-living individuals is not associated with higher BMI compared with non-nut consumers despite the fact that nuts are fat- and energy-dense foods. Isocaloric replacement of nuts for other food in the diet does not increase weight. Research is needed to directly address the effects of nut consumption on energy balance, body weight, and anthropometric parameters. In the meantime, the current data do not indicate that free-living people on self-selected diets including nuts frequently have a higher BMI or increased body weight.

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