Combined effects of a dietary portfolio of plant sterols, vegetable protein, viscous fibre and almonds on LDL particle size

Benoît Lamarche1*, Sophie Desroches1, David J. A. Jenkins2,3,4, Cyril W. C. Kendall2,4, Augustine Marchie2,4, Dorothea Faulkner2,4, Edward Vidgen2,4, Karen G. Lapsley8, Elke A. Trautwein9, Tina L. Parker2,4, Robert G. Josse2,3,4,5, Lawrence A. Leiter2,3,4,5 and Philip W. Connelly3,6,7

1Institute on Nutraceuticals and Functional Foods, Laval University, Ste-Foy, Québec, Canada
2Clinical Nutrition and Risk Factor Modification Center and
3Department of Medicine, Division of Endocrinology and Metabolism, St Michael’s Hospital, Toronto, Ontario, Canada
4Departments of 5Nutritional Sciences, 6Medicine, 7Biochemistry and 8Laboratory Medicine and Pathobiology, Faculty of Medicine, University of Toronto, Toronto, Ontario, Canada
5The Almond Board of California, Modesto, California, USA
6Unilever Health Institute, Unilever R&D Vlaardingen, The Netherlands

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Studies conducted in the last 20 years have led to the identification of small dense LDL as an important risk factor for CVD. Consumption of plant sterols, soyabean proteins, viscous fibre and nuts are known to modulate the risk of CVD favourably through their cholesterol (Chol)-lowering properties, both independently and more recently in combination. Nevertheless, their combined impact on the LDL particle size phenotype has never been tested. In the present study, we assessed the effect of incorporating concurrently plant sterols (1 g/4.2 MJ), soyabean protein (23 g/4.2 MJ), viscous fibre (9 g/4.2 MJ) and almonds (15 g/4.2 MJ) into a diet very low in saturated fat in twelve patients with mildly elevated plasma LDL-Chol levels. Fasting blood lipids were obtained at the start of the study and at 2-week intervals during the 4-week study. The diet-induced reduction in plasma LDL-Chol of 30.0 (SE 3.0) % (P < 0.001) and small (<25.5 nm, 0.001) LDL particles, with near maximal reductions seen by week 2. These results indicate that foods and dietary components advocated for their potential to reduce the risk of CVD are effective in reducing serum concentrations of all LDL fractions including small dense LDL, thus potentially further contributing to an overall lower risk of CVD.

LDL particle size: Plant sterols: Soyabean protein: Viscous dietary fibre: Almonds

Small dense LDL particles have been associated with an increased risk of CVD (Gardner et al. 1996; Lamarche et al. 1997, 1999). Whether measurement of LDL particle size or phenotype provides additional or complementary information on CVD risk, independent of more traditional lipid risk factors such as plasma LDL-cholesterol (Chol) or triacylglycerol (TG) levels, remains a matter of debate (Lamarche et al. 1999). Recent results from the Québe Cardiovascular Study have provided new insights into the relationship between small dense LDL and the risk of CVD (St Pierre et al. 2001). First, LDL peak particle diameter, which has been used widely as a surrogate for small dense LDL particles in most previous epidemiological studies, appeared as a weak multivariate predictor of CVD risk. However, other electrophoretic characteristics of LDL particles, such as the proportion of small LDL particles (LDL<sub>%</sub>&lt;255 nm) and the Chol concentration of LDL particles <25.5 nm (LDL-Chol<sub>255 nm</sub>) were identified as very powerful and independent risk predictors in middle-aged men, even after adjustment for other variables of the CVD risk profile, including the more traditional measure of the LDL-peak particle diameter (PPD; St Pierre et al. 2001).

Recent studies from our group (Jenkins et al. 2002, 2003) have demonstrated the potent Chol-lowering properties of a combination (portfolio) diet simultaneously incorporating viscous fibre, plant sterols, vegetable protein (soyabean) and nuts (almonds), four dietary components that have been accepted for health claims by the Food and Drug Administration in the USA (US Department of Agriculture, 2003). There is currently only limited data on the individual effect that these dietary components may have on the LDL particle size phenotype (Merz-Demlow et al. 2000; Almario et al. 2001; Wangen et al.)

Abbreviations: Chol, cholesterol; PPD, peak particle diameter; TG, triacylglycerol.
* Corresponding author: Dr Benoît Lamarche, fax +1 418 656 5877, email benoit.lamarche@inaf.ulaval.ca
Materials and methods

Subjects

Data on the impact of the combination diet on LDL-Chol and on several cardiovascular risk factors have been reported earlier (Jenkins et al. 2002, 2003). For the present analysis, LDL particle size phenotype was obtained in twelve of the thirteen original subjects (six men and six postmenopausal women), aged 65 (SE 3) (range 43–84) years with a mean BMI 25·8 (SE 3·4) (range 20·6–30·7) kg/m². Their mean baseline LDL-Chol level was 4·20 (SE 0·12) (range 3·51–4·99) mmol/l. Briefly, participants were recruited from patients attending the Risk Factor Modification Center, St Michael’s Hospital, Toronto, Ont., Canada. All subjects had taken part in previous dietary studies and were experienced in following dietary protocols. The main inclusion criteria were to previously have had raised LDL-Chol levels (>4·1 mmol/l), to have a BMI <38 kg/m² and to live within 16 km of St Michael’s Hospital for practical purposes. At the time of the study, five subjects had raised LDL-Chol levels, one subject had raised TG levels (>2·4 mmol/l), three subjects had both raised Chol and TG levels, one subject had low HDL-Chol levels (<0·9 mmol/l) and three subjects had blood lipids in the normal range. No subjects had a history of diabetes, renal or liver disease and none were taking medications known to influence serum lipids.

Study protocol

Subjects were monitored on their own usual low-saturated fat therapeutic diets for 1 week preceding the 4-week intervention with the combination diet, for which most foods were provided to participants (Jenkins et al. 2002). This allowed us to document the nature of the diet that participants had been following in the preceding months and that would be reflected in the blood sample obtained at the end of that ‘run-in’ week. A 2-week washout period, during which participants returned to their low-saturated fat therapeutic diets, followed the combination diet. Blood samples and body weights were obtained after 12 h overnight fasts at 1-week intervals and at week 2 of the washout period. Weighed diet histories (7 d), during which subjects weighed all the food they consumed, were obtained for 7 d prior to and 2 weeks following the combination diet, as described previously (Jenkins et al. 2002). During the 4 weeks on the combination diet, completed menu checklists were returned at weekly intervals.

The study was approved by the Ethics Committee of the University of Toronto and St Michael’s Hospital and informed consent was obtained from all the subjects.

Diets

The diets eaten before and after the 4-week combination diet were the subjects’ routine therapeutic low-fat diets, which followed the National Cholesterol Education Program Step 2 guidelines (<7 % energy from saturated fat and <200 mg dietary Chol/d) (Table 1) (National Cholesterol Education Program, 2002). Subjects were provided with self-taring electronic scales and asked to weigh all food items consumed during the study period. During the combination diet period, all foods to be consumed by the subjects were provided at weekly clinic visits with the exception of fruits and low-energy vegetables, i.e. non-starch-containing vegetables, which subjects were instructed to obtain from their local stores as previously described (Jenkins et al. 2002, 2003). A 4-week intervention period was judged adequate based on previous work from our

Table 1. Calculated macronutrient intakes during the baseline, test and run-out periods of the combination diet

<table>
<thead>
<tr>
<th></th>
<th>Baseline (week 0)</th>
<th>Test (mean of weeks 2–4)</th>
<th>Run-out (week 6)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
</tr>
<tr>
<td>Energy (MJ/d)</td>
<td>7·19a</td>
<td>1·73</td>
<td>8·36b</td>
</tr>
<tr>
<td>Total protein (% energy)</td>
<td>17·3a</td>
<td>2·8</td>
<td>22·4b</td>
</tr>
<tr>
<td>Vegetable protein (% protein)</td>
<td>48·7a</td>
<td>12·1</td>
<td>96·8c</td>
</tr>
<tr>
<td>Available carbohydrate (% energy)</td>
<td>52·9ab</td>
<td>9·7</td>
<td>50·6a</td>
</tr>
<tr>
<td>Total dietary fibre (g/4·2 MJ)</td>
<td>17·1a</td>
<td>6·6</td>
<td>30·7b</td>
</tr>
<tr>
<td>Total fat (% energy)</td>
<td>28·3</td>
<td>8·7</td>
<td>27·0</td>
</tr>
<tr>
<td>SFA (% energy)</td>
<td>7.7c</td>
<td>2·4</td>
<td>4·3a</td>
</tr>
<tr>
<td>MUFA (% energy)</td>
<td>11·9</td>
<td>5·5</td>
<td>11·8</td>
</tr>
<tr>
<td>PUFA (% energy)</td>
<td>6·0a</td>
<td>1·4</td>
<td>9·9f</td>
</tr>
<tr>
<td>Dietary cholesterol (mg/4·2 MJ)</td>
<td>99ab</td>
<td>45</td>
<td>10b</td>
</tr>
<tr>
<td>Alcohol (% energy)</td>
<td>1·5a</td>
<td>1·7</td>
<td>0·2b</td>
</tr>
</tbody>
</table>

SFA, saturated fatty acids.

a,b Mean values within a row with unlike superscript letters were significantly different (P<0·05).

* For details of subjects and procedures, see pp. 658–659.
group, which has shown that near maximum falls in serum Chol occurred within the first week of the dietary regimen (Wolvere et al. 1997; Jenkins et al. 2001).

The aim of the combination diet was to provide: 1 g plant sterols/4·2 MJ as an enriched margarine, 8·2 g viscous fibre/4·2 MJ from oats, barley and psyllium, and 22·7 g soyabean protein/4·2 MJ. Raw unblanched almonds also provided vegetable protein (2·9 g/4·2 MJ). Emphasis was placed on aubergine and okra as additional sources of viscous fibre (0·55 g/4·2 MJ and 0·67 g/4·2 MJ respectively). Thus, 200 g aubergine and 100 g okra/d were prescribed to be eaten on a 8·4 MJ/d diet. Weight-maintaining diets were provided based on estimated energy requirements. Diets were analysed using a program based on US Department of Agriculture data; additional values were obtained from foods analysed in the laboratory for protein, total fat and dietary fibre using AOAC methods (Association of Official Analytical Chemists, 1980) and fatty acids by GC (Jenkins et al. 2002). Additional dietary fibre values were obtained from the tables of Anderson & Bridges (1988). Compliance was assessed from the completed weekly checklists and from the return of uneaten food items.

Analyses

Serum was analysed according to the Lipid Research Clinics protocols (US Department of Health & Human Services, 1982) for total Chol, TG and HDL-Chol levels, after dextran sulfate–MgCl₂ precipitation (Warnick et al. 1982) for total Chol, TG and HDL-Chol levels, after Clinics protocols (US Department of Health & Human Services, 1982). Analyses from the return of uneaten food items. was assessed from the completed weekly checklists and from the tables of Anderson & Bridges (1988). Compliance dietary fibre using AOAC methods (Association of Official Analytical Chemists, 1980) and fatty acids by GC (Jenkins et al. 2002). Additional dietary fibre values were obtained from the tables of Anderson & Bridges (1988). Compliance was assessed from the completed weekly checklists and from the return of uneaten food items.

Results

As reported previously, compliance in terms of energy intake was good in most subjects, with a mean value of 92·5 (SD 2·9) % of the prescribed energy consumed (Jenkins et al. 2002). The mean energy intake was greater on the combination diet compared with the baseline and run-out values. The combination diet was associated with a slight body-weight reduction after 4 weeks compared with baseline values (mean Δweightweek 2þ4 = 0·000 ± 0·40 (SD 0·80) kg), which was accentuated during the washout period (mean Δweightweek 6 = 0·000 ± 0·20 (SD 0·40) kg). Subjects lost body weight at an average rate of −0·10 (SD 0·05) kg per week on the experimental combination diet and −0·02 (SD 0·05) kg per week during the run-out phase.

Blood lipids

Full details of the blood lipid responses have been reported previously (Jenkins et al. 2002). Significant reductions in blood lipids were seen during the experimental diet compared with the baseline (Table 2), including reductions from baseline in LDL-Chol levels (30·0 (SE 3·0) %, P<0·0001) and the total Chol: HDL-Chol ratio (18·6 (SE 3·1) %, P<0·0001, results not shown).

LDL size phenotype

As shown in Table 2, the combination diet had no significant effect on LDL-PPD. On the other hand, the LDL integrated size, which reflects the whole distribution of LDL based on all subclasses in a given individual, was significantly reduced (P<0·01) following the combination diet, suggesting a shift in the distribution of LDL from larger to smaller species. This was confirmed by densitometric
LDL particle size phenotype. As shown in Fig. 1, subjects with increased LDL-Chol$_{25·5	ext{ nm}}$ levels at baseline experienced a significant reduction ($P<0·0001$) in LDL-Chol$_{25·5	ext{ nm}}$ concentrations and showed an increase in LDL-PPD following the experimental diet. Total plasma LDL-Chol levels were also reduced significantly in this group of subjects ($-31·4\%$, $P<0·01$). The further increase in LDL-PPD during the washout phase in subjects with LDL-Chol$_{25·5	ext{ nm}}$ levels $>1·72\text{ mmol/l}$ may be explained by the fact that these subjects showed a greater decrease in body weight (run-out v. baseline) compared with those with LDL-Chol$_{25·5	ext{ nm}}$ $<1·72\text{ mmol/l}$ at baseline (1·53 v. 0·96 kg respectively, results not shown). On the other hand, in subjects with relatively lower LDL-Chol$_{25·5	ext{ nm}}$ levels at baseline, the diet had virtually no impact on LDL-Chol$_{25·5	ext{ nm}}$ levels and LDL-PPD was reduced by 0·3 nm. However, the magnitude of the diet-induced reduction in total plasma LDL-Chol levels ($-28·6\%$, $P<0·0001$) was not attenuated in this subgroup of subjects compared with individuals with high levels of LDL-Chol$_{25·5	ext{ nm}}$ at baseline.

**Discussion**

The results from the present study have been reported previously, showing that a diet incorporating soyabean protein, phytosterols, viscous fibre and almonds to the diet of hypercholesterolaemic subjects produced some of the largest reductions in LDL-Chol reported for largely weight-maintaining dietary interventions (Jenkins et al. 2002, 2003). The question therefore arose as to whether the total LDL-Chol reduction was also reflected in a corresponding reduction in the levels of the most atherogenic LDL particles (LDL with diameter $<25·5\text{ nm}$) (St Pierre et al. 2001).

Despite a reduction in the proportion of larger LDL particles on the combination diet leading to a relative increase in the proportion of small LDL particles and a

<table>
<thead>
<tr>
<th>Variable</th>
<th>Baseline (week 0)</th>
<th>Mean treatment (weeks 2–4)</th>
<th>Run-out (week 6)</th>
<th>Statistical significance of effect: $P$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight (kg)</td>
<td>70·0 ± 13·7</td>
<td>69·5 ± 13·1</td>
<td>68·7 ± 13·0</td>
<td>0·02</td>
</tr>
<tr>
<td>Total cholesterol (mmol/l)†</td>
<td>6·37 ± 0·80</td>
<td>4·97 ± 0·76</td>
<td>5·95 ± 0·82</td>
<td>$&lt;0·0001$</td>
</tr>
<tr>
<td>LDL-cholesterol (mmol/l)</td>
<td>4·20 ± 0·42</td>
<td>2·95 ± 0·63</td>
<td>3·82 ± 0·76</td>
<td>$&lt;0·0001$</td>
</tr>
<tr>
<td>HDL-cholesterol (mmol/l)</td>
<td>1·38 ± 0·41</td>
<td>1·33 ± 0·43</td>
<td>1·34 ± 0·44</td>
<td>0·44</td>
</tr>
<tr>
<td>Triacylglycerol (mmol/l)‡</td>
<td>1·87 ± 1·31</td>
<td>1·52 ± 0·76</td>
<td>1·72 ± 1·01</td>
<td>0·04</td>
</tr>
<tr>
<td>Apo B (g/l)</td>
<td>1·31 ± 0·17</td>
<td>1·00 ± 0·19</td>
<td>1·26 ± 0·20</td>
<td>$&lt;0·0001$</td>
</tr>
<tr>
<td>LDL particle size phenotype</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LDL-peak particle diameter (Å)</td>
<td>255·4 ± 5·3</td>
<td>255·4 ± 4·3</td>
<td>256·3 ± 4·7</td>
<td>0·98</td>
</tr>
<tr>
<td>LDL-integrated size (Å)</td>
<td>257·0 ± 4·0</td>
<td>255·7 ± 2·9</td>
<td>256·0 ± 3·9</td>
<td>$&lt;0·01$</td>
</tr>
<tr>
<td>LDL$_{&gt;260\text{nm}}$§</td>
<td>37·8 ± 13·4</td>
<td>33·9 ± 8·0</td>
<td>38·2 ± 10·0</td>
<td>$&lt;0·05$</td>
</tr>
<tr>
<td>LDL$_{25·5–260\text{nm}}$§</td>
<td>19·1 ± 3·1</td>
<td>19·6 ± 3·0</td>
<td>20·4 ± 5·1</td>
<td>0·35</td>
</tr>
<tr>
<td>LDL$_{&lt;25·5\text{nm}}$§</td>
<td>43·1 ± 15·8</td>
<td>46·5 ± 11·3</td>
<td>41·5 ± 14·1</td>
<td>0·05</td>
</tr>
<tr>
<td>LDL-Chol$_{26\text{nm}}$ (mmol/l)</td>
<td>1·55 ± 0·49</td>
<td>0·99 ± 0·24</td>
<td>1·45 ± 0·45</td>
<td>$&lt;0·0001$</td>
</tr>
<tr>
<td>LDL-Chol$_{25·5–26\text{nm}}$ (mmol/l)</td>
<td>0·80 ± 0·13</td>
<td>0·57 ± 0·14</td>
<td>0·78 ± 0·27</td>
<td>$&lt;0·0001$</td>
</tr>
<tr>
<td>LDL-Chol$_{&lt;25·5\text{nm}}$ (mmol/l)</td>
<td>1·85 ± 0·82</td>
<td>1·39 ± 0·56</td>
<td>1·59 ± 0·68</td>
<td>$&lt;0·01$</td>
</tr>
</tbody>
</table>

*For details of diets, subjects and procedures, see pp. 658–659.
† n 11.
‡ Values were log-transformed before statistical analyses.
§ Relative proportion of LDL with diameters $<25·0$, 22·5–26·0 or $<25·5\text{ nm}$ respectively.
P values were significantly different from those at baseline: *.

Chol, objects, diets and procedures, see pp. 658–659. LDL-Chol errors shown by vertical bars for twelve subjects. For details of sub-

A small weight loss occurred on the combination diet when energy intake was greater than when subjects were consuming their usual therapeutic diets ad libitum. We believe this discrepancy may be largely explained by the tendency to under-reporting often seen in dietary records of self-selected diets, which has been shown to account for as much as 20% total energy intake (Sawaya et al. 1996). It has been reported previously that body weight changes induced by exercise or diet were important determinants of concurrent changes in LDL-PPD (Williams et al. 1990) and that individuals with a predominance of small LDL experienced a greater responsiveness to dietary changes (Krauss & Dreon, 1995) and weight loss (Purnell et al. 2000).

In our previous analyses, we have shown that baseline total LDL-Chol concentrations or diet-induced variation in body weight did not appear to influence the lipoprotein-lipid response to the combination diet (Jenkins et al. 2002). Perhaps the most interesting finding of the present study is the fact that high-risk individuals (risk based on the LDL size phenotype at baseline) had a more favourable response to the combination diet compared with low-risk individuals. Our present results may be explained by the fact that subjects with LDL-Chol_{25.5 nm} levels >1.72 mmol/l at baseline also had smaller LDL-PPD at baseline and lost more weight compared with subjects with LDL-Chol_{25.5 nm} levels <1.72 mmol/l. Although the cut-off point that we used to define low- v. high-risk individuals was arbitrarily defined as the median LDL-Chol_{25.5 nm} levels in our present twelve subjects, results from the Québec Cardiovascular Study indicate that men with LDL-Chol_{25.5 nm} levels >1.72 mmol/l had a 60% greater risk of IHD compared with those with LDL-Chol_{25.5 nm} levels <1.72 mmol/l (B Lamarche, AC St Pierre, B Cantin, GR Dagenais and JP Després, unpublished results). In subjects with low levels of LDL-Chol_{25.5 nm} at baseline, the impact of the combination diet on LDL particle size phenotype was marginal. LDL-PPD decreased by 3 Å, but the on-diet value (25.6 nm) did not go below the cut-off point that we have identified as being the critical value below which the risk of CVD significantly increases in men (25.6 nm; Lamarche et al. 2001). In addition, plasma levels of LDL-Chol_{25.5 nm} were not altered (positively or negatively) by the experimental diet in patients with low levels of LDL-Chol_{25.5 nm} at baseline. While we fully recognise that these sub-analyses are based on small numbers of subjects with the need to reproduce them in larger cohorts of patients, our present results suggest that the combination diet may be of tremendous value clinically in high-risk patients presenting with one of the typical features of the metabolic syndrome, i.e. small dense LDL particles. The extent to which variations in body weight contributed to alterations in the LDL size phenotype associated with the combination diet will also have to be addressed in future studies.

There is currently limited data on the individual effects of viscous fibre, soyabean protein, phytosterols and almonds on the characteristics of the LDL particle size phenotype. Although perhaps counterintuitive, low-saturated fat diets have been shown to reduce LDL-PPD (Krauss &

![Fig. 1. Impact of the combination diet on the relative proportion of LDL-cholesterol (chol) with a diameter <25.5 nm (LDL-Chol_{25.5 nm}) levels (A) and LDL peak particle diameter (PPD, (B)) in subjects with high or low levels of LDL-Chol_{25.5 nm} at baseline. ▲ Baseline LDL-Chol_{25.5 nm}<1.72 mmol/l; ■ baseline LDL-Chol_{25.5 nm}>1.72 mmol/l; Values are means with their standard errors shown by vertical bars for twelve subjects. For details of subjects, diets and procedures, see pp. 658–659. LDL-Chol_{25.5 nm} corresponds to the Chol levels in the small LDL subfraction, i.e. LDL with a diameter <25.5 nm and 1.72 mmol/l is the median value at baseline among the study participants. There were a significant group x treatment interactions: (A), P = 0.02; (B), P = 0.002. Mean values were significantly different from those at baseline: *P<0.05, **P<0.001.

corresponding decrease in LDL integrated size, plasma Chol levels in the small LDL subfraction (LDL-Chol_{25.5 nm}) were reduced significantly (P<0.01). The magnitude of the reduction in LDL-Chol_{25.5 nm} levels of 0.45 mmol/l seen with the experimental diet is almost equivalent to the difference of 0.6 mmol/l in LDL-Chol_{25.5 nm} levels between men who remained free from heart disease (1.5 (SD 0.9) mmol/l) compared with those who developed IHD (2.1 (SD 0.8) mmol/l) over the 5-year follow-up in the Québec Cardiovascular Study (St Pierre et al. 2001). This mean reduction in LDL-Chol_{25.5 nm} levels attributable solely to the combination diet would translate into a 30% reduction in the 5-year risk of IHD based on the Québec Cardiovascular Study results. These results from more than 2000 men followed for 5 years demonstrated that LDL-Chol_{25.5 nm} levels were a much more potent risk factor for IHD than the more traditionally used LDL-PPD (St Pierre et al. 2001).
Dreon, 1995; Dreon et al. 1997, 1998). To our knowledge, three studies have investigated the effect of soyabean protein on LDL particle size. The first two of these studies were conducted in free-living normolipidaemic premenopausal and in normolipidaemic or mildly hypercholesterolaemic postmenopausal women. These subjects consumed their usual diet, which was supplemented with one of the three isolated-soyabean-protein beverage powders, accounting for slightly less than one-half of the daily protein intake (Merz-Demlow et al. 2000; Wangen et al. 2001). Neither study reported a significant change in LDL-PPD as a result of incorporating isolated-soyabean-protein beverage powders into the diet. On the other hand, we have recently shown in a study conducted under strictly controlled conditions that the daily consumption of >50 g soyabean protein, with or without isolavones, increased LDL-PPD and induced a favourable redistribution of Chol from small to large LDL subclasses when compared with an animal-protein based diet (Desroches et al. 2004).

The impact of almonds or other nuts on LDL size is unclear. A study conducted in free-living patients showed that incorporating walnuts in the subjects’ habitual diets (approximately 24 g/4.2 MJ) induced a significant reduction in the Chol content of small LDL subfractions, while LDL-PPD and LDL-Chol remained unchanged compared with the subjects’ habitual diets (Almario et al. 2001). It must be stressed that incorporation of walnuts in the diet also led to a non-significant but important increase in the mean energy intake from fat (31.4 to 37.2 %), as well as with a significant change in the composition of dietary fat. However, substituting saturated fat with PUFA or MUFA does not seem to alter LDL size (Dreon et al. 1990; Kratz et al. 2002; Rivellese et al. 2003).

To the best of our knowledge, only two studies have investigated the impact of dietary phytosterols on LDL particle size phenotype. In the first study, it was shown that the daily consumption of 2.7 g phytosterol-supplemented ground beef had no significant impact on LDL-PPD; no other measures of LDL particle size phenotype were investigated (Matvienko et al. 2002). In a more recent study, we have shown that in moderately hypercholesterolaemic subjects, consumption of unesterified stanols, sterols, or both as butter, did not induce significant changes in LDL-PPD (Charest et al. 2004). The phytosterol-induced reduction in LDL-Chol levels were attributable only to a reduction in the Chol content of large LDL particles (>260 nm), with no change in the Chol levels of small particles (<25–5 nm; Charest et al. 2004).

Finally, one study has compared the impact of incorporating two servings (14 g dietary fibre/d) of oat cereal or wheat cereal on LDL size (Davy et al. 2002). The results indicated that LDL particle size was comparable after consumption of the two cereals, but that Chol within small LDL particles was reduced with oat cereal, whereas it was increased with the wheat cereal.

Taken together, results from the studies described earlier suggest that the individual nutritional components of our present combination diet have either no effect or beneficial effects on LDL particle phenotype. We hypothesise that the soyabean protein, almonds and viscous fibre may be responsible for the reduction in the Chol content of the small LDL particles, whereas the phytosterol component of the combination diet may have been responsible for the reduction in the Chol content within large LDL particles. It cannot be firmly excluded that part of the diet-induced changes in LDL size phenotype may be attributed to other differences between the baseline and the experimental diet, such as levels of dietary Chol and of saturated fatty acids and PUFA.

We conclude that a dietary combination of foods that have been shown to induce marked reduction in plasma LDL-Chol levels may also favourably alter important aspects of the LDL particle size phenotype, including LDL-Chol;<25.5 nm levels, particularly in subjects with a suboptimal phenotype at baseline. These results need to be replicated in larger samples of subjects and for a longer time period in order to address effectiveness of the diet over the longer-term, when availability of foods and compliance are major issues. Nevertheless, the magnitude of the reduction in plasma LDL-Chol levels, which essentially matches that achieved using pharmacological therapy (Jenkins et al. 2003), combined with the favourable changes in small dense LDL-Chol levels (LDL-Chol;<25.5 nm), identify this diet as a potentially potent dietary strategy to reduce the risk of CVD in high-risk individuals.

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