

Effects of peanut processing on body weight and fasting plasma lipids

Fiona McKiernan¹, Phoebe Lokko², Anna Kuevi², Regiane L. Sales³, Neuza M. B. Costa³, Josefina Bressan³, Rita C. G. Alfnas³ and Richard D. Mattes^{1*}

¹Department of Foods and Nutrition, Purdue University, 212 Stone Hall, 700 West State Street, West Lafayette, IN, USA

²CSIR-Food Research Institute, PO Box M20, Accra, Ghana

³Department of Nutrition and Health, Federal University of Viçosa, Viçosa, MG 36570-000, Brazil

(Received 10 August 2009 – Revised 20 January 2010 – Accepted 3 February 2010 – First published online 11 May 2010)

Peanuts and peanut butter are commonly consumed as a snack, meal component and ingredient in various commercial products. Their consumption is associated with reduced CVD risk and they pose little threat to positive energy balance. However, questions have arisen as to whether product form (e.g. whole nut *v.* butter) and processing properties (e.g. roasting and adding flavours) may compromise their positive health effects. The present study investigated the effects of peanut form and processing on two CVD risk factors: fasting plasma lipids and body weight. One hundred and eighteen adults (forty-seven males and seventy-one females; age 29.2 (SD 8.4) years; BMI 30.0 (SD 4.5) kg/m²) from Brazil, Ghana and the United States were randomised to consume 56 g of raw unsalted (*n* 23), roasted unsalted (*n* 24), roasted salted (*n* 23) or honey roasted (*n* 24) peanuts, or peanut butter (*n* 24) daily for 4 weeks. Peanut form and processing did not differentially affect body weight or fasting plasma lipid responses in the total sample. However, HDL-cholesterol increased significantly at the group level, and total cholesterol, LDL-cholesterol and TAG concentrations decreased significantly in individuals classified as having elevated fasting plasma lipids compared with those with normal fasting plasma lipids. These observations suggest that the processing attributes assessed in this trial do not compromise the lipid-lowering effects of peanuts, and do not negatively impact body weight. Further studies are warranted to determine the effects of form and processing on other health risk factors.

Peanuts: Peanut butter: Processing: Plasma lipids: Body weight

CVD is the leading cause of death in the USA, accounting for one in every 2.8 deaths⁽¹⁾. With an ageing population, the prevalence is predicted to double by 2050⁽²⁾. CVD is also expected to have an increasing detrimental effect in other nations throughout the world⁽³⁾. Rates of CVD and stroke are projected to triple in Latin America and sub-Saharan Africa in the next two decades⁽³⁾.

Peanuts and tree nuts are increasingly recognised for their role in CVD risk reduction, as acknowledged by a Food and Drug Administration qualified health claim in 2003⁽⁴⁾. Epidemiological studies estimate an approximate 35% reduction in the incidence of CVD in the highest nut-consuming groups^(5–8). Multiple components of peanuts including arginine, folate, tocopherols and fatty acids probably mediate their cardioprotective effects.

Clinical studies indicate that tree nuts, with most of the evidence derived from almonds and walnuts, reduce LDL-cholesterol (LDL-C) by 3–19% compared with reference diets, including habitual, lower fat and average American diets⁽⁹⁾. Reductions of up to 11% in total cholesterol and 14% in LDL-C have been reported for peanut interventions compared with similar reference diets^(10–12). Consistent with a more general literature^(13–16), the degree of reduction in plasma cholesterol concentrations in response to peanut consumption is inversely related to baseline concentrations⁽¹¹⁾.

While promoting improved lipid profiles, nut consumption has limited impact on body weight⁽¹⁷⁾. Epidemiological studies reveal either a negative association or a lack of association between nut consumption and BMI^(5–7). Clinical studies support a lack of association under a variety of conditions⁽¹⁷⁾, and may actually aid weight loss through improved dietary compliance⁽¹⁸⁾. Because central obesity is an independent risk factor for CVD, and weight loss leads to a reduction in disease risk⁽¹⁹⁾, moderate consumption of nuts may be a functional component in a cardioprotective diet⁽²⁰⁾.

Clinical intervention studies exploring the effects of nuts on CVD risk and body weight have used natural, unprocessed nuts; lightly salted, roasted nuts; or an unspecified nut variety. Since numerous flavours and forms of nuts are currently available on the market, questions have arisen as to whether processing properties (e.g. grinding to butter, roasting and boiling) and the addition of flavours (e.g. salt, spices and sugar) may alter the health effects⁽²¹⁾. For example, grinding nuts into butter form ruptures the parenchymal cell walls that encapsulate the intracellular components⁽²²⁾. While the complete effects of this alteration in nut form remain unknown, it results in significantly less faecal fat, protein and tocopherol losses compared with the whole nut form^(23,24). Furthermore, concerns have arisen as to the possible adverse effects of the addition of hydrogenated oils to peanuts to prolong shelf life.

Abbreviations: HDL-C, HDL-cholesterol; LDL-C, LDL-cholesterol.

* **Corresponding author:** Richard D. Mattes, fax +1 765 494 0674, email mattes@purdue.edu

Because hydrogenated fats are potential sources of *trans* fats, which have detrimental effects on plasma lipids and CVD risk^(25,26), the addition of hydrogenated fats during processing could compromise the cardiovascular health benefits associated with nut consumption. Analyses of common peanut butter brands reveal non-detectable levels of *trans* fats in the brands analysed⁽²⁷⁾, but concerns persist. Limited evidence also indicates that the modification of rheological properties during processing may alter the satiety properties of nuts⁽²⁸⁾ with possible implications for energy balance.

The primary aim of the present study was to determine whether the form, flavour and processing of peanuts alter the fasting plasma lipid profile and body weight response to their consumption over a 1-month intervention period. Peanuts were used as the test nut as they are the most commonly consumed nut (actually a legume) in the USA, and are available in many flavours and forms⁽²⁹⁾. Based on evidence that greater effects may be observed in individuals with the greatest baseline cholesterol concentrations, differential responses between normolipidaemic and hyperlipidaemic individuals were explored.

Methods

Participants

A total of 120 participants from three countries (Brazil, Ghana and the USA) participated in this multi-centre trial. Forty participants were recruited at each site. Eligibility criteria included stable weight (no deviations >2.5 kg over the prior 3 months); BMI ≥ 25 kg/m²; pre-menopausal; having no known lipid disorders or other acute or chronic diseases; using no prescription medications apart from birth control; and having no nut allergies. The final sample included 118 participants (forty-seven males and seventy-one females; age 29.2 (SD 8.4) years; BMI 30.0 (SD 4.5) kg/m²; Table 1), as the final outcome measures from two participants in Ghana were not available for analyses. The present study was conducted according to the guidelines laid down in the Declaration of Helsinki, and all procedures involving human subjects/patients were approved by human research review boards at each location. Written informed consent was obtained from all the subjects/patients.

Experimental design

The study used a parallel group experimental design. Participants were sequentially assigned to incorporate 56 g (2 oz) of one of five peanut forms into their diet daily for 4 weeks. Each country distributed the participants into five groups with eight people per group. The five peanut forms were whole raw unsalted, whole roasted unsalted, whole roasted salted, whole honey roasted peanuts, and peanut butter. The daily energy and nutrient composition of each treatment are presented in Table 2. Participants were allowed to consume the peanuts/peanut butter at any time of the day and in any manner they chose, but they were requested to restrict consumption of all other nut products during the intervention period. No additional dietary instructions were provided. To ensure consistency across the research sites, all peanuts and peanut butter were provided by a single site (USA). Participants collected their daily peanut rations at the research site, pre-weighed and labelled, on a weekly basis.

Anthropometrics

After a 10 h overnight fast and after voiding, body weight was measured (± 0.1 kg) using calibrated scales (model TBF-305; Tanita, Arlington Heights, IL, USA), with participants wearing no shoes and a light gown, at baseline and post treatment (week 4). Standing height was measured (± 0.1 cm) using a wall-mounted stadiometer (Holtain Limited, Crymych, Dyfed, UK). To allow sub-group analyses, participants were classified, according to BMI, into overweight (BMI 25–29.9 kg/m²; *n* 72) and obese (BMI ≥ 30 kg/m²; *n* 46) categories. Participants were requested to maintain their customary activity levels during the study period, so any changes in body weight were presumed to be due to the dietary intervention.

Fasting plasma lipids

After a 10 h overnight fast, 6 ml of blood were collected at baseline and post-treatment into vacutainers containing EDTA. The samples were immediately placed on ice, and were then centrifuged (3000 rpm \times 15 min at 4 °C), separated and stored at -80 °C until analyses. Samples were analysed

Table 1. Age, weight and BMI, and the distribution of participants based on lipids for the total group and by country at baseline (Mean values and standard deviations)

	Total			Brazil			Ghana			USA		
	<i>n</i>	Mean	SD	<i>n</i>	Mean	SD	<i>n</i>	Mean	SD	<i>n</i>	Mean	SD
Age (years)		29	8	32 ^a	10		29 ^{a,b}	6		27 ^b	8	
Weight (kg)		85	15	82 ^{a,b}	16		82 ^a	13		90 ^b	16	
Weight change (kg)		0.3	0.1	0.6	0.3		0.5	0.0		0	0.1	
BMI (kg/m ²)		30	4	29	4		31	5		30	4	
Total cholesterol < 2000 mg/l	85			25			30			30		
Total cholesterol \geq 2000 mg/l	33			15			8			10		
LDL-C < 1300 mg/l	99			27			36			36		
LDL-C \geq 1300 mg/l	19			13			2			4		
TAG < 1500 mg/l	92			24			36			32		
TAG \geq 2000 mg/l	26			16			2			8		

LDL-C, LDL-cholesterol.

^{a,b} Mean values for age and weight within a row with unlike superscript letters were significantly different from each other ($P < 0.05$).

Table 2. Mean energy and nutrient composition of 56 g of raw unsalted, roasted unsalted and roasted salted, honey roasted peanuts, and peanut butter

	Raw	Roasted unsalted	Roasted salted	Honey roasted	Peanut butter
Energy (kJ)	1329	1403	1403	1308	1378
Energy (kcal)	318	335	335	313	329
Total fat (g)	27.6	29.4	29.4	25.5	28.2
Saturated fat (g)	3.8	4.9	4.9	4.2	5.8
MUFA (g)	13.7	14.5	14.5	12.6	13.3
PUFA (g)	8.7	8.6	8.6	7.4	7.8
Carbohydrate (g)	9.0	8.6	8.6	13.3	11.0
Dietary fibre (g)	4.8	5.3	5.3	4.6	3.4
Protein (g)	14.5	15.7	15.7	13.6	14.1

in duplicate for total cholesterol, LDL-C, HDL-cholesterol (HDL-C) and TAG concentrations using an automated clinical chemistry analyser (COBAS Integra 400, Roche Diagnostic Systems, Branchburg, NJ, USA).

The ratios of total cholesterol to HDL-C, HDL-C to LDL-C and TAG to HDL-C were calculated based on evidence that the ratios of these lipids may be more important and more robust predictors of CVD risk than any lipid fraction alone^(30,31).

Due to evidence indicating that plasma lipid responses to cholesterol-lowering interventions may be greatest in individuals with the highest baseline lipid concentrations⁽¹¹⁾, participants were categorised based on baseline total cholesterol, LDL-C and TAG concentrations to allow sub-group analyses. The total cholesterol groups were classified as normal (total cholesterol concentrations < 2000 mg/l; *n* 85) or high (total cholesterol concentrations ≥ 2000 mg/l; *n* 33); LDL-C groups as normal (LDL-C concentrations < 1300 mg/l; *n* 100) or high (LDL-C concentrations ≥ 1300 mg/l; *n* 18); and TAG groups as normal (TAG concentrations < 1500 mg/l; *n* 92) or high (TAG concentrations ≥ 1500 mg/l; *n* 26)⁽³²⁾.

Dietary intake

Three-day food records (two non-consecutive weekdays and one weekend day) were recorded at baseline and during weeks 2 and 4 of the intervention. Training was provided on the method for estimating food portion sizes using food models. Food records were reviewed with participants once they were completed to clarify details and obtain any additional information deemed necessary. Each study site analysed the food records using country-specific nutrient databases, focusing specifically on the daily intake of energy, total fat, carbohydrate and protein. In addition, US food records were analysed for SFA, MUFA, PUFA, cholesterol, total dietary fibre, soluble fibre, insoluble fibre, total α -tocopherol, folate, Mg and arginine.

Peanut form palatability

At baseline and post treatment, each of the five peanut forms, weighing between 1.38 and 1.42 g, was sampled in a randomised order and rated for palatability on a hedonic scale end anchored with 'not at all palatable' and 'extremely palatable'. Participants rinsed thoroughly between samples. The mean palatability of the peanut form consumed daily during the intervention period was compared with the mean palatability

of the other four non-intervention peanut forms to assess the impact of frequency of consumption on palatability ratings.

Appetite ratings

An additional component of the US study required participants to record the subjective sensations of hunger, fullness, desire to eat, desire to eat something sweet, desire to eat something salty, prospective consumption and thirst on visual analogue scales (developed by W. Horn) on personal digital assistants. The scales were end anchored with 'not at all' and 'extremely'. Each scale was completed every waking hour for 24 h during one weekday of baseline and weeks 2 and 4 of the intervention. The day of the week on which the recordings were made was held constant for each participant.

Statistical analyses

Statistical analyses were performed using SPSS (version 16.0, SPSS Inc., Chicago, IL, USA). Treatment effects were tested by repeated-measures ANOVA, with time as the within-subject factor and peanut form as the between-subject factor. Country was not entered as a between-subject factor in the final analyses because there were no significant differences between them with respect to the main outcome variables. Sub-group analyses were conducted for sex, BMI and lipid categories using the grouping variable as the between-subject factor in a repeated-measures ANOVA and time as the within-subject factor. Paired *t* tests were performed for *post hoc* analyses with the Bonferroni adjustment when the main effects were significant. A significance level of $P < 0.05$, two-tailed, was set as the criterion for significance. All data are expressed as mean values and standard deviations.

Results

Body weight/BMI and sex

Independent sample *t* tests revealed no statistically significant BMI/body weight or sex effects within any of the groups for the main outcome measures. There were no significant differences between peanut form groups at baseline with respect to body weight (84.6 (SD 15.2) kg). There was also no significant time or time \times peanut form interaction with respect to change in body weight following the intervention. Mean body weight at the end of week 4 was 84.9 (SD 15.1) kg.

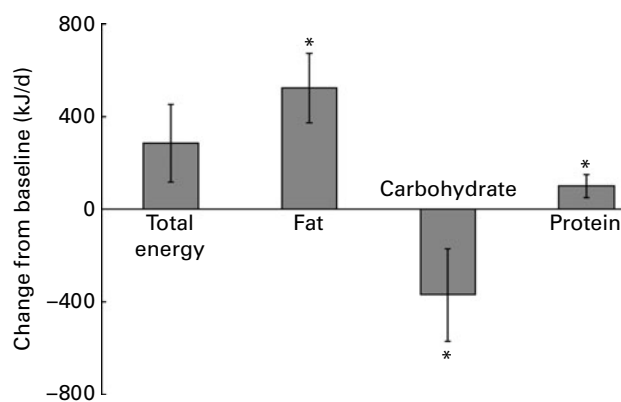


Fig. 1. Changes in daily total energy and macronutrient intakes (as kJ/d) from baseline for the total group (n 118). Values were represented as means and standard deviations. *Mean values were significantly different from baseline ($P < 0.05$).

Dietary intake

Total daily energy intake did not change significantly between baseline and post treatment (Fig. 1). Total fat intake increased significantly ($F(1,112) = 41.6$, $P < 0.01$), carbohydrate intake decreased significantly ($F(1,112) = 12.7$, $P < 0.01$) and protein intake increased significantly ($F(1,112) = 5.9$, $P = 0.02$) from baseline (Fig. 1). There were no significant differences between the peanut form groups with respect to changes in total daily energy intake or intake of the macronutrients.

In the US sample, there were significant increases in MUFA ($F(1,35) = 26.1$, $P < 0.01$), PUFA ($F(1,35) = 6.5$, $P = 0.02$), fibre ($F(1,35) = 13.4$, $P < 0.01$), folate ($F(1,35) = 13.7$, $P < 0.01$) and arginine ($F(1,35) = 22.8$, $P < 0.01$) intakes relative to baseline (Table 3). There was a trend towards a significant increase in total α -tocopherol, but this just failed to reach statistical significance ($F(1,35) = 3.9$, $P = 0.06$). There was no significant change in SFA intake.

Plasma lipids

Baseline measurements of total cholesterol were significantly higher in the roasted unsalted group than in the peanut butter group ($P = 0.01$; Table 4). There were no significant differences in LDL-C, HDL-C or TAG concentrations at baseline between treatment groups. Furthermore, baseline measurements of total cholesterol:HDL-C, HDL-C:LDL-C and TAG:HDL-C ratios did not differ significantly between peanut form treatment groups.

In the full sample, total cholesterol and LDL-C concentrations did not change significantly from baseline to post treatment (Table 4). HDL-C concentrations increased significantly from baseline ($F(1,113) = 6.9$, $P = 0.01$). Mean serum TAG concentrations decreased by 5% from baseline to post treatment, but these failed to reach statistical significance ($F(1,113) = 1.6$, $P = 0.21$). The total cholesterol:HDL-C ratio did not change significantly from baseline to post treatment. There was a trend towards an increase in the HDL-C:LDL-C ratio ($F(1,113) = 2.8$, $P = 0.097$). The TAG:HDL-C ratio decreased significantly from baseline to post treatment ($F(1,113) = 4.1$, $P = 0.04$).

There were no significant differences between peanut form treatment groups with respect to changes in total cholesterol, LDL-C, HDL-C or TAG concentrations. No significant treatment group differences were noted in the change in total cholesterol:HDL-C, HDL-C:LDL-C or TAG:HDL-C ratios.

Sub-group analyses revealed a significant time \times lipid category interaction for total cholesterol and LDL-C concentrations (Fig. 2). Individuals in the high total cholesterol group (≥ 2000 mg/l) had significantly greater decreases of total cholesterol and LDL-C concentrations than individuals in the normal total cholesterol group ($F(1,116) = 6.6$, $P = 0.01$ and $F(1,116) = 6.2$, $P = 0.02$, respectively). Individuals with high LDL-C concentrations had significantly greater decreases of total cholesterol and LDL-C concentrations than individuals with normal LDL-C concentrations ($F(1,116) = 13.9$, $P < 0.001$ and $F(1,116) = 14.1$, $P < 0.001$,

Table 3. Total daily energy and nutrient intakes at baseline and post treatment for the US sample only (n 40) (Mean values and standard deviations)

	Baseline		Post treatment		<i>P</i>
	Mean	SD	Mean	SD	
Total daily energy (kJ/d)	8426	2478	8468	1901	0.681
Total daily energy (kcal/d)	2014	592	2024	454	0.681
Total fat (g/d)	78	32	88*	25	0.005
Saturated fat (g/d)	27	12	27	9	0.287
MUFA (g/d)	29	12	36*	10	< 0.01
PUFA (g/d)	16	8	19*	7	0.015
Cholesterol (mg/d)	230	153	217	135	0.496
Carbohydrate (g/d)	250	75	229*	63	0.029
Total dietary fibre (g/d)	17	5	18	5	0.059
Soluble dietary fibre (g/d)	4.7	1.7	4.3	1.3	0.253
Insoluble dietary fibre (g/d)	11.7	4.1	13.5*	4.1	< 0.001
Total protein (g/d)	82	27	86	26	0.186
Total α -tocopherol (mg)	12.1	11.7	14.3	13.9	0.057
Natural folate (μ g/d)	199	84	242*	85	0.001
Mg (mg/d)	285	104	319*	82	0.022
Arginine (g/d)	4.3	1.7	5.3*	1.7	< 0.001

* Mean values were significantly different from baseline ($P < 0.05$).

Table 4. Serum lipid concentrations at baseline and post treatment by nut treatment group (Mean values and standard deviations)

Serum lipid	Nut treatment	n	Baseline		Post treatment		P
			Mean	SD	Mean	SD	
Total cholesterol (mg/l)	Honey roasted	24	1810 ^{a,b}	360	1780 ^{a,b}	300	0.573
	Roasted salted	23	1820 ^{a,b}	250	1840 ^{a,b}	240	
	Raw	23	1770 ^{a,b}	300	1840 ^{a,b}	360	
	Roasted unsalted	24	2030 ^a	460	2010 ^a	380	
	Peanut butter	24	1720 ^b	230	1750 ^b	250	
	Total	118	1830	340	1840	320	
LDL-cholesterol (mg/l)	Honey roasted	24	1040	280	1010	240	0.887
	Roasted salted	23	1050	240	1060	250	
	Raw	23	1040	260	1050	260	
	Roasted unsalted	24	1190	400	1170	360	
	Peanut butter	24	990	200	1030	230	
	Total	118	1060	290	1060	280	
HDL-cholesterol (mg/l)	Honey roasted	24	510	160	530	180	0.009
	Roasted salted	23	530	160	560	180	
	Raw	23	520	160	540	200	
	Roasted unsalted	24	610	220	630	240	
	Peanut butter	24	490	120	510	120	
	Total	118	530	170	560*	190	
TAG (mg/l)	Honey roasted	24	1330	730	1220	680	0.197
	Roasted salted	23	1120	690	1100	730	
	Raw	23	1080	420	1190	530	
	Roasted unsalted	24	1170	620	1060	480	
	Peanut butter	24	1120	540	980	460	
	Total	118	1160	610	1110	580	

^{a,b} Mean values within a column with unlike superscript letters were significantly different from each other and lipid category ($P < 0.05$).

* Mean values were significantly different from baseline ($P < 0.05$).

respectively). Individuals with elevated baseline TAG concentrations had significantly greater decreases of TAG concentrations relative to individuals with normal TAG concentrations ($F(1,116) = 9.6$, $P < 0.01$).

The lipid subgroups did not differ significantly with respect to baseline body weight, change in body weight, or change in total daily energy, protein, fat or carbohydrate intake. However, the individuals classified as having high total cholesterol were significantly older than individuals with normal total cholesterol (28 (SD 7) and 33 (SD 10) years, respectively, $P < 0.05$). Similarly, individuals classified as having high LDL-C concentrations were significantly older than individuals with normal LDL-C concentrations (28 (SD 7) and 34 (SD 11) years, respectively, $P < 0.05$).

Appetite ratings

Self-reported hunger, fullness, desire to eat, desire to eat something sweet or salty, prospective consumption and thirst ratings did not change significantly with time. Furthermore, there was no time \times peanut form interaction for any of these variables.

Peanut form palatability

While peanut butter was rated as the most palatable (78 (SD 20)) form and raw peanuts were rated as the least palatable (34 (SD 24)) form, there were no significant differences in palatability between the nut treatments at baseline. Honey roasted, roasted salted and roasted unsalted peanuts were rated at 75 (SD 22), 73 (SD 15) and 60 (SD 18), respectively.

The palatability ratings of the nuts consumed daily during the intervention decreased with time, but not significantly (mean rating at baseline: 70 (SD 23); mean rating post treatment: 65 (SD 25)). Furthermore, there was no significant time \times peanut form interaction for this variable. The rate of change in palatability ratings was not significantly different between the nuts consumed daily during the intervention and the other four nuts that were not consumed daily.

Discussion

Epidemiological and clinical evidence support a beneficial effect of nut consumption on CVD risk factors, in particular plasma lipid concentrations^(8,9,33). Benefits are achieved while having a limited impact on body weight⁽¹⁷⁾. However, the evidence supporting this association is mainly derived from unprocessed nuts, and changes introduced during processing have been hypothesised to alter these findings⁽²¹⁾. The present study suggests that processing, specifically the addition of flavours, grinding to butter and roasting, does not alter the lipid-lowering effects of peanuts. Significant lipid-lowering effects were observed in hyperlipidaemic individuals with all peanut varieties. Furthermore, these benefits were achieved without altering body weight status.

In the present study, significant reductions in total cholesterol, LDL-C and TAG concentrations were observed when hyperlipidaemic individuals consumed 56 g of whole raw, roasted unsalted, roasted salted or honey roasted peanuts, or ground peanut butter daily for 4 weeks. There were no significant differences between the peanut treatments with respect to these lipid-lowering responses despite differences in

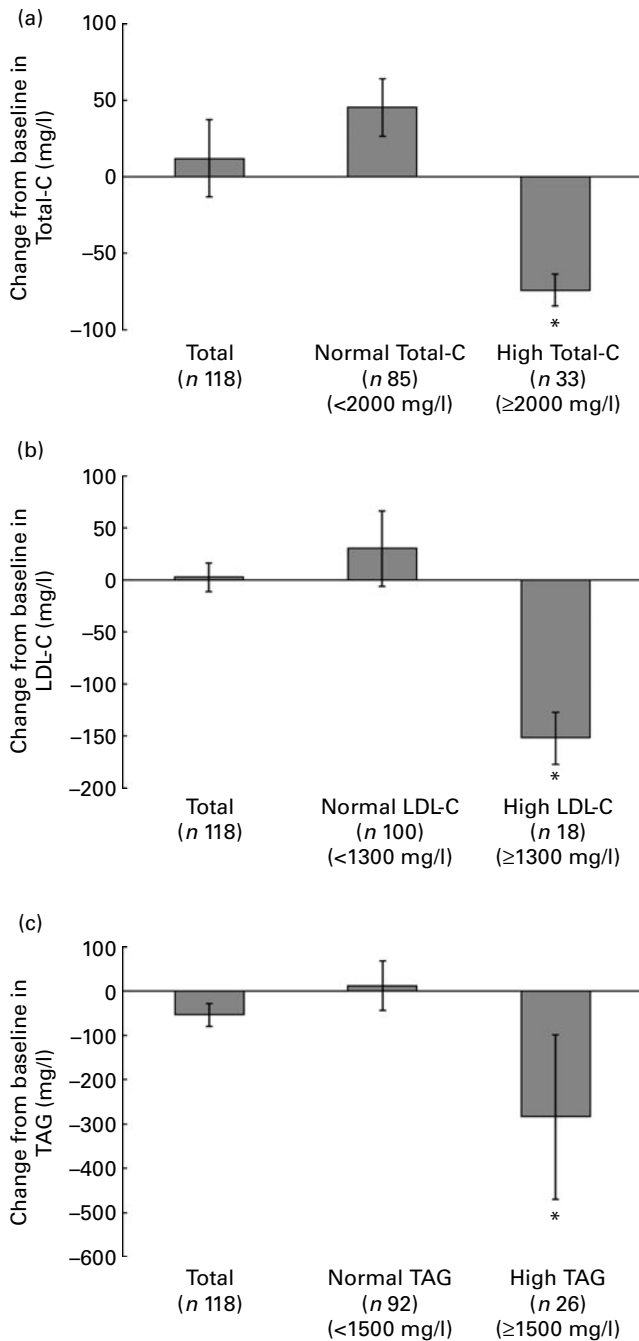


Fig. 2. Changes in (a) total cholesterol (Total-C), (b) LDL-cholesterol (LDL-C) and (c) TAG from baseline for the total group and according to lipid subgroups. Values were represented as means and standard deviations. (a) * Mean values were significantly different from normal Total-C group ($P < 0.05$). (b) * Mean values were significantly different from normal LDL-C group ($P < 0.05$). (c) * Mean values were significantly different from normal TAG group ($P < 0.05$).

processing such as grinding and roasting before consumption. Overall, total cholesterol decreased by 3 % (74 mg/l), LDL-C decreased by 10 % (150 mg/l) and TAG concentrations decreased by 13 % (290 mg/l) from baseline. O'Byrne *et al.*⁽¹⁰⁾ reported greater reductions in total cholesterol and LDL-C in hyperlipidaemic women following peanut consumption, with concentrations decreasing by 10 and 12 %, respectively. However, the peanuts used in that study were enriched

with oleic acid, containing 60–70 % more than other commercially available varieties of peanuts. Also differences in the contribution of dietary SFA to total daily energy intake may account for the disparities in the reported cholesterol-lowering effects. In the study by O'Byrne *et al.*⁽¹⁰⁾, SFA contributed 5 % to total energy intake in contrast to 12 % in the present study. Since a direct, positive, dose-dependent relationship exists between SFA and plasma total cholesterol and LDL-C⁽²⁰⁾, the differences in dietary SFA likely account, at least partially, for the differences observed. The cholesterol-lowering effects in the present study are, thus, predicted to be even greater if accompanied by a lower SFA diet.

While the unsaturated fatty acid profile of nuts (high MUFA and PUFA) is thought to mediate the majority of the favourable effects on plasma lipids, other components such as fibre and phytosterols may also contribute^(8,9). Furthermore, in the present study, the lower TAG concentration may stem from the spontaneous reduction in carbohydrate intake when the peanuts were added to the diet. Reductions in carbohydrate intake are associated with decreases in TAG concentrations⁽³⁴⁾, and thus, the decreases in carbohydrate intake reported may have had an independent effect on lipid concentrations. It is estimated that a 370 mg/l (1 mmol/l) reduction in total cholesterol and LDL-C results in 24–28 % decreases in the relative risk of CHD mortality⁽³⁵⁾, and that an 880 mg/l (1.0 mmol/l) decrease in TAG is associated with a 14–37 % reduction in overall CVD risk⁽³⁶⁾.

In contrast to the hyperlipidaemic individuals, no significant changes in plasma lipids were observed in the individuals with normal lipid concentrations. This is in contrast to a peanut intervention study that reported a 12 % reduction in total cholesterol and a 10 % reduction in LDL-C in normocholesterolaemic individuals consuming whole peanuts and peanut butter for 24 d⁽¹¹⁾. In that study, MUFA from peanuts were substituted for SFA, resulting in a decrease in the contribution of SFA to the total daily energy intake from 16 to 7 % during the peanut intervention⁽¹¹⁾. However, in the present study, no substitutions were made, and SFA intake levels did not change from baseline and were maintained at 12 % of total daily energy intake during the intervention. A similar study also failed to report significant changes in total cholesterol or LDL-C following a peanut intervention trial in normocholesterolaemic individuals when the contribution of SFA to the diet remained relatively stable at 10 % of total daily energy intake⁽³⁷⁾. These findings indicate that a simultaneous reduction in SFA and an increase in MUFA may be necessary to elicit changes in plasma lipids in normolipidaemic individuals with peanut consumption. However, of note, the reductions observed by Kris-Etherton *et al.*⁽¹¹⁾ among normocholesterolaemic individuals were greater in those with the highest concentrations at baseline. This is consistent with the present findings and with several reports from other lipid-lowering dietary interventions with foods such as oats^(13–16).

The present study supports the findings from epidemiological and clinical studies reporting that peanut consumption has limited effects on body weight^(5,6,10,12,38,39). However, the results extend beyond these findings to indicate that neither peanut form nor flavour affects this outcome measure. Consumption of four different flavours of whole peanuts or

peanut butter at a level of approximately 1363.9 kJ/d (mean daily energy contributed from the five peanut treatments) for 4 weeks did not cause any significant changes in body weight. The mean theoretical weight gain due to the peanut intervention was calculated to be 1.2 kg over the 4-week period assuming no compensation. As the mean change in body weight was 0.3 (SD 0.1) kg, a strong compensation for the peanut energy load is indicated. The lack of effect on body weight could be due to dietary compensation and increased satiety, limited efficiency of absorption of energy from the peanuts or increased energy expenditure⁽¹⁷⁾.

Beneficial changes in dietary intake beyond MUFA and PUFA intakes were observed in the present study, as reflected by the US food intake records. Arginine, folate and Mg intakes increased significantly. Since arginine is the precursor of NO, which has many bioactive properties, including vasodilation and reduced platelet aggregation⁽⁴⁰⁾, increases in dietary intake may contribute to cardioprotective effects beyond those associated with lipid-lowering. Increasing folate intake may also improve plasma homocysteine status⁽⁴¹⁾. Elevated homocysteine concentrations are an independent risk factor for the development of atherosclerosis⁽⁴²⁾, and thus, by functioning as a methyl donor in the conversion of homocysteine to methionine, dietary folate can lower plasma homocysteine concentrations and lower CVD risk⁽⁴¹⁾. Increases in Mg intake may afford additional benefits if they translate into increases in plasma Mg concentrations, as the risk of CVD is inversely related to the concentration of plasma Mg^(43,44). The potential mechanisms mediating this beneficial outcome include a reduction in the formation of free oxygen radicals and pro-inflammatory molecules⁽⁴⁴⁾. Furthermore, while α -tocopherol intakes only tended to rise, its antioxidant properties, along with those of other antioxidants in nuts, are hypothesised to reduce atherogenic oxidative processes^(45,46). A reduction in lipid peroxidation has been noted with peanut consumption⁽⁴⁷⁾, and improvements in oxidative markers have also been documented for other nuts^(48,49). The increases in intake of cardioprotective nutrients other than MUFA and PUFA noted in the present study are similar to those reported previously⁽³⁷⁾.

While the present study failed to observe an effect of processing on the lipid-lowering effects of peanuts, future studies are warranted to determine the impact of processing on other CVD risk parameters such as blood pressure, oxidative stress, inflammation, insulin sensitivity and endothelial function. It is plausible that the lipid-lowering effects are maintained, but the overall health effects are altered. Furthermore, while the present research suggests that the addition of salt and sugar, grinding to butter and roasting do not have implications for the short-term lipid-lowering effects of peanuts, the effects of other processing procedures on health outcomes merit investigation, e.g. boiling of peanuts and removal of skin. Boiling of peanuts in water could lead to the leaching out of cardioprotective, water-soluble nutrients⁽⁵⁰⁾, while removal of the skins of peanuts during processing significantly alters the antioxidant capacity⁽⁵¹⁾. The effects of processing on other nut varieties also warrant exploration.

Similar to previous reports^(52,53), the present study noted a decrease in palatability ratings over time. However, the hedonic ratings were not significantly different at the end of the intervention compared with baseline. Thus, while a degree

of monotony occurred, it was not significant. Alper & Mattes⁽³⁹⁾ reported a similar stability of palatability ratings with daily consumption of peanuts for 8 weeks. This indicates a tolerance of daily nut consumption. Furthermore, given the comparable health effects noted with the different peanut forms, varying the sensory properties may aid regular use without compromising the benefits.

Failure to measure compliance by an objective measure such as changes in erythrocyte membrane fatty acid composition is a limitation of the present research. Furthermore, while food intake diaries are frequently used to estimate food intake in free-living individuals, they are not without error. This technique has been shown to underestimate intakes, especially in obese individuals⁽⁵⁴⁾. And while measures were taken to improve accuracy (e.g. participants received education on how to record food intake accurately, and records were reviewed for accuracy with participants), the mean daily energy intakes were more reflective of the energy needs of a normal weight population (approximately 8368 kJ/d) than of those of the present overweight population.

Conclusions

Different forms and flavours of peanuts, when consumed in moderate quantities, lead to a less atherogenic lipid profile in hyperlipidaemic individuals. Such changes may be achieved without significant impact on body weight. The continued high palatability of the peanuts over the trial period suggests that monotony will not be a barrier to regular consumption. The lack of significant country effects also indicates that nut consumption may be a feasible intervention to reduce CVD risk globally.

Acknowledgements

We thank the assistance of William Horn for the development and adaptation of the Appetite Log VAS software (US Department of Agriculture, Agricultural Research Service, Western Human Nutrition Research Center, Davis, CA 95 616, USA). None of the authors have a conflict of interest related to this work of manuscript. R. D. M. contributed to the design and analysis of the study and manuscript preparation, and had primary responsibility for its final content. F. M. contributed to the design, conduct and analysis of the study and manuscript preparation. P. L., A. K., R. L. S., N. M. B. C., J. B. and R. C. G. A. contributed to the conduct and analysis of the study as well as to the manuscript preparation. This research was supported by a grant from the US Agency for International Development Peanut Collaborative Research Support Program #RD309-022/4092094 and FAPEMIG APQ-0957-4.08/07.

References

1. Rosamond W, Flegal K, Furie K, *et al.* (2008) Heart disease and stroke statistics – 2008 update: a report from the American Heart Association Statistics Committee and Stroke Statistics Subcommittee. *Circulation* **117**, 25–146.
2. Foot DK, Lewis RP, Pearson TA, *et al.* (2000) Demographics and cardiology, 1950–2050. *J Am Coll Cardiol* **35**, B66–B80.
3. Callow AD (2006) Cardiovascular disease 2005 – the global picture. *Vascul Pharmacol* **45**, 302–307.

4. Food and Drug Administration (FDA) (2008) Qualifier health claims: letter of enforcement discretion – nuts and coronary heart disease. <http://www.cfsan.fda.gov/~dms/qhcnuts2.html> (accessed 28 September 2008).
5. Fraser GE, Sabaté J, Beeson WL, *et al.* (1992) A possible protective effect of nut consumption on risk of coronary heart disease. The Adventist Health Study. *Arch Intern Med* **152**, 1416–1424.
6. Ellsworth JL, Kushi LH & Folsom AR (2001) Frequent nut intake and risk of death from coronary heart disease and all causes in postmenopausal women: the Iowa Women's Health Study. *Nutr Metab Cardiovasc Dis* **11**, 372–377.
7. Albert CM, Gaziano JM, Willett WC, *et al.* (2002) Nut consumption and decreased risk of sudden cardiac death in the Physicians' Health Study. *Arch Intern Med* **162**, 1382–1387.
8. Kris-Etherton PM, Hu FB, Ros E, *et al.* (2008) The role of tree nuts and peanuts in the prevention of coronary heart disease: multiple potential mechanisms. *J Nutr* **138**, S1746–S1751.
9. Griel AE & Kris-Etherton PM (2006) Tree nuts and the lipid profile: a review of clinical studies. *Br J Nutr* **96**, S68–S78.
10. O'Byrne DJ, Knauft DA & Shireman RB (1997) Low fat-monounsaturated rich diets containing high-oleic peanuts improve serum lipoprotein profiles. *Lipids* **32**, 687–695.
11. Kris-Etherton PM, Pearson TA, Wan Y, *et al.* (1999) High-monounsaturated fatty acid diets lower both plasma cholesterol and triacylglycerol concentrations. *Am J Clin Nutr* **70**, 1009–1015.
12. Lokko P, Lartey A, Armar-Klemesu M, *et al.* (2007) Regular peanut consumption improves plasma lipid levels in healthy Ghanaians. *Int J Food Sci Nutr* **58**, 190–200.
13. Anderson JW, Spencer DB, Hamilton CC, *et al.* (1990) Oat-bran cereal lowers serum total and LDL cholesterol in hypercholesterolemic men. *Am J Clin Nutr* **52**, 495–499.
14. Ripsin CM, Keenan JM, Jacobs DR Jr, *et al.* (1992) Oat products and lipid lowering. A meta-analysis. *JAMA* **267**, 3317–3325.
15. Anderson JW, Johnstone BM & Cook-Newell ME (1995) Meta-analysis of the effects of soy protein intake on serum lipids. *N Engl J Med* **333**, 276–282.
16. Romero AL, Romero JE, Galaviz S, *et al.* (1998) Cookies enriched with psyllium or oat bran lower plasma LDL cholesterol in normal and hypercholesterolemic men from Northern Mexico. *J Am Coll Nutr* **17**, 601–608.
17. Mattes RD, Kris-Etherton PM & Foster GD (2008) Impact of peanuts and tree nuts on body weight and healthy weight loss in adults. *J Nutr* **138**, S1741–S1745.
18. McManus K, Antinoro L & Sacks F (2001) A randomized controlled trial of a moderate-fat, low-energy diet compared with a low fat, low-energy diet for weight loss in overweight adults. *Int J Obes Relat Metab Disord* **25**, 1503–1511.
19. Sowers JR (2003) Obesity as a cardiovascular risk factor. *Am J Med* **115**, S37–S41.
20. Van Horn L, McCain M, Kris-Etherton PM, *et al.* (2008) The evidence for dietary prevention and treatment of cardiovascular disease. *J Am Diet Assoc* **108**, 287–331.
21. King JC, Blumberg J, Ingwersen L, *et al.* (2008) Tree nuts and peanuts as components of a healthy diet. *J Nutr* **138**, S1736–S1740.
22. Ellis PR, Kendall CW, Ren Y, *et al.* (2004) Role of cell walls in the bioaccessibility of lipids in almond seeds. *Am J Clin Nutr* **80**, 604–613.
23. Levine AS & Silvis SE (1980) Absorption of whole peanuts, peanut oil, and peanut butter. *N Engl J Med* **303**, 917–918.
24. Traoret CJ, Lokko P, Cruz AC, *et al.* (2008) Peanut digestion and energy balance. *Int J Obes (Lond)* **32**, 322–328.
25. Mensink RP, Zock PL, Kester AD, *et al.* (2003) Effects of dietary fatty acids and carbohydrates on the ratio of serum total to HDL cholesterol and on serum lipids and apolipoproteins: a meta-analysis of 60 controlled trials. *Am J Clin Nutr* **77**, 1146–1155.
26. Mozaffarian D, Katan MB, Ascherio A, *et al.* (2006) *Trans* fatty acids and cardiovascular disease. *N Engl J Med* **354**, 1601–1613.
27. Sanders TH (2001) Non-detectable levels of *trans*-fatty acids in peanut butter. *J Agric Food Chem* **49**, 2349–2351.
28. Kirkmeyer SV & Mattes RD (2000) Effects of food attributes on hunger and food intake. *Int J Obes Relat Metab Disord* **24**, 1167–1175.
29. He S, Fletcher S & Rimal A (2005) Snack peanut consumption: type preference and consumption manners. *J Food Distrib Res* **36**, 79–85.
30. Fernandez ML & Webb D (2008) The LDL to HDL cholesterol ratio as a valuable tool to evaluate coronary heart disease risk. *J Am Coll Nutr* **27**, 1–5.
31. Da Luz PL, Favarato D, Faria-Neto JR Jr, *et al.* (2008) High ratio of triglycerides to HDL-cholesterol predicts extensive coronary disease. *Clinics* **63**, 427–432.
32. American Heart Association (2003) *Heart Disease and Stroke*. Dallas, TX: American Heart Association.
33. Mukuddem-Petersen J, Oosthuizen W & Jerling JC (2005) A systematic review of the effects of nuts on blood lipid profiles in humans. *J Nutr* **135**, 2082–2089.
34. Appel LJ, Sacks FM, Carey VJ, *et al.* (2005) Effects of protein, monounsaturated fat, and carbohydrate intake on blood pressure and serum lipids: results of the OmniHeart randomized trial. *JAMA* **294**, 2455–2464.
35. Gould AL, Davies GM, Alemao E, *et al.* (2007) Cholesterol reduction yields clinical benefits: meta-analysis including recent trials. *Clin Ther* **29**, 778–794.
36. Cullen P (2000) Evidence that triglycerides are an independent coronary heart disease risk factor. *Am J Cardiol* **86**, 943–949.
37. Alper CM & Mattes RD (2003) Peanut consumption improves indices of cardiovascular disease risk in healthy adults. *J Am Coll Nutr* **22**, 133–141.
38. Hu FB, Stampfer MJ, Manson JE, *et al.* (1998) Frequent nut consumption and risk of coronary heart disease in women: prospective cohort study. *BMJ* **317**, 1341–1345.
39. Alper CM & Mattes RD (2002) Effects of chronic peanut consumption on energy balance and hedonics. *Int J Obes Relat Metab Disord* **26**, 1129–1137.
40. Wells BJ, Mainous AG & Everett CJ (2005) Association between dietary arginine and C-reactive protein. *Nutrition* **21**, 125–130.
41. Weikert C, Hoffmann K, Dierkes J, *et al.* (2005) A homocysteine metabolism-related dietary pattern and the risk of coronary heart disease in two independent German study populations. *J Nutr* **135**, 1981–1988.
42. Boushey CJ, Beresford SA, Omenn GS, *et al.* (1995) A quantitative assessment of plasma homocysteine as a risk factor for vascular disease. Probable benefits of increasing folic acid intakes. *JAMA* **274**, 1049–1057.
43. Singh RB, Gupta UC, Mittal N, *et al.* (1997) Epidemiologic study of trace elements and magnesium on risk of coronary artery disease in rural and urban Indian populations. *J Am Coll Nutr* **16**, 62–67.
44. Chakraborti S, Chakraborti T, Mandal M, *et al.* (2002) Protective role of magnesium in cardiovascular diseases: a review. *Mol Cell Biochem* **238**, 163–179.
45. Blomhoff R (2005) Dietary antioxidants and cardiovascular disease. *Curr Opin Lipidol* **16**, 47–54.
46. Moskaug JØ, Carlsen H, Myhrstad MC, *et al.* (2005) Polyphenols and glutathione synthesis regulation. *Am J Clin Nutr* **81**, S277–S283.
47. O'Byrne DJ, O'Keefe SF & Shireman RB (1998) Low-fat, monounsaturated-rich diets reduce susceptibility of low density lipoproteins to peroxidation *ex vivo*. *Lipids* **33**, 149–157.

48. Fitó M, Guxens M, Corella D, *et al.* (2007) Effect of a traditional Mediterranean diet on lipoprotein oxidation: a randomized controlled trial. *Arch Intern Med* **167**, 1195–1203.
49. Jenkins DJ, Kendall CW, Marchie A, *et al.* (2008) Effect of almonds on insulin secretion and insulin resistance in nondiabetic hyperlipidemic subjects: a randomized controlled cross-over trial. *Metabolism* **57**, 882–887.
50. O'Connor L (2008) Peanut processing: peanut butter and boiled peanuts <http://www.calstatela.edu/faculty/hsingh2/NTRS%20519%20topics/Peanuts%20by%20lauren.pdf> (accessed 10 October 2008).
51. Blomhoff R, Carlsen MH, Andersen LF, *et al.* (2006) Health benefits of nuts: potential role of antioxidants. *Br J Nutr* **96**, S52–S60.
52. Zandstra EH, De Graaf C, Mela DJ, *et al.* (2000) Short- and long-term effects of changes in pleasantness on food intake. *Appetite* **34**, 253–260.
53. Hetherington MM, Pirie LM & Nabb S (2002) Stimulus satiation: effects of repeated exposure to foods on pleasantness and intake. *Appetite* **38**, 19–28.
54. Braam L, Ocke M, Bueno-de-Mesquita HB, *et al.* (1998) Determinants of obesity-related underreporting of energy intake. *Am J Epidemiol* **147**, 1081–1086.