

Dietary fats, teas, dairy, and nuts: potential functional foods for weight control?¹⁻³

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ABSTRACT

Functional foods are similar to conventional foods in appearance, but they have benefits that extend beyond their basic nutritional properties. For example, functional foods have been studied for the prevention of osteoporosis, cancer, and cardiovascular disease. They have yet to be related to the prevention of obesity, although obesity is one of the major health problems today. The inclusion of foods or the replacement of habitual foods with others that may enhance energy expenditure (EE) or improve satiety may be a practical way to maintain a stable body weight or assist in achieving weight loss; such foods may act as functional foods in body weight control. Some foods that might be classified as functional foods for weight control because of their effects on EE and appetite—including medium-chain triacylglycerols, diacylglycerols, tea, milk, and nuts—are reviewed here. Only human studies reporting EE, appetite, or body weight are discussed. When studies of whole food items are unavailable, studies of nutraceuticals, the capsular equivalents of functional foods, are reviewed. To date, dietary fats seem to be most promising and have been the most extensively studied for their effects on body weight control. However, the weight loss observed is small and should be considered mostly as a measure to prevent weight gain. Carefully conducted clinical studies are needed to firmly ascertain the effect of tea, milk, and nuts on body weight maintenance, to assess their potential to assist in weight-loss efforts, and to ascertain dose-response relations and mechanisms of action for the 4 food types examined. *Am J Clin Nutr* 2005;81:7–15.

KEY WORDS Medium-chain triacylglycerols, diacylglycerols, tea, milk, nuts

INTRODUCTION

Maintenance of a constant body weight requires a balance between energy intake (EI) and energy expenditure (EE), and even a slight imbalance in this energy equilibrium can lead to significant changes in body weight over time and may eventually result in obesity (1). Obesity is one of the major health problems worldwide, and it is a risk factor for several chronic disorders, but there is no functional food for obesity, such as there is for cardiovascular disease (CVD) or cancer. In the Health Professionals Follow-up Study, mean weight change over a 10-y period was 1.8 kg (2). Even small changes in energy balance may lead to such a weight gain, which therefore may be prevented by slight modifications in food intake, such as the inclusion of functional foods for weight management.

Although it is known that dietary restriction and increased physical activity can lead to weight loss, such lifestyle changes may be difficult to implement and maintain—thus, the high rate of recidivism among weight losers (3). Functional foods that effect energy metabolism and fat partitioning may be helpful adjuncts to a dietary approach to body weight control. The current review examines current literature to identify potential functional foods that may be useful in the prevention of weight gain or as adjuncts to weight-loss efforts. It is not within the scope of this review to examine overall diets or individual food components, such as vegetarian diets and fiber or protein, so that only foods that have been studied for their effects on body weight, EE, or satiety (or all 3) are reviewed. Such potential functional foods that may be of interest include medium-chain triacylglycerols (MCTs), diacylglycerols, tea, milk, and nuts.

DIETARY FATS

Medium-chain triacylglycerols

MCTs are those triacylglycerols composed of fatty acids that contain 6–12 carbon atoms. These triacylglycerols differ from long-chain triacylglycerols (LCTs) not only in their chemical composition but also in the manner in which they are absorbed and transported from the gastrointestinal tract to organs. Both MCTs and LCTs are digested to their respective medium- and long-chain fatty acids (MCFAs and LCFAs, respectively) in the gastrointestinal tract. Unlike LCFAs, which are repackaged as LCTs into chylomicrons for transport through the peripheral circulation, MCFAs, because of their shorter chain lengths, do not require chylomicron formation for their absorption and transport (4, 5). As a result, MCFAs travel directly to the liver via the portal circulation; therefore, they bypass peripheral tissues such as adipose tissue, which makes them less susceptible to the actions of hormone-sensitive lipase and to deposition into adipose tissue stores (**Figure 1**). In fact, MCFAs are mostly oxidized by

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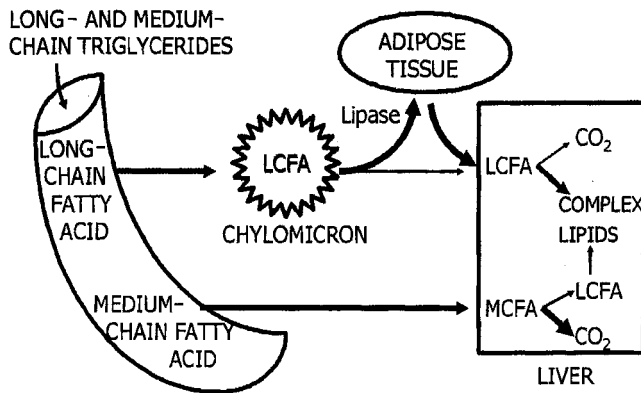


FIGURE 1. Long- and medium-chain triacylglycerols are digested to their respective fatty acids in the gastrointestinal tract. Long-chain fatty acids (LCFAs) are packaged into chylomicrons for their transport to peripheral tissues, whereas medium-chain fatty acids (MCFAs) travel directly to the liver via the portal circulation. As a result, LCFAs are mostly deposited into adipose tissue, whereas MCFAs are mostly oxidized to carbon dioxide in the liver, and small amounts are elongated to LCFAs and incorporated into complex lipids.

the liver for use as a source of energy and thus have been reported to behave more like glucose than like fats (5). The metabolic differences between MCTs and LCTs prompted researchers to examine their effects on EE and body composition. Several reviews discussed the physiologic processing of MCTs (4–6).

Early studies examined the effects of MCTs on EE in humans (7–9). Those investigators mostly reported a greater thermogenic effect of MCTs than of LCTs, but their studies were of a short duration (10). As a result, although it was suggested that the consumption of MCTs could lead to an energy imbalance that may assist in weight loss or in the prevention of obesity (7–9), those early studies did not provide evidence of the longer-term effects of MCTs on thermogenesis. Furthermore, the short-term nature of the studies precluded the possibility of examining body-composition changes that would theoretically result from the energy imbalance.

In the School of Dietetics and Human Nutrition at McGill University, we recently used a 4-wk crossover feeding experiment to compare the effects of MCT and LCT consumption on EE and body composition in both men (11) and women (12). In both studies, subjects were fed controlled diets that were designed to maintain body weight and that differed only in the type of added dietary fat: MCTs or LCTs. Diets were fed for 4 wk and were separated by a 4-wk washout period. EE and body composition, measured by magnetic resonance imaging, were assessed at baseline and the endpoint of each experimental phase. As a result, we could determine whether the greater effects of MCTs than of LCTs on thermogenesis were maintained over a 4-wk period and whether any increase in EE with MCT consumption over that seen with LCT consumption would result in significant changes in body composition, regardless of the isoenergetic content of the diets. We found that EE increased with the consumption of MCTs more than it did with that of LCTs at baseline and at the endpoint in men and women; however, the extent to which thermogenesis was increased varied slightly. In women, differences in daily EE, extrapolated from 6.5 h of indirect calorimetry measurements, equated to 167.2 kJ (40 kcal) from measurements taken at baseline and to 209 kJ (50 kcal) from measurements taken at the endpoint (12). In men, differences in daily EE,

extrapolated in the same manner, equated to 263.3 kJ (63 kcal) at baseline and 179.7 kJ (43 kcal) at the endpoint (11). Although body weights did not change significantly in women, variations in body weight in both men and women could be explained by the differences observed between EE with MCT consumption and that with LCT consumption. Moreover, total adipose tissue, subcutaneous adipose tissue, and upper-body adipose tissue stores in men decreased significantly with MCT consumption but not with LCT consumption (11).

Furthermore, data suggest that MCT consumption increases satiety more than does LCT consumption (10). In a subgroup of men, we tested the potential effect of MCT consumption on food intake at a subsequent meal (12). After consuming the fixed-intake breakfast containing either MCTs or LCTs, men were instructed to eat as much as they liked of a different, ad libitum lunch that did not contain the experimental fats. Although the sample was small ($n = 5$), there was a trend toward lower EI at the lunch after the MCT-containing breakfast than at the lunch after the LCT-containing breakfast. The slightly lower EI [–925 kJ (221 kcal)] was due to significantly lower fat intake (–12.4 g) at the lunch after the MCT-containing breakfast than at the lunch after the LCT-containing breakfast (12).

These results add to the body of literature examining the effects of MCT consumption on EE and further confirm the potential of MCTs to act as dietary adjuncts for improved body weight maintenance or even, possibly, weight loss. However, weight-loss studies are needed to confirm this latter role of MCTs. Found in the form of liquid oil extracted from coconut oil, MCTs could easily be incorporated into the North American diet as a replacement for other LCT-rich vegetable oils. A study examining the acceptability (ie, appeal to the consumer) of different food items made with an MCT oil found that the drop cookies, muffins, and quick loaf breads were acceptable, but the rolled cookies were not (13). In clinical studies in which one-half of the total fat intake was from an MCT oil, it was found that incorporation of an MCT-rich oil into cakes and cookies produced acceptable products. Moreover, subjects had no complaints about the taste of the oil or of the mashed potatoes, pasta, or desserts in which it was incorporated (M-P St-Onge, personal observations, 1999–2001). One must keep in mind, however, that baked goods are typically high in energy, and these foods should be consumed sparingly even if the replacement of LCTs by MCTs contributes to the enhancement of EE and satiety. An oil containing only MCT could be incorporated into foods as part of a salad dressing, but it may not be appropriate for frying because of its low smoke point. One possible way to circumvent this problem would be to combine an MCT oil with other vegetable oils having higher smoke points, such as canola oil or safflower oil, which would then make the MCT oil more suitable for stir-frying and baking.

There are, however, some concerns regarding the effects of MCT consumption on plasma lipid concentrations (14). In fact, a recent study (15) found an intake of 70 g MCT oil/d for 21 d increased total cholesterol, LDL-cholesterol, triacylglycerol, and glucose concentrations by 11%, 12%, 22%, and 4%, respectively, relative to an equivalent intake of high-oleic sunflower oil. There is thus some concern regarding the cardiovascular effects of MCTs. One possible way to prevent adverse cardiovascular effects would be to combine plant sterols, which have showed benefits for cholesterol concentrations (16), with an MCT oil. When this combination is consumed, total and LDL-cholesterol



concentrations are lower than those observed after LCT consumption (17, 18).

Diacylglycerols

Diacylglycerols have also been reported to increase thermogenesis more than triacylglycerols do and to have the potential to assist in weight loss (19) because of a mechanism of action similar to that of MCTs. Diacylglycerols of the 1,3 conformation are catabolized to 2 free fatty acids and a glycerol moiety, as opposed to 2 free fatty acids and a 2-monoacylglycerol molecule, after hydrolysis of triacylglycerol. In the case of triacylglycerol, the 2-monoacylglycerol molecule acts as a backbone for the reformation of a triacylglycerol molecule for packaging into chylomicrons (20). Because of the absence of 2-monoacylglycerol molecules, diacylglycerols cannot be reformed for packaging into chylomicrons, and thus the free fatty acid and glycerol molecules travel as such in the circulation and are diverted to the liver, where they are mostly oxidized (21). In fact, several enzymes involved in fatty acid oxidation have been found to increase with diacylglycerol consumption for 14 d, whereas those involved in synthesis decreased (22).

Few studies (19, 23, 24) have compared the effects of diacylglycerols and of triacylglycerols on EE and body composition in humans. In the study by Nagao et al (19), 38 normal-weight men supplemented their diets with 10 g/d of either diacylglycerol or triacylglycerol. The experimental fats were provided in bread, mayonnaise, and shortbread, which was for consumption at breakfast only. The remainder of the diet was self-selected, and subjects were not counseled to reduce EIs to promote weight loss. The authors measured body composition by using single-slice computed tomography scanning and air-displacement plethysmography. After 16 wk of supplementation, there was a significant decrease in body weight (−2.6 kg) in the diacylglycerol-supplemented group, whereas the weight increase in the triacylglycerol-supplemented group was 1.1 kg. The reductions in body mass index (BMI; in kg/m²), waist circumference, and visceral and subcutaneous adipose tissue at the level of the umbilicus were significantly greater with diacylglycerol than with triacylglycerol supplementation. The authors concluded that diacylglycerol supplementation suppresses body weight and regional fat deposition. However, in both groups, total fat consumption, including the test or control fat, did not meet the 50 g/d requirement and was assessed to be 43 g/d. This lack of compliance with the study protocol may have been partly responsible for the changes observed in body composition. Furthermore, energy and fat intakes for each group were not provided.

A weight-loss study including overweight and obese men and women found that body weight and fat mass decreased to a greater extent in subjects consuming diacylglycerols than in those consuming triacylglycerols (24). All men and women included in this randomized, parallel-arm experiment had a waist circumference > 90 cm (men) or > 87 cm (women). Subjects were asked to reduce their caloric intakes by 2090–3344 kJ/d (500–800 kcal/d) for a period of 24 wk, during which they incorporated foods containing diacylglycerols or triacylglycerols. Foods provided 16–45 g/d of either diacylglycerols or triacylglycerols, or 15% of the subjects' energy requirements. Body composition was assessed by whole-body dual-energy X-ray absorptiometry and single-slice computed tomography scanning at the level of the L4–L5 vertebrae. Both groups lost a significant amount of body weight and fat mass, but the changes were greater

in subjects who were supplementing their diets with diacylglycerol-containing foods than in those who were supplementing their diets with triacylglycerol-containing foods (body weight change: −3.6% and −2.5%, respectively). The percentage change in intraabdominal adipose tissue did not differ significantly between the groups.

Although these previous studies did not examine the potential mechanism leading to greater effects of diacylglycerols than of triacylglycerols on loss of fat mass, it seems likely that the effects could be due to greater EE, fat oxidation, or reductions in appetite with diacylglycerol than with triacylglycerol consumption rather than to differences in the energy contents of diacylglycerol and triacylglycerol. Diacylglycerol and triacylglycerol have been shown to have similar energy values [37 kJ/g (9 kcal/g)] (25). In addition, a recent study showed that, when 12% of total daily EI was provided by diacylglycerol in foods, fat oxidation was greater than that when EI was provided by triacylglycerol in foods (23). EE did not differ significantly between the groups, but subjects reported being less hungry (area under the curve score: 281 and 472 mm · h, respectively) after diacylglycerol consumption than after triacylglycerol consumption. This study provides some information on the reasons for weight loss with diacylglycerol consumption, but much remains to be established. Furthermore, the previous feeding experiments (19, 24) were parallel-arm supplementation studies, and therefore the exact food intake of subjects could not be established with certainty, which may have worked to confound the results obtained.

Diacylglycerols occur naturally in small concentrations in several edible oils; the 9.5% concentration in cottonseed oil is among the highest (26). A diacylglycerol-rich cooking oil has been produced that contains >80% diacylglycerols and that looks and tastes like a conventional oil (26). Such an oil could therefore be incorporated into foods or consumed as a salad dressing without imparting a distinct flavor to the food.

Studies examining the effect of changes in dietary fat type (11, 12, 19, 24) seem to indicate that this slight dietary modification may be beneficial for body weight control and weight loss. In fact, the Japanese government has approved diacylglycerol as a food for specific health use to control postmeal blood lipids and body fat (26). However, the magnitude of this effect is small when observed in a controlled setting, and therefore dietary fats may be most helpful in the prevention of weight gain when used alone or in the enhancement of weight loss when incorporated in a more rigorous weight-loss plan.

BEVERAGES

Tea

Tea is the beverage with the greatest consumption worldwide (27). There are 3 categories of tea—black, green, and oolong—and the consumption of black tea accounts for 80% of total tea intake (27). Black tea leaves are fermented and contain mostly theaflavins and thearubigins as active components (27). Green tea is a nonoxidized, nonfermented tea, which contains polyphenolic compounds such as epicatechin, epicatechin gallate, epigallocatechin, and epigallocatechin gallate (EGCG), whereas oolong tea is partially oxidized and contains a considerable amount of polyphenols (27). Tea polyphenols have been found to be powerful antioxidants that may reduce LDL oxidation and the formation of oxidized DNA metabolites, thus contributing to lower risks of CVD and cancer (28).

Few studies have carefully examined the effects of tea consumption on body weight or EE. In fact, only 2 studies have examined the effect of tea consumption, as a beverage, on EE (29, 30). Rumpler et al (29) tested whether oolong tea increased EE or modulated substrate oxidation rates more than did control beverages. Four different beverages were consumed 5 times/d for 3 d each: full-strength oolong tea (3 g tea leaves in 300 mL water), half-strength oolong tea (1.5 g tea leaves in 300 mL water), water, and water + caffeine (caffeine content equivalent to that of full-strength tea). Each serving of full-strength oolong tea contained 48.7 mg EGCG and 53.7 mg caffeine, for a total intake of 244 mg EGCG and 270 mg caffeine. Servings were consumed every 1.5 h from 0830 to 1430 as part of a controlled diet providing 115% of energy requirements during the initial 2 d and 100% of energy requirements on the 3rd day. Twenty-four-hour EE with full-strength tea and water + caffeine consumption was significantly greater than that with water alone. The increase in EE with consumption of full-strength tea and for water + caffeine was, respectively, 2.9% [281 kJ (67 kcal)] and 3.4% [331 kJ (79 kcal)] greater than that for water. Fat oxidation increased by 12% relative to baseline for full-strength tea and by 8% for water + caffeine. However, only the consumption of full-strength tea resulted in significantly greater fat oxidation (13.1%) than did the consumption of water. The authors concluded that oolong tea stimulated EE and fat oxidation in normal-weight males and could have some beneficial effects on a person's ability to maintain lower body fat. However, they also cautioned that weight maintenance would be facilitated only if the effects of tea consumption on EE and fat oxidation were sustainable and if no dietary compensation occurred to offset the slight energy imbalance.

Another recent study examined the effects of oolong tea and green tea consumption on fasting EE (30). Eleven healthy normal-weight women were tested after drinking water and again after drinking oolong and green tea in random order. Oolong tea was prepared by brewing 15 g tea leaves in 300 mL water, and each serving contained 77 mg caffeine and 81 mg EGCG. Green tea was prepared by dissolving 5 g powdered green tea in 300 mL water. Each serving of green tea contained 161 mg caffeine and 156 mg EGCG. EE was measured by using the Douglas bag method both at baseline and for 2 h after beverage consumption. Resting EE was similar in the 2 groups before consumption of the different beverages and remained low after water and green tea consumption but increased significantly after oolong tea consumption. The cumulative increase in EE over resting EE after the consumption of oolong tea, green tea, and water was 110.7 (26.5 kcal), 49.5 (11.8 kcal), and 11.2 kJ (2.7 kcal), respectively, over the 2-h measuring period. Respiratory quotients did not differ between the 3 treatments. The authors concluded that, because oolong tea had less caffeine and EGCG than did green tea, the rise in EE must be due to the presence in oolong tea of more polymerized polyphenols than are found in green tea. However, the measurement period in this study was very short, and it is not known whether the increased EE would extend over the full postprandial period—typically 6–7 h—or whether increases would be observed on subsequent tea-drinking occasions.

Another study that examined the effects of tea on thermogenesis (31) used tea as a nutraceutical rather than a functional food. Nutraceuticals are components that are isolated or purified from a food or beverage, that have been shown to have health benefits or reduce the risk of chronic disease, and that are usually found

in capsular or nonfood format (32). Healthy young men underwent three 24-h testing periods in a metabolic chamber to examine whether 2 capsules of a green tea extract containing 50 mg caffeine and 90 mg EGCG, taken 3 times/d, stimulated thermogenesis to a greater extent than did caffeine alone or placebo. Twenty-four-hour EE with green tea extract treatment was greater than that with caffeine and placebo—9867 kJ (2360.5 kcal) and 9599 (2296.4 kcal) and 9538 kJ (2281.8 kcal), respectively—which corresponds to an increase of 2.8% and 3.5% [268 kJ (64.1 kcal) and 329 kJ (78.7 kcal), respectively] over the EE with caffeine and placebo, respectively. The 24-h respiratory quotient with green tea extract treatment was lower than that with caffeine and placebo—0.852 compared with 0.873 and 0.881, respectively—which is indicative of greater fat oxidation with green tea extract treatment. It was concluded that oral administration of a green tea extract stimulated thermogenesis and fat oxidation and, therefore, that green tea extract has the potential to influence body weight and body composition. However, the short-term nature of the study prevented the direct observation of the effect of green tea extract intake on body composition.

Dulloo et al (33) also confirmed the mechanism of action of the green tea extract in an *in vitro* experiment. This experiment showed that the green tea extract significantly increased the rate of intrascapular brown adipose tissue oxygen uptake, but caffeine alone did not. The authors concluded that the green tea extract was a more effective potentiator of sympathetically mediated thermogenesis than was caffeine alone. In humans, this green tea extract was also found to reduce body weight by 4.6% and waist circumference by 4.5% when 2 capsules were taken twice daily for 12 wk as part of a regular, self-selected diet (34). Each capsule contained 375 mg catechins, including 270 mg EGCG. However, this study did not include a control group or a report of the statistical procedures, and therefore clear conclusions cannot be drawn from these results.

To date, no study has established the potential of tea as a functional food for weight maintenance. The only studies that examined tea as a functional food found modest effects on EE and were of very short duration (29, 30). Whether these slight increases in EE and fat oxidation persist over a long period remains to be established. Therefore, more research is necessary to ascertain whether tea can be of assistance in better weight maintenance or in weight-loss programs and whether its effects are more than those exerted by its caffeine content. Moreover, differences between types of tea with respect to their effect on EE should be explored further. As yet, the quantity of tea that must be consumed to obtain an effect on body weight has not been established. A study in rats in which intraperitoneal injections of 100 mg EGCG/kg body weight for 7 d resulted in losses in body weight and fat mass found that plasma concentrations of EGCG after injection were 24, 2, 4, 1, and 1 $\mu\text{mol/L}$ at 0.5, 1, 2, 5, and 24 h, respectively (35). A concentration of 1 $\mu\text{mol/L}$ would be similar to that in a 70-kg person 1 h after drinking 6–12 200-mL servings of green tea.

Milk

Although a meta-analysis of calcium consumption and weight loss has not shown that calcium consumption is linked to greater loss of body weight (36), there is increasing evidence that dairy calcium may play a role in body weight regulation (37, 38). Recently, Heaney (39) reported the effects of calcium consumption on body weight and the rate of body weight change in a



longitudinal cohort of women. This study showed that predicted BMI decreased with an increased ratio of calcium to protein, so that a ratio of 10 predicted a BMI of 22.5, whereas a ratio of 20 predicted a BMI of 19.3. Calcium:protein of 9, corresponding to approximately the 25th percentile of the currently recommended intakes, predicted weight gain at midlife is 0.425 kg/y. The observed rate of weight gain was, however, 1 kg/y. If calcium:protein was 20, representative of the current recommendations, the predicted weight change would be -0.011 kg/y, and only 3.7% of women would be predicted to gain 1 kg/y. The author suggested that the prevalence of obesity could be decreased by 60–80% in women if their calcium intakes were at recommended amounts (39). These observations corroborate those of others (40, 41), who reported associations between calcium intakes and body weight. From the third National Health and Nutrition Examination Survey, it was found that the risk of obesity was 85% lower in those in the highest quartile of calcium intakes, after adjustment for age, sex, race, and EIs, than in those in the lowest quartile (41). Similarly, in the Coronary Artery Risk Development in Young Adults Study, dairy consumption was inversely associated with the prevalence of obesity (40), and Davies et al (42) reported that the odds ratio for overweight with calcium:protein below the median (≈ 12 mg/g) was 2.25.

Moreover, calcium intakes recently were reported to be negatively associated with fat mass in the Quebec Family Study (43). The authors of this study found that women who consumed < 600 mg calcium/d had greater body weight (82.3 kg compared with 69.8 and 65.0 kg for those consuming 600–1000 mg and > 1000 mg calcium/d, respectively), BMI (31.8 compared with 27.0 and 25.0, respectively), percentage fat mass (37.3% compared with 31.3% and 28.9%, respectively), absolute fat mass, waist circumference, and abdominal adipose tissue than did women who consumed greater amounts of calcium, even after adjustments for EI, percentage of energy consumed as fat, dietary protein intake, socioeconomic status, and age.

A potential mechanism of action of milk in the promotion of weight loss has already been put forth (44). It is proposed that intracellular calcium plays a role in adipocyte metabolism and that its concentrations are modulated by calcitrophic hormones. An increase in calcium intake in foods would down-regulate 1, 25-dihydroxyvitamin D, which would result in a decrease in the absorption of calcium into adipocytes and pancreatic islet cells (Figure 2). Within the adipocyte, intracellular calcium increases fatty acid synthase transcription and inhibits lipolysis. Within the pancreas, intracellular calcium stimulates insulin release, which further acts to inhibit lipolysis and stimulate fatty acid synthase transcription. Therefore, any reduction in intracellular calcium would lead to a reduction in lipogenesis and the stimulation of lipolysis (44). This is supported by recent data showing that acute dietary calcium intake was correlated positively with 24-h fat oxidation and negatively with the 24-h respiratory quotient (45). However, in a controlled feeding experiment in which subjects were given diets containing 500 or 1400 mg dairy calcium/d in a crossover design for 7 d each, EE and substrate oxidation did not differ significantly between diets (46). However, other compounds within dairy products may act in concert with dietary calcium to produce antiobesity effects. Such compounds that have been proposed are whey proteins (44), conjugated linoleic acid (47), and branched-chain amino acids (48). However, a recent study showed no effect of various conjugated linoleic acid isomers on body weight loss over a period of 18 wk (49).

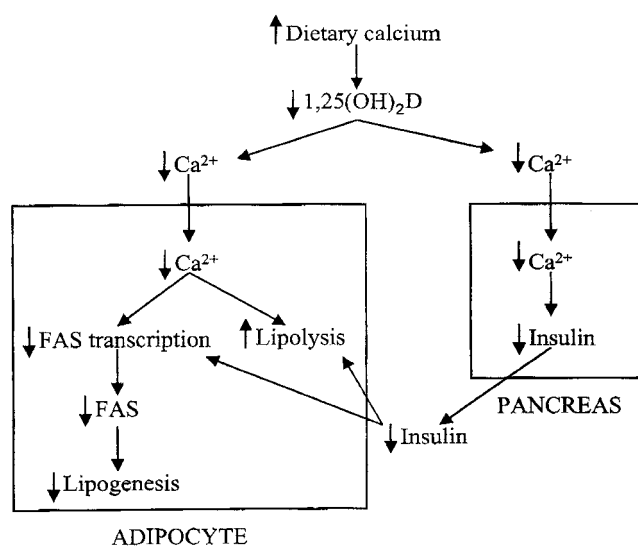


FIGURE 2. An increase in dietary calcium reduces 1,25-dihydroxyvitamin D [$1,25(\text{OH})_2\text{D}$] concentrations, resulting in down-regulation of calcium transfer into adipose and pancreatic cells. Inside adipocytes, a reduction in intracellular calcium leads to decreased fatty acid synthase (FAS) transcription that results in a lowering of lipogenesis and increased lipolysis. In pancreatic cells, reduced intracellular calcium concentrations decrease insulin output, which results in reduced lipogenesis and enhanced lipolysis in adipocytes. In combination, these processes would help reduce fat deposition into and storage in adipose tissue. Adapted with permission from *J Nutr* 2003;133:252S–6S (44).

Cross-sectional studies (40, 41, 43) have thus found a relation between milk or calcium consumption and body weight. Such cross-sectional studies, however, do not establish causal relations, and, therefore, whether the relation between milk consumption and body weight is due to other characteristics of milk consumers is not known. For example, adults who do not eat fast food drink more milk than do those who do eat fast food (50). In addition, milk may be replacing other, more energy-dense beverages in the diet. This, however, is debatable, because some find lower milk intakes in those who consume more soft drinks (51) and others find that soft drink intake is not associated with lower calcium intakes in children and adolescents (52). Furthermore, results have been ambiguous in clinical studies: some showed no effect of calcium (53, 54), and another showed a beneficial effect (55) on weight loss. One randomized clinical study recently examined the effects on bone turnover of protein-rich weight-loss diets that are high in dairy protein and calcium and in mixed protein sources (53). Diets were not strictly controlled, but each subject was given instructions on dietary requirements and was provided with a digital kitchen scale and foods to cover 60% of their total energy prescription. The entire study consisted of 2 phases, an energy-restriction phase of 12 wk and a subsequent energy-balance phase of 4 wk. The dairy protein diet contained 2371 mg calcium, compared with 509 mg calcium for the mixed protein diet, and 62% of its total protein content was from dairy products. Overall body weight loss was 10% during the energy-restriction phase, irrespective of dietary treatment. As planned, there was no weight loss during the energy-balance phase. Although the primary endpoint of interest in this study was not weight loss per se, these results show that, as part of a weight-loss diet, high dairy protein and calcium consumption does not lead to

greater weight loss than does a diet consisting of mixed protein sources and <600 mg calcium/d.

Similarly, in a study examining the effects of calcium supplementation in limiting bone loss during weight loss (54), there was no effect of calcium on body weight and fat mass loss. Obese postmenopausal and premenopausal women were randomly allocated to a weight-loss diet with a 2100 kJ/d (500 kcal/d) energy deficit with or without 1000 mg elemental calcium/d for 25 wk. Calcium supplements were provided as 2 pills to be taken at breakfast and dinner. The control group received placebo pills. Weight loss did not differ significantly between groups—6.2 and 7.0 kg for the placebo and the calcium supplement group, respectively. Accordingly, fat-mass loss also did not differ significantly between placebo and calcium groups—4.5 and 5.5 kg, respectively. However, this study was not powered to detect changes in body weight and fat mass between groups, and a post hoc power analysis showed that 500 subjects/group would have been necessary to detect a 0.8-kg difference in body weight, and 265 subjects/group would have been required to detect a 1.0-kg difference in fat mass between groups, with 80% power and 95% CIs. The authors thus proposed that the direction of change observed in this study suggests that, over longer periods and with an adequate number of subjects, calcium may have an effect on body weight loss. Moreover, this study provided calcium in its elemental form, and it may be that calcium from dairy products has a greater effect than does elemental calcium because of other components present in dairy foods. However, the results obtained by Bowen et al (53) suggest that this may not be the case.

A recent clinical trial aimed to ascertain whether dairy or elemental calcium supplementation enhanced weight loss in obese men and women (55). Subjects were examined after a 2-wk lead-in period and were then randomly assigned to 1 of 3 groups: group 1 consisted of control subjects who were restricting their caloric intake by 2100 kJ/d (500 kcal/d) and consuming 0–1 serving dairy products/d while taking a 400–500 mg calcium supplement and a placebo pill (low-dairy group); group 2 received the same dietary prescription as group 1, but the placebo pill was replaced by 800 mg calcium carbonate (high-calcium group); and group 3 had the same dietary prescription as group 1 but consumed 3 servings dairy products/d (high-dairy group). Diets were followed for 24 wk. All subjects initially consumed <1 serving dairy products/d. Of the 41 subjects enrolled, 32 completed the study; the data reported include completers only. At the end of the weight-loss period, subjects in the low-dairy, high-calcium, and high-dairy groups lost 6.4%, 8.6%, and 10.9% of body weight, respectively. Fat-mass loss followed the same trend: the low-dairy, high-calcium, and high-dairy groups lost 8.1%, 11.6%, and 14.1%, respectively, of total fat mass. Fat loss from the abdominal region represented 19% of the total fat loss in subjects in the low-dairy group and 50.1% and 66.2% for those in the high-calcium and high-dairy groups, respectively. These data show that calcium, particularly that from dairy products, can enhance weight loss in obese persons. However, only data from those who completed the study were analyzed, and thus results may not represent all subjects. Furthermore, it is not known whether similar results would be obtained in persons who regularly consume larger amounts of dairy products.

A potential mechanism has been postulated to explain a possible role of calcium in body weight control, and yet it remains unclear whether there is a weight-loss effect of calcium. Only one clinical study was specifically conducted to examine the role of

calcium as part of a weight-loss program, but data for all of the subjects initially enrolled in the study were not reported (55). Other clinical studies were post hoc analyses and were not specifically designed to test the hypothesis that calcium may play a role in body weight regulation (53, 54). More studies are therefore needed to draw definitive conclusions about the effects of calcium and dairy products on weight management. There is also some concern regarding a possible link between milk consumption and prostate cancer. A recent meta-analysis found an odds ratio of 1.68 for prostate cancer in subjects with high milk consumption when examining 11 case-control studies (56). However, other longitudinal studies have found either no association (57) or a weak positive association (58) between higher milk consumption and prostate cancer (odds ratio: 1.34).

NUTS

Nuts vary widely in caloric content and fat composition and have often been excluded from diets because of their high fat content (59). As a result, some concern existed with respect to the potential effects on body weight of incorporating nuts in the diet. However, many of the supplementation studies that have examined the effects of nuts on lipid profiles have not found negative effects on body weight (60–68), but they did show that nuts—whether almonds (61, 65, 68, 69), walnuts (60, 67, 68), pecans (63), pistachios (66), or peanuts (62, 64)—improve plasma lipid profiles and can have a beneficial effect on CVD risk. Because very few studies have specifically examined the effects of nuts on body weight (70, 71), those studies that examined the effects of nut consumption on plasma lipids and also reported body weight at baseline and endpoint (60–69) will be reviewed here. These studies were not designed to produce weight loss.

Studies examining the effect of almonds on plasma cholesterol concentrations have also reported body weight changes during the experimental and control feeding periods (61, 65, 69). In the study of Spiller et al (61), male and female subjects were examined after a 2-wk baseline period of no intervention and again after 9 wk of supplementation with 100 g almonds/d [2424.4 kJ/d (580 kcal/d)]; half of the almonds were whole blanched almonds, and the other half were ground almonds. Subjects were also provided with almond oil to replace other, normally used cooking fats, and they were asked to eliminate margarine, butter, vegetable oils, mayonnaise, most meats, shellfish, whole-fat dairy, high-fat bakery products, potato chips, ice cream, avocado, and all other nuts from their diet. Total fat intake increased from 67 to 90 g/d, and protein intake increased from 88 to 103 g/d, whereas carbohydrate intake decreased by 33 g/d. Although total EI, assessed from 3-d food records at baseline and at weeks 4 and 8 of the intervention period, increased by 338.6 kJ/d (81 kcal/d) over the study period, body weights did not change (74.9 kg at baseline and 74.3 kg at week 9). On the basis of the difference in daily EIs, a theoretical weight gain of 0.66 kg would have been expected over the 9-wk period. Moreover, if the subjects had not partially compensated for the added calories provided by the almonds, a weight gain of 10 kg would have been expected.

More recently, Jenkins et al (65) studied 3 supplements in a randomized crossover design: control muffin, full-dose almond, and half-dose muffin + half-dose almond. The subjects, who were hyperlipidemic men and women, consumed self-selected National Cholesterol Education Program Step 2 diets and were counseled to maintain a stable body weight. Depending on



each subject's energy requirements, full supplement doses provided 1200 (287 kcal/d), 1797.4 (430 kcal/d), and 2399.3 kJ/d (574 kcal/d) for total energy requirements of 6688 (1600 kcal/d), 6688–10 032 (1600–2400 kcal/d), and >10 032 kJ/d (>2400 kcal/d), respectively. Authors found a dose effect of almonds on CVD risk factors but no change in body weights over the 1-mo supplementation periods. In this study, the lack of body weight change with all supplements may have been due to good compliance with counseled weight-management strategies and not necessarily to a satiating effect of the almonds. Nevertheless, food records during the supplementation periods indicated an energy intake during the full-dose almond phase that was 560.1 kJ/d (134 kcal/d) more than that during the full-dose muffin phase. This greater caloric intake should theoretically have led to a weight gain during the full-dose almond phase that was 0.5 kg more than the gain during the full-dose muffin phase. Therefore, assuming consistent reporting of dietary intakes during all phases of this study, there may be some unabsorbable energy in almonds that negated the energy imbalance caused by the greater intakes.

Another supplementation study, however, found slight but significant body weight gains when men and premenopausal women supplemented their diets with 100 g almonds/d for 4 wk (69). This slight weight gain of 0.9 kg for men and 0.3 kg for women occurred despite recommendations to reduce EIs by an amount equivalent to that provided by the almonds: ≈ 2424.4 kJ/d (580 kcal/d).

One study examined the effect of walnut consumption on plasma lipid concentrations in hypercholesterolemic men and women (60). Two different diets were tested in a randomized crossover design for 6 mo each. During both phases, subjects were instructed to consume a Mediterranean diet of prescribed energy content, which emphasized vegetable products and fish and limited red meat and eggs. For the control phase, olive oil was advised for cooking, and nuts were not allowed. During the walnut phase, subjects consumed 41–56 g walnuts/d in partial replacement of the olive oil and other fatty foods in the control diet. Despite a trend toward greater EIs during the walnut phase than during the control phase [$\bar{x} \pm \text{SD}$: 7624.3 \pm 744 kJ/d (1824 \pm 178 kcal/d) and 7402.8 \pm 635.4 kJ/d (1771 \pm 152 kcal/d), respectively; $P = 0.11$], body weights remained the same during both phases (69.9 and 70.1 kg for walnut and control phases, respectively). The difference in EIs between diets theoretically should have led to a weight gain of 1.2 kg over the 6-mo period.

Another study examined the effect of walnut consumption on plasma lipid concentrations by feeding a habitual diet and a low-fat, free-living diet (67). Men and postmenopausal women consumed their habitual diets for 4 wk and then supplemented those diets with 48 g walnuts/d for 6 wk; they then followed a low-fat (20% of energy) diet for 6 wk and, finally, a low-fat + walnut diet for 6 wk. The walnut dose provided 1570 kJ/d (375.6 kcal/d). EIs during the walnut phases exceeded those during the walnut-free phases by 1651 kJ/d (395 kcal/d) for the habitual diet and 1514 kJ/d (362.2 kcal/d) for the low-fat diet. Regardless of the additional EIs during the walnut supplementation phases, body weights did not increase during the walnut phases.

Similar observations were made when subjects consumed self-selected 8-wk diets with 68 g pecans/d or no nuts (63). Pecan supplementation provided an extra 1918.6 kJ/d (459 kcal/d) and 44 g fat/d. As was observed with walnuts (67), neither body

weights nor BMI changed during the supplementation period, despite an increase in EIs and fat intakes.

In a study of pistachio nuts, men and women maintained their regular diets for 3 wk and replaced 20% of their energy intakes with pistachio nuts for the subsequent 3 wk (66). As assessed by food records, subjects consumed the same number of calories and macronutrients during the 2 phases. As expected, there was no significant change in body weight during the study, which showed good compliance with the dietary replacement protocol. Although of short duration, this study shows that subjects can successfully substitute pistachio nuts for other foods in a regular, free-living diet without increasing their body weights.

All of the above-mentioned studies except that of Lovejoy et al (69) found no weight gain with nut consumption, despite increases in EIs. If we assume adequate reporting of EIs, this absence of body weight gain may have been due to some degree of malabsorption of energy in nuts or to an increase in EE with nut consumption. The absence of body weight gain with nut supplementation led to studies that examined the effects of nut consumption on body weight and energy balance (70, 71).

The study by Fraser et al (71) tested the effect of consuming 1338 kJ almonds/d (320 kcal/d) for 6 mo on body weight in men and women ranging in age from 25 to 70 y and with a BMI <95th percentile. The almond supplement provided 15% of daily energy requirements for each person, and no dietary advice or recipes were provided. Subjects could incorporate the nuts as they wished. There was a nonsignificant weight gain of 0.4 kg for the group overall, but, when subjects were separated by sex, the men gained 0.65 kg ($P < 0.01$), and the women gained only 0.11 kg ($P = 0.79$). When the sexes were studied together, only persons in the lowest and middle tertiles of BMI gained weight. The estimated compensation for total EI was 78.2%. This study showed that incorporating nuts in a regular, free-living diet does not lead to weight gain; however, no concurrent group without the almond supplement was studied during that same period. It is therefore not known whether study participation had an influence on compliance or whether it helped improve dietary compensation for the added energy.

Another study examined the effects of peanut consumption on energy balance and the hedonic ratings for peanuts and other snack foods (70). Normal-weight men and women were given ≈ 89 g peanuts/d, which is equivalent to 2113 kJ/d (505 kcal/d), to consume as they wished for 8 wk (free-feeding phase); this phase was followed by a 3-wk phase during which the peanuts were added to the baseline diet (addition phase) and an 8-wk phase during which peanuts replaced fat in the diet (substitution phase). The phases of the study were separated by 4-wk washout periods. During the free-feeding phase, mean energy compensation was 66%, and the observed weight gain was 1.0 kg, which is significantly lower than the theoretical, expected weight gain of 3.6 kg. Weight gain (0.6 kg) was also significantly lower during the addition phase than predicted (1.4 kg), and there was no change in body weight during the substitution phase. Resting EE increased by 11% after 19 wk of regular peanut consumption, even after adjustment for changes in body weight. These results show that nut consumption may have a small effect on EE, which may partly explain the lower-than-expected weight gain observed in the free-feeding and addition phases.


Finally, results from nut supplementation studies do not imply that nut consumption can assist in weight loss, and there are no definitive data on their ability to assist in the maintenance of



stable weight. Nevertheless, their satiating power may play a role in weight maintenance. In addition, the Nurses' Health Study (72) and the Seventh Day Adventist Study (73) found lower body weights with increased nut consumption. However, these cross-sectional studies may also reflect a healthier lifestyle pattern associated with nut consumption. Weight-loss studies incorporating nuts should be conducted to ascertain whether nuts may assist in weight loss. At present, available research allows us only to speculate that nuts may assist in controlling body weight, perhaps via increased satiety levels, increased resting EE, or energy malabsorption.

CONCLUSION

The studies reviewed here show that some foods have the potential to enhance weight loss or prevent weight gain. The body of literature seems to provide more support for the replacement of dietary LCTs with dietary MCTs or diacylglycerols than for the inclusion of other potential functional foods as part of the diet. However, available data suggest that beverages such as tea and milk may also be of value. Additional clinical studies are needed to ascertain whether tea and milk consumption can help improve body weight maintenance and perhaps also assist in weight loss. More research is also necessary to reach conclusions about the effects of nut consumption on body weight, and weight-loss studies with a control group are necessary to determine with greater accuracy the possible effects of all of the potential functional foods reviewed here. Such studies would, however, require a very large sample size because the effect size is expected to be small.

As yet, no weight-loss claim can be made for any of the foods examined here. Nevertheless, the data do suggest that incorporating some of these foods in a healthy and balanced diet could be beneficial for weight maintenance. Perhaps the individual effect of each dietary component on weight control might be too small to result in meaningful body-composition changes, but, if the dietary components were combined, their effects could be significant. It is nonetheless important that these measures be combined with energy restriction and increased physical activity to achieve significant weight loss. 

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