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PAPER

Effects of chronic peanut consumption on energy balance and hedonics

CM Alper[1] and RD Mattes[1]*

[1]*Purdue University, Department of Foods and Nutrition, West-Lafayette, Indiana, USA*

OBJECTIVE: To investigate the effects of chronic peanut consumption on energy balance and hedonics.
DESIGN: Thirty-week, cross-over, intervention study. Participants were provided 2113 ± 494 kJ/day (505 ± 118 kcal/day) as peanuts for 8 weeks with no dietary guidance (free feeding — FF), 3 weeks with instructions to add peanuts to their customary diet (addition — ADD) and 8 weeks where peanuts replaced an equal amount of other fats in the diet (substitution — SUB).
SUBJECTS: Fifteen, healthy, normal-weight (BMI of 23.3 ± 1.8) adults, aged 33 ± 9 y.
MEASUREMENTS: Dietary intake, appetitive indices, energy expenditure, body weight and hedonics.
RESULTS: During FF, peanut consumption elicited a strong compensatory dietary response (ie subjects compensated for 66% of the energy provided by the nuts) and body weight gain (1.0 kg) was significantly lower than predicted (3.6 kg; $P < 0.01$). When customary dietary fat was replaced with the energy from peanuts, energy intake, as well as body weight, were maintained precisely. Participants were unaware that body weight was a research focus. Resting energy expenditure was increased by 11% after regular peanut consumption for 19 weeks ($P < 0.01$). Chronic consumption of peanuts did not lead to a decline in pleasantness or hunger ratings for peanuts nor did it lead to any hedonic shift for selected snack foods with other taste qualities during any of the three treatments.
CONCLUSIONS: Despite being energy dense, peanuts have a high satiety value and chronic ingestion evokes strong dietary compensation and little change in energy balance.
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Keywords: peanuts; energy balance; body weight; hedonics

Introduction

Diets containing nuts reduce coronary heart disease risk[1–3] predominantly through their lipid-lowering effect.[4] Yet, nuts are often avoided due to their high fat and energy content. In contrast to this concern, epidemiological evidence reveals a negative association between nut consumption and body weight.[1,2] Further, several experimental human feeding studies, involving a variety of nuts, have shown no change or reductions in body weight among nut consumers.[5–8]

In the United States, peanut (a legume) consumption is 2.6 kg/capita, roughly 2.5 times greater than all tree nuts combined (1 kg/capita).[9] However, there is limited quantitative data on the effects of peanut consumption on energy balance. The reported inverse association with body weight or body mass index (BMI) may be attributable to several mechanisms. First, peanuts hold strong satiety effects. They are energy dense, but are also rich sources of fiber and protein, both of which enhance satiety.[10–12] In an acute preload study, peanuts exerted a strong suppression of hunger and energy compensation.[12]

Second, earlier work demonstrated that whole peanuts are inefficiently absorbed.[13] When subjects were fed whole peanuts, 17.8% of dietary fat was excreted in the stool. In a chronic feeding study investigating the effects of high oleic peanuts on blood lipids, weight loss was observed, despite participants being asked to maintain their weight and activity level. They also had energy intakes comparable to a control group.[5] One explanation may involve poor absorption.

Third, peanuts may enhance energy expenditure. The highly unsaturated fatty acid composition of peanuts[14] and their protein content[15] may promote increased energy expenditure. Unsaturated fatty acids are preferentially

*Correspondence: RD Mattes, Professor of Foods and Nutrition, Purdue University, Department of Foods and Nutrition, 1264 Stone Hall, Room 212, West Lafayette, IN 47907-1264, USA.
E-mail: mattesr@cfs.purdue.edu
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oxidized compared to saturated fatty acids.[16] In rats, oxygen consumption is significantly greater on a high safflower oil diet compared to a high beef tallow diet.[17] In humans, a high dietary polyunsaturated to saturated dietary fatty acid ratio can increase resting energy expenditure (REE) and diet-induced thermogenesis (DIT).[14]

To our knowledge, no study has directly investigated the effects of chronic peanut consumption on satiety, energy expenditure and body weight. The present study assessed the effects of daily peanut consumption on appetitive measures, dietary intake, body weight, REE and DIT in free-living adults enrolled in a three-arm cross-over study. The three treatments entailed adding peanuts to the customary diet, substituting peanuts for fat in the diet and providing the peanuts with no dietary guidance.

Given the accumulating evidence of health promoting properties of nuts (peanuts), the question of whether their sensory appeal is maintained with daily ingestion warrants consideration. Repeated exposure to novel foods increases pleasantness ratings[18] while inconsistent responses have been noted with familiar foods.[19] This may depend, in part, on initial palatability or type of food.[19–21] Repeated consumption of a salty, high-fat snack (fries) did not diminish pleasantness ratings while a decline was noted after regular exposure to a sweet high fat snack (chocolate).[20] Thus, regular peanut consumption may lead to decreased acceptance due to monotony or, alternatively, hedonics may be preserved due to high initial acceptance or sensory profile. This study examined the effects of chronic peanut consumption on hedonic ratings for peanuts and other snack foods varying in their predominant taste qualities.

Subjects and methods

Subjects

Fifteen healthy adults (seven female and eight male), age 33 ± 9 y (mean ± s.d.), using no prescription medications reported to influence study variables were recruited by public advertisement. Subjects were of normal body weight (mean BMI 23 ± 1.8, body fat 24.3 ± 8.5%) and had no recent history of weight change exceeding 2.3 kg (5 lb) within the prior 3 months. All were non-smokers and unrestrained eaters (score < 14 on the three factor eating questionnaire[22]) and were required to control the purchase and preparation of the majority of foods they consumed.

The study protocol was approved by the Committee on the Use of Human Research Subjects at Purdue University.

Experimental design

Three treatment arms were sequentially assigned to ensure representation of all possible treatment orders. A schematic diagram of the study design is presented in Figure 1. The free-feeding (FF) arm was an 8 week trial where 50% of dietary fat energy, based on customary energy and fat intake, was provided to participants with instructions to consume peanuts daily at any time and in any manner they chose while the background diet was not controlled. No other dietary advice was provided. The addition (ADD) arm was a 3 week trial where 50% of dietary fat energy supplied by peanuts was added to a prescribed diet isocaloric to each participant's estimated customary energy and fat intake. Energy intake was estimated from REE measurements and physical activity level. Fat intake was estimated from a food frequency questionnaire. The substitution (SUB) arm was an 8 week trial where participants decreased fat intake by 50% and this was replaced with an equivalent amount of fat from peanuts. Washout periods between each arm of the study were 4 weeks.

Diets

During ADD and SUB, each subject received an individualized meal plan and an exchange booklet as a reference manual that specified portion sizes for exchanges. Diets were prescribed according to the American Dietetic Association Exchange list system.[23] The ADD diet supplied 34% energy from fat, 49% from carbohydrate and 17% from protein, yielding 41% energy from fat after including the test peanuts. The SUB diet required subjects to follow a low-fat diet based on 17% energy from fat, 49% from carbohydrate and 17% from protein, yielding 34% energy from fat after inclusion of the fat from peanuts. Provision of peanuts amounted to an average of 89 ± 21 g/day or 2113 ± 494 kJ/day (505 ± 118 kcal/day) for all arms.

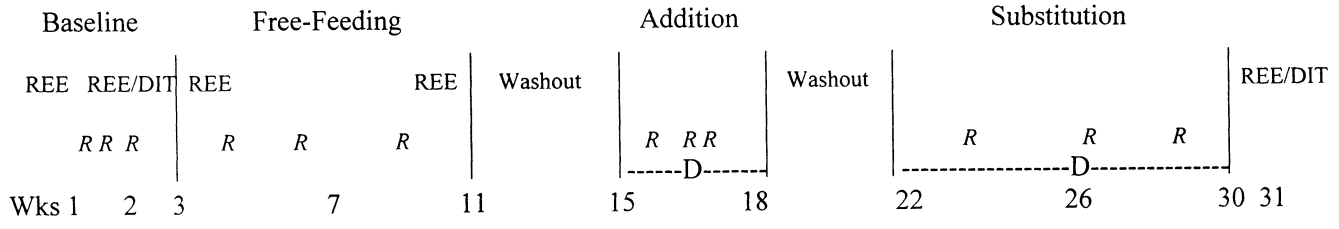

Figure 1 Schematic diagram of study design. Only one of six possible treatment orders is shown. Diet prescription occurred during addition and substitution (D). Diet recalls (R) were performed unannounced on random days on three separate occasions during baseline, free feeding, addition and substitution. Resting energy expenditure (REE) was measured at baseline, before and after free feeding and prior to each diet-induced thermogenesis (DIT) measurement. DIT was measured continuously for 4 h after subjects consumed 300 kcal of peanuts with 4 oz of water.

Dietary assessment

Dietary intake was assessed by random 24 h recall telephone interviews. During baseline, participants were trained to estimate food portions with plastic food models and measuring cups for one week at home. Participants were called unannounced on three separate occasions during baseline and each treatment period. Adequacy of reported energy intake was evaluated according to the Goldberg cut-off limits for under-reporting (energy intake/measured REE below 0.92 for individuals).[24]

Anthropometric measurements

Body weight was measured in the fasted state with participants in a hospital gown at weeks 1, 2, 3, 7, 11, 15, 18, 22, 26, 30 using a clinical scale (Scale-Tronix, White Plains, NY, USA). BMI was calculated as the ratio of body weight to body height squared (kg/m^2). Dual-energy X-ray absorptiometry (DEXA) was used to measure body composition at baseline as well as pre and post FF.

Energy expenditure

REE and DIT were measured by indirect calorimetry using a metabolic cart (Sensormedics Vmax 29, SensorMedics Corporation, Yorba Linda, CA, USA) and a ventilated respiratory canopy. Analyzers were calibrated with room air and standard calibration gas mixtures (4% CO_2, 16% O_2, balance N_2 and 0% CO_2, 26% O_2, balance N_2). Energy expenditure was calculated based on the Weir equation (REE kcal/day = (3.94 (VO_2) + 1.106 (V CO_2) 1440)[25]). Participants were instructed to refrain from exercise the day before their appointment and to minimize activity in the morning of the measurement. After an overnight fast, subjects arrived in the laboratory and rested for at least 10 min on a hospital bed. REE measurements were performed in the supine position for approximately 45 min at baseline, before and after FF and prior to each DIT measurement (Figure 1). Data were recorded in 30 s intervals and averaged over the last 10 min of each REE measurement. Subjects were required to stay awake and minimize motion for the duration of the measurement. An individual activity factor[26] was applied to measured REE to determine daily energy requirements. DIT was measured during baseline and at the end of the entire 19 week trial under the same conditions as REE. Immediately after an REE measurement, subjects consumed 52.5 g (300 kcal) of peanuts with 4 oz of water within 10 min. This was followed by continuous gas exchange measurement for 4 h. Data were averaged at 30, 60, 90, 120, 150, 180, 210 and 240 min and were also expressed in absolute terms (kJ/240 min).

Physical activity assessment

Subjects completed a 7 day physical activity questionnaire during baseline as well as before and after each treatment.[27] Participants recorded information about time spent sleeping and the intensity and duration of their physical activities by comparing their own activities to a provided list of activities at specified levels of intensity. Total energy expenditure was calculated from hours spent in sleep and in moderate, hard and very hard intensity physical activities. Time spent in light activities was obtained by subtraction.

Appetitive ratings

Subjects completed a hunger questionnaire every hour they were awake for 24 h before and after each treatment. The day of the week that participants completed the questionnaire was held constant for that individual throughout the study. Responses for hunger were recorded on a nine-point category scale with anchors of 1 = not at all hungry and 9 = extremely hungry. Desire to eat, fullness and prospective consumption were rated on comparable scales.

Hedonic assessment

Commercially available snack foods, including peanuts, presented in random order were rated for pleasantness on a nine-point category scale (1 = dislike extremely, 9 = like extremely) by all subjects before and after each treatment. Foods represented different predominant taste qualities (eg

Table 1 Commercially available snack foods rated for pleasantness, frequency and preferred frequency, amount and preferred amount of consumption before and after each treatment

Salty	*Sweet*	*Sour*	*Bitter*	*High fat (> 10 g fat/serving)*	*Low fat (< 3 g fat/serving)*
Corn chips and salsa	Chocolate/candy bar	Lemon drops	Black licorice	Almonds	Dates/raisins
Crackers	Cookies	Pickles		Cashew nuts	Fresh fruit
Peanuts	Dates/raisins			Chocolate/candy bar	Jelly beans
Pistachio nuts	Doughnuts			Doughnuts	Marshmallows
Popcorn	Fresh fruit			Ice cream	Pretzels
Potato chips	Frozen yogurt			Macadamia nuts	Rice cakes
Pretzels	Ice cream			Peanuts	
Vegetables and dip	Jelly beans			Pecans	
	Marshmallows			Pistachio nuts	
				Potato chips	

salty, sweet, sour, bitter) as well as low-fat (< 3 g fat/serving) and high-fat categories (> 10 g fat/serving; Table 1). To monitor changes in acceptability of these foods, subjects also recorded actual as well as preferred frequency (not at all to more than once a day) and amount of consumption.

Compliance

Erythrocyte membrane fatty acid analysis. Peanut consumption was monitored by comparing erythrocyte membrane fatty acid composition at baseline and after the 8 week SUB treatment (where diet was most tightly controlled) in six participants administered this treatment first (to avoid carryover effects). A 5 ml fasting blood sample was collected into vacutainers with EDTA, the erythrocyte portion was separated from plasma by centrifugation and frozen at -40°C. Erythrocyte membranes were prepared by hemolyzing the cells two times in deionized distilled water followed by centrifugation at 3000 *g* for 10 min at 4°C. The method of Lepage and Roy[28] was used to methylate the fatty acids. Fatty acid composition was determined by gas liquid chromatography using a 50 m capillary column with 0.25 mm inner diameter (CP-Sil 88, Varian Analytical Inst., Walnut Creek, CA, USA). The temperature of the oven was 150°C for 8 min and rose at the rate of 4°C/min to reach a final temperature of 200°C until the analysis was completed. The temperature of the injection port and the flame ionization detector was 300°C. Nitrogen was used as the carrier gas. Peaks were identified relative to authentic standards obtained from Supelco (Bellfonte, PA, USA) and Nu-Check-Prep (Elysian, MN, USA). The areas under the peaks were measured by integration (Shinadzu, Columbia, MD, USA). Data are expressed as percentages of total fatty acids.

To improve compliance, subjects were required to provide an unstimulated, 3 min saliva sample each time they visited the laboratory. They were informed that after completion of the study, all samples would be analyzed for a compound in the peanuts what would indicate their level of protocol compliance. For each sample indicating non-compliance, 5% of the total payment would be deducted when, in fact, such an analysis is not available and did not take place.

Statistics

Treatment effects were tested by one-way and two-way repeated measures analysis of variance (ANOVA). *Post hoc* analyses were performed with paired *t*-tests. The Wilcoxon matched pairs test for non-parametric data was applied to analyze categorical data from hunger and food preference questionnaires. Reliability of the hunger questionnaire was assessed by Cronbach's α. Percentage dietary energy compensation during FF was calculated as ((baseline energy intake + 2135 kJ) − (FF energy intake)/2135 kJ)×100. The criterion for statistical significance was set at $P < 0.05$. The Bonferroni correction for multiple comparisons was applied for the hunger and food preference questionnaire analyses. Statistical analyses were performed with the SPSS software package, release 10.0.5 (SPSS Inc. Chicago, IL, USA).

Results

Compliance

Mean percentages of erythrocyte membrane fatty acids are presented in Figure 2. SFA decreased significantly from 41.0% at baseline to 36.4% at the end of SUB ($P < 0.05$, $n = 6$). During the same time period, the mean percentage of PUFA increased significantly from 33.5 to 40.6% ($P < 0.05$, $n = 6$). Differences in MUFA were not statistically significant. The ratio of unsaturated fatty acids to SFA increased significantly from 1.45 to 1.76 ($P < 0.05$, $n = 6$). Observed *vs* prescribed energy compensation for the energy intake supplied by the nuts was 56 *vs* 0% during ADD and 98 *vs* 100% during SUB.

Dietary intakes

Evaluation of adequacy of reported energy intake did not reveal underreporting. Mean daily energy and macronutrient intakes are presented in Table 2.[29] During FF, total daily energy intake was significantly lower than predicted ($P < 0.05$). The mean energy compensation score was 66%. Eleven of 15 subjects reduced their free-feeding daily energy intake at least by 1255 kJ (300 kcal) to accommodate the additional energy provided by the test peanuts.

Body weight

Mean body weight changes are shown in Figure 3. Theoretical weight gain, ie without dietary compensation was 3.6 kg over the 8 week FF study period. This is based on the

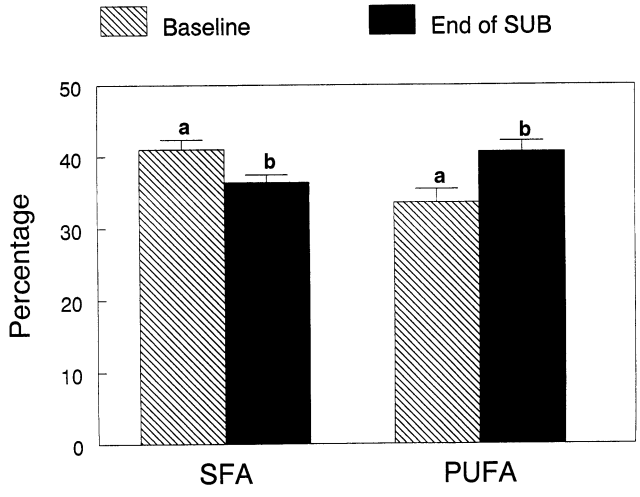

Figure 2 Mean percentage (± s.e.m.) of erythrocyte membrane fatty acids of six healthy subjects who consumed 500 kcal of peanuts daily over 8 weeks on a low-fat diet (Substitution). Means with different letters are significantly different ($P < 0.05$).

Table 2 Mean daily nutrient intakes from three random-day 24 h recalls[a]

	Baseline	*Free feeding*	*Addition*	*Substitution*
Energy (kJ/day)	9570±650	10 300±680	10 490±700	9600±640
(kcal/day)	2290±150	2460±160	2510±170	2300±150
Fat (% energy)	30.8±1.9[a]	38.8±1.2[b,c]	38.8±1.6[b,c]	34.9±1.6[b,d]
SFA	10.6±0.9[a]	10.3±0.6[a]	9.9±0.6[a]	8.13±0.5[b]
MUFA	9.2±0.8[a]	13.4±0.7[b]	14.7±1.1[b]	12.9±1.6[b]
PUFA	4.5±0.4[a]	7.9±0.5[b]	8.4±0.6[b]	8.8±0.5[b]
NFA lipid material	6.4±1.1	7.1±1.0	5.7±1.0	5.0±0.6
Cholesterol (mg/day)	196.8±23.6	177.6±23.3	213.1±23.1	173.3±22.6
Protein (% energy)	13.7±0.8[a]	15.1±0.7[c]	17.0±0.4[b,d]	16.6±0.6[b,d]
CHO (% energy)	55.8±2.1[a]	48.1±1.4[b]	46.9±1.7[b,c]	51.0±1.4[b,d]
Dietary fiber, g/day	18.37±2.2[a]	26.29±2.6[b]	29.08±2.4[b]	28.4±2.7[b]
α-TE (mg/day)	5.87±0.8[a]	12.59±1.0[b,c]	15.58±1.9[b]	15.31±1.4[b,d]
Folate (μg/day)	318.5±27[a]	458.8±39[b,c]	565.2±50[b,d]	563.15±46[b,d]
Magnesium (mg/day)	260.1±29[a]	387.8±34[b]	433.7±34[b]	408.2±36[b]
Copper (mg/day)	1.06±0.13[a]	1.88±0.16[b,c]	2.17±0.18[b,d]	2.02±0.17[b]

[a]Values are mean±s.e.m.; $n=15$. Means with different letters are statistically significant ($P<0.05$). SFA, saturated fatty acids; MUFA, monounsaturated fatty acids; PUFA, polyunsaturated fatty acids; NFA, non-fatty acid lipid material such as glycerol or sterols;[29] CHO, carbohydrate; α-TE, alpha tocopherol equivalents.

assumption that a daily mean energy surplus of 500 kcal yields an increment of 3500 kcal per week that equates to a 0.45 kg increase in body weight. Observed body weight gain (1.0 kg) was significantly lower than predicted from pretreatment to week 8 ($P<0.01$). There was no significant change in body weight from pretreatment to week 4, however, a significant increase of 0.8 kg was observed from week 4 to week 8 ($P<0.05$) during FF. Mean percentage of body fat increased from 24.6 to 25.2% from pretreatment to week 8 during FF, but this change was not statistically significant. During ADD observed body weight gain (0.6 kg) was significantly lower than predicted (1.4 kg; $P<0.05$), yet increased significantly from pretreatment to week 3 ($P<0.05$). There was no change in body weight during SUB.

Energy expenditure

Figure 4 contains DIT data before incorporation of peanuts into the diet and after regular peanut consumption for 19 weeks. Two-way repeated measures ANOVA revealed no significant treatment or time effect for DIT. REE (Figure 5) was significantly greater by 11% after regular peanut consumption for 19 weeks compared to baseline ($P<0.01$). This difference persisted after adjustment for body weight ($P<0.01$).

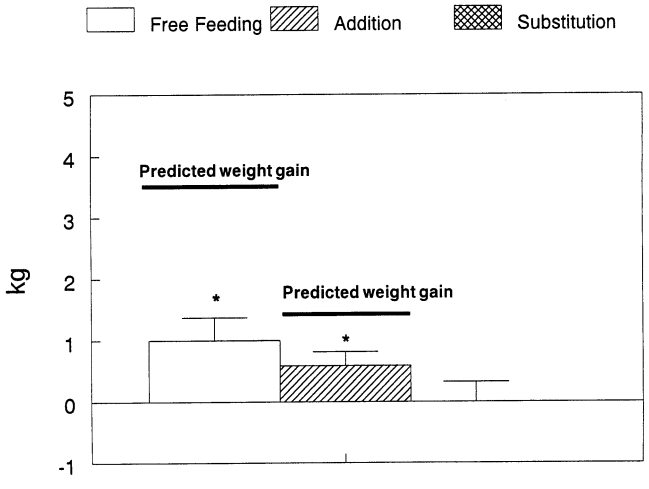

Figure 3 Mean changes (±s.e.m.) in body weight of 15 healthy subjects who consumed 500 kcal of peanuts daily over 8 weeks without restriction of the customary diet (Free-Feeding), over 3 weeks on an isocaloric diet (Addition) and over 8 weeks on a low-fat diet (Substitution). $P<0.05$.

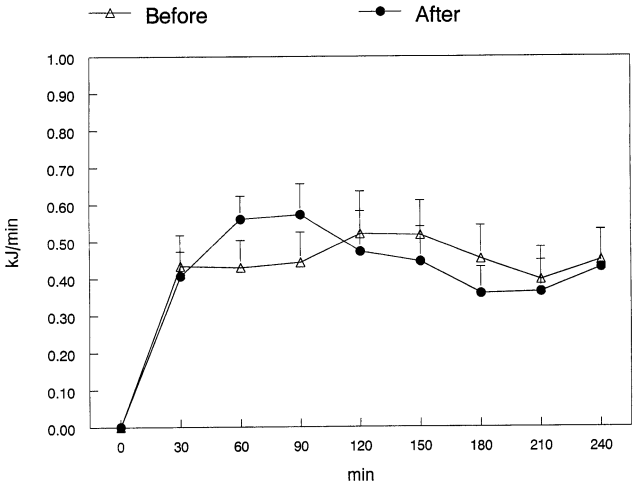

Figure 4 Diet-induced thermogenesis before incorporation of peanuts into the diet and after regular peanut consumption for 19 weeks ($n=15$). The test meal contained 52.5 g (300 kcal) of peanuts.

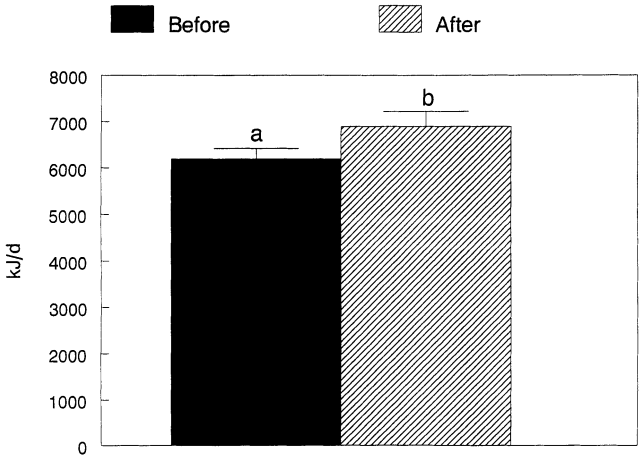

Figure 5 Resting energy expenditure before incorporation of peanuts into the diet and after regular peanut consumption for 19 weeks excluding wash-out periods ($n=15$). Means with different letters are statistically significant ($P<0.05$).

Physical activity

Estimated energy expenditure from 7-day physical activity recalls was not significantly different during FF, ADD and SUB (data not shown).

Appetitive ratings

There was no significant difference in mean hunger ratings during FF, ADD and SUB. Mean desire to eat, mean prospective consumption and mean fullness ratings also did not differ significantly during any treatment arm. The Cronbach's alpha reliability coefficient was 0.89 for hunger and desire to eat, 0.72 for hunger and prospective consumption and -0.76 for hunger and fullness ratings indicating good inter-item consistency, ie reliability of the measure.

Hedonics

There were no significant differences in food preferences, actual and preferred frequency of consumption or actual and preferred amounts of snack foods characterized as predominantly sour, bitter, sweet, salty, high-fat or low-fat during any treatment arm. As expected, the frequency and amount of peanut consumption increased significantly during FF, ADD and SUB (all $P \leq 0.004$).

Discussion

The results of the erythrocyte membrane fatty acid analysis confirm adherence to the dietary intervention. The utility of this method has previously been established.[6] In this study, both MUFA and PUFA were increased in the diet, thus no change in the ratio of these fatty acids was expected. Yet, an increased ratio of unsaturated to SFA in erythrocyte membranes was predicted and observed. The lack of an independent increase in MUFA may be due to the high baseline erythrocyte MUFA concentrations[30] in our subjects. Additionally, fasting serum magnesium concentrations (data reported elsewhere) increased significantly in 13 out of 15 participants during FF, reflecting dietary changes and indicating good compliance with the dietary intervention.[31]

During ADD, subjects reported difficulties with consumption of the prescribed diets and spontaneously commented on the high satiety value of the nuts. Thus, compared to baseline, energy intake of the background diet was reduced and energy addition was only 56% of the planned amount. During SUB, dietary compliance was nearly complete (98%) with respect to total energy intake. The intent was to replace 50% of customary dietary fat with the fat intake from peanuts. In practice, our free-living subjects increased their energy intake from fat by about 4% which was associated with a decrease in carbohydrate intake.

The Nurses Health study noted that women who ate nuts more frequently were leaner.[2] In a more homogenous group (with respect to education, health beliefs and diet) of Seventh Day Adventists, a statistically significant negative association was found between nut consumption and BMI.[1] Our findings do not indicate peanut consumption promotes weight loss, but they also pose limited risk for weight gain. In the FF arm, daily provision of 2100 kJ (500 kcal) peanuts over 8 weeks, without dietary restrictions, resulted in significantly lower body weight gain than predicted. In contrast to a theoretical body weight gain of 3.6 kg, weight remained very stable during the first 4 weeks of the experimental period (0.2 kg gain) followed by an average gain of 0.8 kg during the second 4 weeks. Whether the strong compensatory response will hold over longer time frames remains to be established.

The large caloric challenge (roughly twice the serving recommended by the food guide pyramid) was provided to maximize detection of treatment effects and may have made compensation especially difficult. Body weight changes in this study are also consistent with a chronic feeding study using a daily almond supplement of 2 oz (340 kcal). When no dietary advice was given to participants, mean body weight gain (0.4 kg) was less than predicted (1.1 kg) over 6 months.[32] As in our study, participants were unaware that body weight was a research focus.

The findings of this study suggest that dietary compensation is primarily responsible for the lack of predicted weight gain. Findings from a meta-analysis indicate there is a stronger compensatory dietary response for solid foods compared to liquids.[33] For solid foods, the mean compensatory score was 64%. Consistent with this finding, the present study observed a value of 66% for the peanuts. A compensation score of 30% was reported from an 8 week chronic feeding study that provided a daily caloric challenge comparable to that used here, but with pecans.[8] However, that study used self-recorded food records and noted an increase in energy intake of 2017 kJ (482 kcal) from baseline to week 2 while body weight decreased by 1 kg. Assuming there was no

marked change in energy expenditure, this raises questions about the self-reported intake values.

Strong dietary compensation may be due to the satiety inducing property of peanuts conveyed by their fiber, protein and energy content, as well as their solid food form. In an acute setting, peanut consumption has been reported to induce a strong satiety effect.[12] In the present study, during ADD, additional energy intake was only 44% of the prescribed level due, in part, to participant complaints that the peanuts were too filling. This experimental complication is not solely attributable to the energy value of the nuts. In another study, participants freely added a 1883 kJ (450 kcal) liquid supplement daily for 28 days without adjusting their self-selected background diet or complaints about satiety.[34] Cognitive influences could also contribute. Participants were non-restrained eaters, but may have consciously choosen to reduce their food intake to accommodate the additional energy provided by the peanuts.

One proposed mechanism underlying the relationship between nut consumption and negative energy balance is lipid malabsorption. The findings of this study do not support such a phenomenon from the peanuts. If lipids were poorly absorbed, participants should have lost weight during the isocaloric SUB condition. This was not the case. In fact, subjects precisely maintained their weight during SUB. Further, during ADD, the energy surplus was about 19330 kJ (4620 kcal) over the 3 weeks relative to baseline intakes. This translates to a theoretical increase in body weight of 0.6 kg. That change was in fact observed. Consistent with our findings another study reported when participants were fed 2261 kJ (636 kcal) of almonds daily for 4 weeks, substituting customary dietary fat with the fat from almonds, energy intake and body weight remained stable.[7]

A recent study showed[35] increased stool fat on a high almond diet over 4 weeks; however, the increment in fecal fat content (4.1%) was less than previously reported[13] using peanuts (17.8%). The greater degree of malabsorption in the latter is most likely due to the fact that fat intake was almost entirely derived from peanuts (76 of 80 g), as supposed to supplying only 50% energy from fat by almonds. Nevertheless, the losses in the almond study of 5.9 g (control *vs* high almond) of fat equaled 222 kJ (50 kcal) which represents a potential weight loss of 0.45 kg over 10 weeks. Over a longer time period, this level may be substantial, but was likely below the level of detectability in the 10 week study.

Despite instructions to maintain body weight a gradual and sustained weight loss was observed when subjects were placed on a low fat diet supplemented with high oleic peanuts compared to a low fat diet without peanuts.[5] The mean weight loss was 3 kg over 6 months; however, there was an increase of approximately 0.6 kg during the first month, followed by a decrease to 0.2 kg below baseline during the second month. Thus, it is possible that the 8 week intervention period in our study may have been to short to capture the effect of nut consumption on weight loss.

Elevation of energy expenditure may aid in the maintenance of energy balance with nut consumption. This may occur if consumption of nuts results in a diet higher in unsaturated fatty acids. In rats, high PUFA diets are associated with greater oxygen consumption compared to high SFA-high MUFA diets.[17] An effect of dietary fatty acid subclasses on energy expenditure has also been reported in humans.[14,36] When a high PUFA/SFA ratio diet was fed for 7 days, an increase in absolute DIT was observed.[36] In another study, significant increases in REE (3.6%) and DIT (22%) were observed after subjects consumed a high PUFA/SFA ratio diet for 14 days.[14] In each case, a meal with similar fatty acid composition to the background diet was consumed.

In this study, energy intake from PUFA and MUFA increased with peanut consumption while intake from SFA remained relatively stable. Thus the background diet, prior to the second DIT measurement, contained a relatively higher PUFA/SFA ratio compared to baseline. However, the meal composition on the day of the DIT measurements was identical. Therefore, compared to previous studies, the lack of effect on DIT with peanut consumption may be due to the fact that changes in the background diet alone, without corresponding changes in the fatty acid composition of the meal used to measure DIT, are insufficient to induce changes in DIT. However, modification of the background diet induced an 11% increase in REE. These findings are consistent with the notion that dietary fatty acid composition can influence indices of energy expenditure and this may play a role in the maintenance of energy balance with nut consumption.

Monotony has been demonstrated with acute[12,37] and long-term[19,21] exposures to a meal or food. This would predict a decline in pleasantness ratings with chronic peanut consumption. In an acute feeding study with peanuts, participants exhibited a reduced desire to consume the nuts.[12] However, repeated exposure to peanuts, in the present study, did not lessen hedonic ratings for peanuts. There is limited data on the development of monotony with long-term exposure. Earlier work has shown a decline in pleasantness ratings for selected meats and vegetables while ratings for cereals increased and sweet foods were highly variable, when subjects consumed a diet of 41 foods for 5 weeks.[19] Generally, foods with high initial baseline ratings remained palatable. Thus, the high baseline hedonic ratings (mean rating 7 out of 9) for peanuts in our study may have ameliorated a decline in pleasantness ratings with long-term exposure. Taste quality and macronutrient content may also influence the development of monotony. A recent repeat exposure study reported a decline in preference ratings with consumption of a sweet high-fat snack food (chocolate) for 15 days but no change with a salty high fat snack food (fries).[20] The peanuts used in this study were salty and high in fat which may have contributed to the observed stability of pleasantness ratings.

Chronic peanut consumption did not lead to hedonic shifts for sour, bitter, sweet, salty or high- or low-fat snack foods. This is in contrast to a study over 15 days, that reported an increase in preference ratings for sweet foods after repeat exposure to a salty food.[20] In our study, participants were provided with recipes and encouraged to incorporate peanuts into their background diet rather than consuming them in the laboratory as an isolated snack. This may have altered the strictly salty sensory experience from the nuts and account for the discrepancy with previously published results.

In summary, despite inclusion of approximately 500 kcal/day (90 g/day) as peanuts for 8 weeks, little change of body weight was observed. A combination of mechanisms may account for this finding. The predominant reason may be a strong compensatory dietary response due, in part, to the satiety-inducing property of peanuts. Findings from this study do not support a lipid malabsorption phenomenon, although this cannot be excluded since direct measures such as stool fat content were not examined. Elevated energy expenditure in the form of REE was observed and may also contribute to maintenance of energy balance. Hedonic ratings of peanuts remained high throughout the course of the study and no hedonic shifts of other snack foods were observed. This suggests increased chronic consumption of peanuts may be well tolerated and not alter the acceptability of other foods in the diet.

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