

# Phytosterols in low- and nonfat beverages as part of a controlled diet fail to lower plasma lipid levels

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**Abstract** Dietary phytosterols have been shown to reduce plasma cholesterol concentrations when consumed in different food matrices, but their effectiveness in nonfat or low-fat beverages has not been established. The objective of this study was to examine whether phytosterols alter plasma lipid levels when incorporated into nonfat or low-fat beverages. Fifteen moderately hypercholesterolemic men and women consumed three precisely controlled diets for periods of 21 days each in random order. Diets contained either a nonfat placebo beverage (NF), a beverage that is nonfat with added phytosterols (NFPS), or a beverage that is low in fat with added phytosterols (LFPS). Total cholesterol concentrations were not different between groups at endpoint, decreasing ( $P < 0.05$ ) equally by 8.5%, 11.6%, and 10.1% with NF, NFPS, and LFPS consumption, respectively. There was no effect of dietary treatment on LDL cholesterol concentrations, which decreased over time ( $P < 0.05$ ) by 5%, 10.4%, and 8.5% with NF, NFPS, and LFPS, respectively. HDL cholesterol and triacylglycerol concentrations were unaffected by the diets. Provision of phytosterols as part of nonfat and low-fat beverages did not exert any greater hypocholesterolemic effect than a nonfat placebo beverage. **These results show that intake of phytosterols in a low-fat beverage format is not efficacious for lipid level modification.**—Jones, P. J. H., C. A. Vanstone, M. Raeini-Sarjaz, and M-P. St-Onge. **Phytosterols in low- and nonfat beverages as part of a controlled diet fail to lower plasma lipid levels.** *J. Lipid Res.* 2003. 44: 1713–1719.

**Supplementary key words** cholesterol • hypercholesterolemia • placebo • cardiovascular disease

Low-fat diets containing less than 30% of energy as fat and less than 10% as saturated fat have long been promoted for lowering plasma cholesterol concentrations in hypercholesterolemic patients. Recently, the National Cholesterol Education Program Adult Treatment Panel III guidelines have added the inclusion of phytosterols as part of their therapeutic lifestyle changes dietary guidelines (1) in addition to the traditional recommendations to lower total fat, saturated fat, and cholesterol intakes.

Furthermore, in 2000, the US Food and Drug Administration issued a health claim stating that foods containing plant sterol and stanol esters, when consumed in foods low in saturated fat and cholesterol, reduced the risk of coronary heart disease (2). Phytosterols have traditionally been incorporated into margarines, spreads, and other high-fat foods and have been shown to be efficacious in lowering total cholesterol (TC) and LDL cholesterol (3–11). In a recent meta-analysis, it was reported that consumption of more than 2 g of phytosterols/day reduced LDL cholesterol concentrations by 0.33 to 0.54 mmol/l, depending on the age of the subjects studied, and that intakes of less than 2 g/day led to reductions in LDL cholesterol of at least 0.4 to 0.5 mmol/l (3). It is thus generally agreed that phytosterol consumption leads to, on average, a 10% reduction in TC and a 13% reduction in LDL cholesterol concentrations.

Phytosterols exert their cholesterol-lowering action by suppressing intestinal cholesterol absorption while partially suppressing cholesterol biosynthesis (9, 12, 13). While they are only partially absorbed into the circulation, phytosterol solubility and incorporation into intestinal micelles is an important aspect of phytosterol cholesterol-lowering efficacy. A previous study using phytosterols-safflower oil (4:1, v/v) in capsules did not show any reduction in cholesterol concentrations, probably due to matrix interference (4). Phytosterols thus require some fat for solubilization and cholesterol-lowering efficacy. More recently, however, other factors have been found that improve phytosterol solubility. One such factor, lecithin, results in reduced cholesterol absorption at lower phytosterol dosages (14).

In the majority of the previous trials, phytosterols have been provided in a fat matrix and fed as part of a regular or low-fat diet containing between 30% and 35% of energy as fat (15, 16). However, incorporating phytosterols into low-fat foods would be more recommendable in

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Abbreviations: CNRU, Clinical Nutrition Research Unit; LFPS, low-fat with phytosterols; NF, nonfat placebo; NFPS, nonfat with phytosterols; TC, total cholesterol.

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keeping with a heart-healthy diet. Few studies have examined the effects of phytosterols when provided in low-fat or nonfat foods (17–20). These trials have all found reductions in TC and LDL cholesterol with consumption of low-fat foods containing plant sterols; however, weaknesses in study design prevent strong conclusions from being drawn regarding the efficacy of low- or nonfat foods enriched with phytosterols in reducing plasma lipid concentrations. In addition, no study has examined the hypocholesterolemic potential of phytosterol-enriched beverages.

The objective of this trial was thus to assess whether phytosterols, when provided in a low-fat or nonfat beverage, elicit a hypocholesterolemic response in mildly hypercholesterolemic subjects compared with a control diet in which volunteers were provided identical foods. The null hypothesis was that there would be no difference in plasma lipid or plant sterol concentrations across the three diets.

## EXPERIMENTAL PROCEDURES

### Subjects

Fifteen subjects (nine men, six women), ages 22 to 68 years, were recruited to participate in this crossover, placebo-controlled, double-blind feeding trial of three experimental phases. Subjects were selected based on screening values for LDL cholesterol between 3.25 and 6.0 mmol/l, HDL cholesterol >0.8 mmol/l, triacylglycerol (TAG) concentrations <4.0 mmol/l, and body mass index between 22 and 32 kg/m<sup>2</sup>. Subjects were excluded if they were taking lipid-lowering medication or phytosterols within 6 weeks prior to screening, fibrates within 8 weeks prior to screening, probucol in the year prior to screening, or experimental medication 30 days prior to screening. Also, subjects were excluded if they were diagnosed with hypothyroidism; diabetes Types I, III, IV, or V; hyperlipidemia or familial hypercholesterolemia; had uncontrolled hypertension; had a history of drug or alcohol abuse; had a history of liver, kidney, or hematological disease; or had familial sitosterolemia, gastrointestinal disorders, iliac bypass surgery, heart disease, or unstable angina in the 3 months prior to screening. The study protocol was reviewed and approved by the Human Ethical Review Committee of the Faculty of Agriculture and Environmental Sciences of McGill University. The research goals and procedures were explained to each subject prior to the start of the study, and all subjects signed informed consent forms.

### Protocol and diets

Subjects were randomly allocated to one of six treatment sequences. Each treatment phase was of 21 days duration and separated by a 4 week washout period. Treatment phases were controlled feeding periods differing only in the type of beverage supplied, either nonfat placebo (NF), nonfat with phytosterols (NFPS), or low-fat (1% fat by weight) with phytosterols (LFPS). The nonfat beverages contained 85.8% water, 0.3% protein, less than 0.1% fat, 13.6% carbohydrates, and 0.2% fiber. Each serving of the nonfat beverages provided 220 kJ (52 kcal). The low-fat beverage contained the same protein, carbohydrate, and fiber content, but contained 1.0% fat and provided 238 kJ (61 kcal) per 100 g serving. The LFPS and NFPS test drinks were produced by adding plant sterol materials directly into beverages using a proprietary technique. Subjects consumed three 100 g

beverages/day. The mixture of phytosterols provided in the beverages was derived from tall oil and contained ~60% sitosterol, as well as some sitostanol and campestanol, with an overall phytosterol purity of >99%.

Diets were controlled and contained 35% of energy as fat, 15% as protein, and 50% as carbohydrate. Meals followed typical North American habits and included chicken breast and vegetables, pizza, ham sandwich, beef stir-fry, and spaghetti. Diets were provided as three isoenergetic meals and were served with the treatment beverage in equal dosages. All meals were prepared by the staff of the Mary Emily Clinical Nutrition Research Unit (CNRU) of McGill University. Each subject's weight-maintaining energy intake was individually calculated using the Mifflin equation (21) with an activity factor of 1.7. This factor is considered appropriate for weight maintenance in moderately active subjects (22). Subjects were required to consume all foods provided and nothing else. In addition, two meals per day were consumed under supervision at the CNRU, and the other meal was packed for subjects to consume outside of the CNRU.

Complete physical exams were conducted at screening and at the end of the entire study period to detect the presence of hematological or biochemical abnormalities. Brief physical exams were also conducted at the beginning and end of each treatment phase. A physician was available throughout the study in case subjects experienced discomfort.

### Lipid and phytosterol analyses

Fasting blood samples were taken on Days 1, 2, 7, 14, and 21 of each treatment phase for determination of plasma lipid and phytosterol concentrations. Plasma was separated from red blood cells after 15 min of centrifugation at 520 *g* and stored at –80°C until analysis. Plasma TC, HDL cholesterol, and triacylglycerols were analyzed in duplicate using a VP Autoanalyzer and commercial enzymatic kits (Abbott Laboratories, North Chicago, IL). HDL cholesterol concentrations were measured after precipitation of the apolipoprotein B lipoproteins with dextran sulfate and magnesium chloride (23). LDL cholesterol concentrations were calculated using the Friedewald equation (24).

Plasma phytosterol concentrations were analyzed in duplicate by gas-liquid chromatography of the nonsaponifiable materials extracted from plasma lipids, as reported previously (25). Briefly, 0.5 ml of plasma was saponified with 0.5 mol/l methanolic KOH at 100°C for 1 h and the nonsaponifiable materials extracted with petroleum ether. 5- $\alpha$ -Cholestane was used as the internal standard. Samples were injected into a gas-liquid chromatograph equipped with a flame ionization detector (HP 5890 Series II, Hewlett Packard, Palo Alto, CA) and a 30 m capillary column (SAC-5, Supelco, Bellefonte, PA). Detector and injector temperatures were set at 310°C and 300°C, respectively. Duplicate samples were analyzed isothermally at 285°C. Phytosterol peaks were identified by comparison with authenticated standards (Supelco).

### Statistical analyses

Plasma lipid concentrations were analyzed using crossover repeated measures ANOVA with sequence, phase, subject, carry over, and dietary treatment as main effect variables. Baseline values were included as covariates in the analyses of posttreatment absolute values and change from baseline. When significant differences were found for main diet effect, Tukey posthoc tests were used to compare treatment means. Student's paired *t*-tests were used to compare baseline and endpoint values between each dietary treatment. Plasma phytosterol concentrations were analyzed using crossover ANOVA with sequence, subjects, phase, carry over, and treatment as main effects variables. Paired Student's *t*-test was used to compare baseline and endpoint values

for plasma phytosterol and phytosterol-cholesterol ratio within dietary treatment phases. A *P* value of 0.05 was taken as statistically significant. All data are reported as means ± SEM.

## RESULTS

All subjects commencing the study completed the research protocol. Compliance was considered high, because all subjects consumed two meals per day under supervision at the CNRU and reported consuming all of the other meals and beverages. Body weights were unchanged during the trial and there was no difference between dietary treatments on body weights (**Table 1**). Seven (47%) subjects reported a total of eight adverse events (AEs) after screening, five of which occurred during the NF phase and three during the LFPS phase. However, all of the AEs were considered nonserious and unrelated to study treatment. The majority were head colds (*n* = 4, 50%) or respiratory infections (*n* = 3, 37.5%). The remaining were gastrointestinal problems (*n* = 1, 6.7%), a tooth infection (*n* = 1, 6.7%), and a urinary tract infection (*n* = 1, 6.7%). Systolic and diastolic pressures, as well as pulse rate, were not significantly affected by treatment. All endpoint values were similar between treatment groups and not different from baseline values.

Mean baseline and endpoint concentrations as well as percent changes in TC, LDL cholesterol, HDL cholesterol, and TAG concentrations over the 21 day experimental periods are shown in **Table 2**. There was no significant difference in TC and LDL cholesterol concentrations among treatment groups at endpoint. TC concentrations at endpoint were 5.60 ± 0.24, 5.54 ± 0.29, and 5.34 ± 0.25 mmol/l with NF, NFPS, and LFPS beverages, respectively. These endpoint values correspond to reductions in TC concentrations from a baseline of 8.5%, 11.6%, and 10.1% for NF, NFPS, and LFPS, respectively. LDL cholesterol concentrations were also not significantly different between treatments at endpoint. Endpoint LDL cholesterol concentrations after NF, NFPS, and LFPS consumption were 3.80 ± 0.23, 3.73 ± 0.28, and 3.55 ± 0.21 mmol/l, respectively. These endpoint values correspond to reductions in LDL cholesterol concentrations from baseline of 5%, 10.4%, and 8.5% for NF, NFPS, and LFPS, respectively. All treatments resulted in decreases (*P* < 0.05) in TC and LDL cholesterol over time (**Fig. 1**).

Endpoint HDL cholesterol and TAG concentrations were not significantly different between dietary treat-

TABLE 2. Plasma lipid concentrations with dietary treatments with and without phytosterols<sup>a</sup>

	NF	NFPS	LFPS
Total cholesterol			
Baseline, mmol/l	6.12 ± 0.24	6.27 ± 0.35	5.94 ± 0.24
Endpoint, mmol/l	5.60 ± 0.24 <sup>b</sup>	5.54 ± 0.29 <sup>b</sup>	5.34 ± 0.25 <sup>b</sup>
Change, %	-8.5	-11.6	-10.1
LDL cholesterol			
Baseline, mmol/l	4.00 ± 0.24	4.15 ± 0.25	3.88 ± 0.21
Endpoint, mmol/l	3.80 ± 0.23 <sup>b</sup>	3.72 ± 0.28 <sup>b</sup>	3.55 ± 0.21 <sup>b</sup>
Change, %	-5.0	-10.4	-8.5
HDL cholesterol			
Baseline, mmol/l	1.18 ± 0.06	1.21 ± 0.07	1.19 ± 0.07
Endpoint, mmol/l	1.08 ± 0.07 <sup>b</sup>	1.10 ± 0.05 <sup>b</sup>	1.16 ± 0.06
Change, %	-8.5	-9.1	-2.5
Triacylglycerols			
Baseline, mmol/l	2.04 ± 0.38	2.03 ± 0.36	1.93 ± 0.30
Endpoint, mmol/l	1.59 ± 0.18	1.61 ± 0.21	1.80 ± 0.32
Change, %	-22.1	-20.7	-6.7

<sup>a</sup> Data are means ± SEM.

<sup>b</sup> Significantly different from baseline within each diet, *P* < 0.05.

ments, with concentrations ranging from 1.08 to 1.16 mmol/l for HDL cholesterol and between 1.59 to 1.80 mmol/l for TAG. The NF and NFPS treatments caused a decrease (*P* < 0.05) of 8.5% and 9.1%, respectively, in HDL cholesterol concentrations from baseline. Ratios of LDL cholesterol-HDL cholesterol were unchanged by dietary treatments as reflected by similar endpoint values (3.67 ± 0.28, 3.48 ± 0.31, and 3.17 ± 0.22 with NF, NFPS, and LFPS consumption, respectively).

Plasma campesterol concentrations increased (*P* < 0.01) with consumption of LFPS diet versus NF and NFPS (**Table 3**). Values at endpoint were 31.5% higher (*P* < 0.01) relative to baseline with consumption of the LFPS diet. Similarly, plasma sitosterol concentrations increased (*P* < 0.01) with LFPS compared with NF and NFPS consumption. Endpoint concentrations were 37.7% higher (*P* < 0.01) than baseline for LFPS.

## DISCUSSION

This is the first published study to examine the functionality of phytosterols provided in the form of a beverage. Furthermore, it is one of the few trials to test the efficacy of phytosterol-containing nonfat and low-fat foods or beverages in lowering plasma lipid concentrations. The results presented here show that consumption of phytosterols at a level of 1.8 g/day does not lead to reductions in TC and LDL cholesterol concentrations when given in the form of a nonfat or low-fat beverage compared with a placebo drink not containing phytosterols. Given the new plant sterol formulations available on the market, including various foods and capsular preparations, these results show that previous cholesterol-lowering efficacy of plant sterols in full-fat and low-fat spreads may not be readily applied to these novel phytosterol formulations.

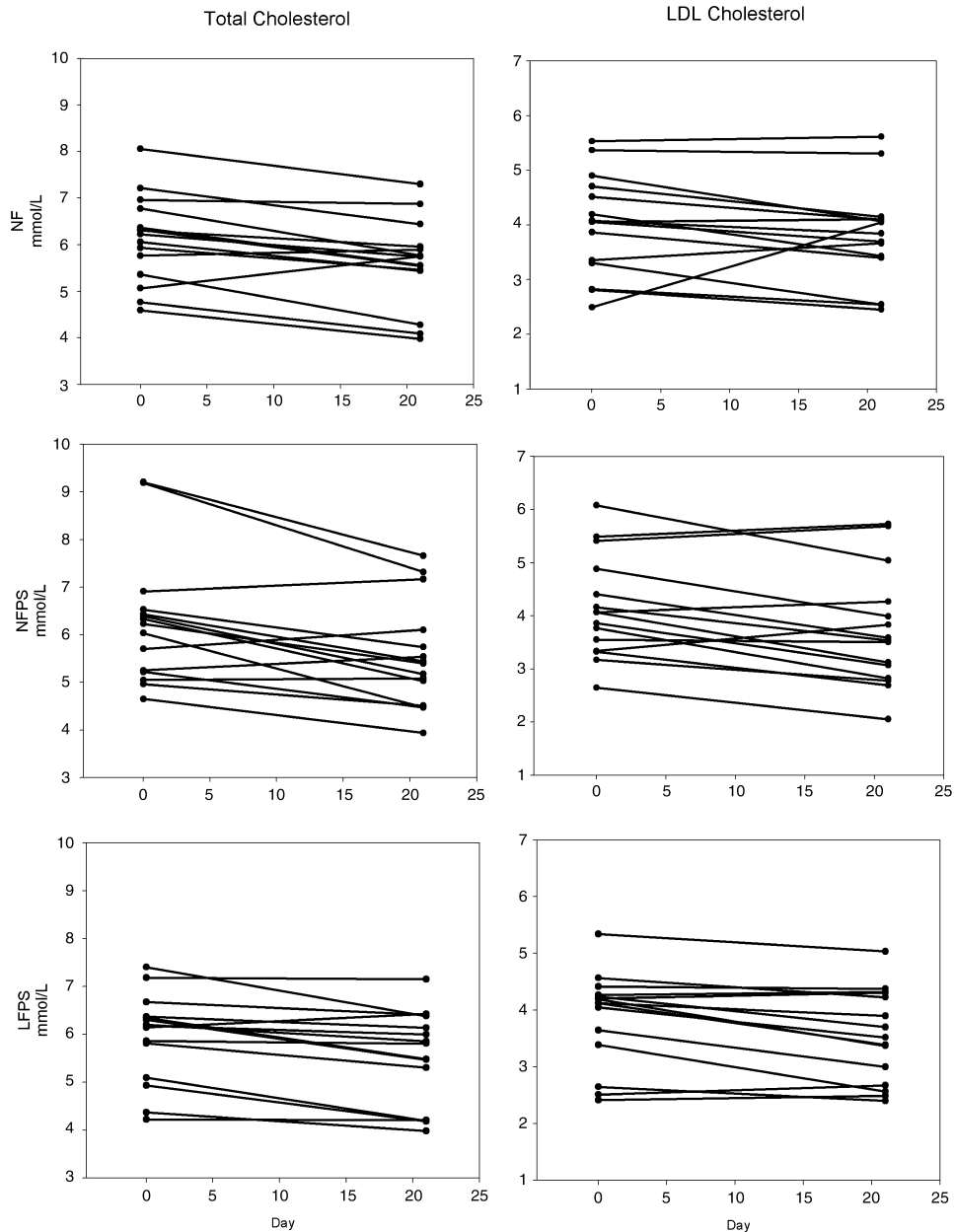
It is highly unlikely that the lack of efficacy of the phytosterol-containing functional beverages was due to weak-

TABLE 1. Mean body weights at baseline and after 21 days of consumption of diets with and without phytosterols<sup>a</sup>

Body Weight	NF	NFPS	LFPS
		kg	
Day 1	80.3 ± 4.2	80.2 ± 4.2	80.3 ± 4.3
Day 21	80.0 ± 4.1	80.1 ± 4.1	80.2 ± 4.1

NF, nonfat; NFPS, nonfat with phytosterols; LFPS, low-fat with phytosterols.

<sup>a</sup> Data are means ± SEM.



**Fig. 1.** Effect of phytosterol-enriched nonfat placebo (NF), nonfat with phytosterol (NFPS), and low-fat with phytosterols (LFPS) beverages over 21 days on individual total and LDL cholesterol concentrations.

nesses in study design. Diets were strictly controlled and identical in macronutrient composition. Furthermore, lack of compliance with the study protocol is improbable. All subjects consumed two meals per day under supervision at the CNRU, and thus at least two thirds of the total daily phytosterol dose was consumed. In addition, the lack of change in body weight suggests that subjects consumed all of the food provided and nothing else. However, there is still a chance that subjects substituted study foods for nonstudy foods. In that case, only 1.2 g of phytosterols would have been consumed. This level of plant sterols or lower has been shown to produce significant reductions in plasma LDL cholesterol concentrations (26–28). Inappropriate dosage of phytosterols is therefore not likely. Each

phytosterol-containing beverage provided 0.6 g of phytosterols for a total daily dose of 1.8 g, a quantity determined by our group (8, 9, 29, 30) and others (3, 6, 7, 11) to be sufficient to elicit a hypocholesterolemic response. This level of consumption of plant sterols, when dispersed in a fat matrix, has been shown to reduce TC and LDL cholesterol concentrations by an average of 10% and 13%, respectively (16).

Lack of adequate power to detect a difference between groups is also unlikely. Based on previous research from our group, a difference of 0.56 mmol/l in endpoint LDL cholesterol concentrations would have been expected between the phytosterol-containing diets and the control diet (8). Using the standard deviation obtained in this

TABLE 3. Plasma campesterol and sitosterol concentrations with dietary treatments with and without phytosterols

	NF	NFPS	LFPS
Campesterol			
Baseline, mmol/l	0.0194 ± 0.003	0.021 ± 0.003	0.019 ± 0.003
Endpoint, mmol/l	0.0195 ± 0.003	0.022 ± 0.003	0.025 ± 0.003
Change, %	-0.5	4.8	31.5 <sup>a</sup>
Sitosterol			
Baseline, mmol/l	0.0074 ± 0.0011	0.0090 ± 0.0012 <sup>b</sup>	0.0077 ± 0.0011
Endpoint, mmol/l	0.0074 ± 0.0010	0.0085 ± 0.0012	0.0106 ± 0.0012 <sup>a</sup>
Change, %	0	-5.6	37.7 <sup>a</sup>
Endpoint campesterol-cholesterol ratio	3.40 ± 0.53	3.64 ± 0.53	4.53 ± 0.60
Endpoint sitosterol-cholesterol ratio	1.30 ± 0.16	1.57 ± 0.21	1.95 ± 0.21
Endpoint campesterol-sitosterol ratio	2.69 ± 0.12	2.51 ± 0.10	2.43 ± 0.10

<sup>a</sup> Significantly different from NF and NFPS treatments,  $P < 0.01$ .

<sup>b</sup> Significantly different from NF and LFPS treatments,  $P < 0.01$ .

previous study, our sample size was sufficient to detect a difference of 0.48 mmol/l with 95% confidence and 80% power. It cannot be ruled out that a greater sample size may have permitted identification of statistically significant differences between treatment groups.

The significant decrease in HDL cholesterol concentrations with NF and NFPS, but not LFPS, was somewhat surprising since previous studies have not shown any effect of plant sterol or stanol consumption on HDL cholesterol concentrations (8, 9, 11, 30). However, nonsignificant differences in changes in TAG concentrations between diets may be partly responsible for this effect. Both NF and NFPS caused 20–22% reductions (NS) in TAG, whereas LFPS reduced TAG by 6.7% (NS).

Most recent studies conducted to examine the lipid-lowering potential of phytosterols incorporated them into a fat matrix: margarine, butter, or dressing. Results from these trials have shown that phytosterol consumption decreases TC and LDL cholesterol concentrations by 3.4% to 11.6% for TC and 5.4% to 15.5% for LDL cholesterol (6–9, 31, 32). Lately, trials have been conducted to establish whether incorporation of phytosterols into low-fat spreads and dressings results in diminished cholesterol concentrations. Maki et al. (26) have reported lowering of TC concentrations by 5.2% and 6.6% with a 50% fat spread providing 1.1 g or 2.2 g phytosterols/day, respectively, compared with a control reduced-fat spread and LDL cholesterol lowering of 7.6 and 8.1%. However, in a trial comparing the effects of 3 g phytosterols/day in a reduced-fat spread versus 6 g phytosterols/day in a 28% fat dressing, and 9 g phytosterols/day provided in reduced-fat spread and dressing (33), there was no difference in cholesterol concentrations in any of the three groups compared with control. Reductions similar to those observed by Davidson et al. (33) had previously been observed when using mayonnaise as a vehicle for phytosterol administration (34, 35). These studies show a high degree of variability in response to phytosterols incorporated into reduced fat matrices.

More recent studies have examined the effects of phytosterols on plasma lipid concentrations when these were incorporated into low-fat or nonfat foods. Of the four studies conducted to date (16–20), all have shown reductions in plasma lipid concentrations. The results of these studies sug-

gest that the variety of functional foods that can be produced with phytosterols as a means of lowering circulating cholesterol concentrations can be increased to include low-fat food items. However, several weaknesses in study design prevent firm conclusions from being drawn. In the study by Nestel et al. (18), one third of the daily phytosterol dose, 0.8 g, was provided in margarine, and the rest incorporated in equal amounts in bread and breakfast cereals. Since 0.8 g phytosterols/day has been shown to be sufficient to elicit a hypocholesterolemic response (34–36), it is possible that the margarine alone produced cholesterol-lowering with the other low-fat foods not contributing to the reductions observed. In the study by Tikkanen et al. (19), the fat content of the foods in which phytosterols were incorporated were not provided. In a third trial (20), significant reductions in TC and LDL cholesterol concentrations of 6.7% and 11.1%, respectively, relative to baseline were observed with consumption of a low-fat (1% fat by weight) yogurt enriched with 1 g of phytosterols. However, there is no mention of the results of a statistical comparison between the phytosterol-containing yogurt and the placebo, which caused a nonsignificant lowering of 2.3% and 4.9% in TC and LDL cholesterol concentrations, respectively. More substantial decreases in TC and LDL cholesterol concentrations of 8.7% and 13.7%, respectively, relative to placebo, were observed by Mensick et al. (17) when providing ~3 g/day of esterified stanols in low-fat yogurts. However, since stanols were esterified, it is uncertain whether similar results would be obtained using free stanols. In the present trial, consumption of free sterols in low- and nonfat beverages did not lead to reductions in TC and LDL cholesterol concentrations relative to the control beverage. A previous report (37) showed decreased cholesterol absorption with consumption of 3 g/day nonesterified stanols in beverage format; however, the short duration of the feeding period, 4 days, prevented conclusions to be made regarding its efficacy in lowering plasma cholesterol concentrations.

There is thus some controversy regarding the true efficacy of nonfat and low-fat foods enriched in plant sterols in reducing plasma lipid concentrations. In the present trial, phytosterols were incorporated into beverages containing either no fat or 1 g of fat/serving. This quantity of fat may not have been sufficient to solubilize phytosterols. It has previously been shown that free sitostanols are not as

effective in reducing cholesterol absorption as lecithin-coated sitosterols (14). Solubility of phytosterols is thus an important determinant of plant sterol efficacy. Also, the use of a liquid as vehicle for phytosterols may not be optimal for their incorporation into micellar contents. Gastric emptying of liquid portions of a meal is more rapid than for solids (38). Liquid emptying is not affected by the presence of solid foods or fats in the meals, whereas gastric emptying of the oil phase is altered by both the quality and quantity of dietary fats (38). Thus, plant sterols consumed in solid foods have more time to be thoroughly mixed with gastrointestinal contents, which may in turn provide better access to mixed micelles in the small bowel.

Consumption of only the LFPS but not NFPS resulted in elevations in plasma plant sterol concentrations in the present trial. From previous controlled feeding studies, it would have been expected that both phytosterol-containing diets would increase plasma plant sterol concentrations (8, 30). Since there was no increase in plasma phytosterol concentrations with NFPS consumption, it is possible that fat may have been necessary to promote micellar absorption of phytosterols. In the case of the LFPS diet, perhaps the fat content enabled some phytosterol absorption, but at a level too low to activate mechanisms that lower cholesterol absorption. Thus, there may exist a threshold level for phytosterol absorption to elicit cholesterol lowering, which was not attained with these low- and nonfat phytosterol formulations.

In conclusion, this study shows that unesterified phytosterols, when provided in low-fat and nonfat beverages, do not lead to greater reductions in TC and LDL cholesterol compared with a control diet. The cholesterol-lowering potential of phytosterols may thus depend upon previous dispersion into a fat matrix and on the physical nature of the food. Alternatively, esterification may be necessary for the cholesterol-lowering action of phytosterols when they are incorporated into liquids. Results from this trial show that, despite changes in plasma phytosterol levels, incorporation of plant sterols in a beverage format is not effective in promoting cholesterol reductions compared with a control beverage. It is therefore important that previous data concerning the cholesterol-lowering efficacy of plant sterols not be liberally applied to all new phytosterol formulations. Research is thus warranted to determine the efficacy of such new products.

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