Cheese intake in large amounts lowers LDL-cholesterol concentrations compared with butter intake of equal fat content\textsuperscript{1–3}

Julie Hjerpsted, Eva Leedo, and Tine Tholstrup

ABSTRACT

Background: Despite its high content of saturated fatty acids, cheese does not seem to increase plasma total and LDL-cholesterol concentrations when compared with an equivalent intake of fat from butter. This effect may be due to the high calcium content of cheese, which results in a higher excretion of fecal fat.

Objectives: The objective was to compare the effects of diets of equal fat content rich in either hard cheese or butter or a habitual diet on blood pressure and fasting serum blood lipids, C-reactive protein, glucose, and insulin. We also examined whether fecal fat excretion differs with the consumption of cheese or butter.

Design: The study was a randomized dietary intervention consisting of two 6-wk crossover periods and a 14-d run-in period during which the subjects consumed their habitual diet. The study included 49 men and women who replaced part of their habitual dietary fat intake with 13% of energy from cheese or butter.

Results: After 6 wk, the cheese intervention resulted in lower serum total, LDL-, and HDL-cholesterol concentrations and higher glucose concentrations than did the butter intervention. Cheese intake did not increase serum total or LDL-cholesterol concentrations compared with the run-in period, during which total fat and saturated fat intakes were lower. Fecal fat excretion did not differ between the cheese and butter periods.

Conclusion: Cheese lowers LDL cholesterol when compared with butter intake of equal fat content and does not increase LDL cholesterol compared with a habitual diet. This trial is registered at clinicaltrials.gov as NCT01140165.

INTRODUCTION

Dairy products are rich in myristic and palmitic acids known to increase serum cholesterol (1, 2). Reports on the effect of fermented dairy products on serum cholesterol remain ambiguous (3–5). Cheese is a high-fat fermented dairy product and would be expected to increase serum cholesterol concentrations, thereby increasing the risk of CVD\textsuperscript{4}. However, a recent prospective cohort study, including 120,852 men and women followed for 10 y, found no association between cheese intake and risk of ischemic heart disease (6). Simple correlation studies support these findings of no, or a negative, relation between cheese intake and death from CVD (7–10). Two case-control studies found an inverse trend regarding intake of cheese and the risk of myocardial infarction (11, 12), whereas a single case-control study found consumption of cheese to be associated with an increased risk of myocardial infarction (13). More recently cross-sectional analyses from the Oslo Health Study showed a positive association between cheese consumption and HDL cholesterol (14).

Three small human intervention studies reported that cheese did not increase plasma cholesterol when compared with butter of equal fat content (15–17). In the study by Tholstrup et al (17), the butter was adjusted to contain the same lactose and casein contents as cheese, in the study by Biog et al (15) it was only adjusted to contain the same content of casein, and in the study by Nestel et al (16) no adjustment was made. The observation of a neutral effect of cheese compared with butter with regard to plasma cholesterol is not yet well established and needs to be confirmed in a larger study over longer time. A possible explanation for the neutral effect of cheese on cholesterol concentrations could be due to the high content of calcium in cheese, because a meta-analysis of randomized controlled trials showed a higher excretion of fecal fat when calcium intake was increased, especially calcium from dairy products (18).

The purpose of this study was to further investigate the effect of cheese and butter intakes, with equal fat contents, and habitual diet on risk markers of CVD—specifically fasting serum lipids (total, LDL, and HDL cholesterol and triacylglycerol), the inflammatory marker CRP, glucose, insulin, and blood pressure—in a larger intervention. The excretion of fecal fat was also measured.

SUBJECTS AND METHODS

Subjects

Subjects ranged in age from 22 to 69 y and were recruited for the study through announcements in local newspapers. Approximately 200 men and women were invited to information meetings, 70 were selected for screening, and 53 were enrolled as study participants. Exclusion criteria were current or previous CVD, diabetes mellitus or other severe chronic disease, BMI

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\textsuperscript{4} Abbreviations used: CRP, C-reactive protein; CVD, cardiovascular disease; hsCRP, high-sensitivity C-reactive protein.

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(kg/m^2) >32, use of lipid-lowering agents, and known or suspected abuse of alcohol, drugs, or medication. Subjects were asked to refrain from dietary supplements that might interfere with study measurements, blood donation, and dairy products other than those supplied by the department. They were instructed to maintain the same level of physical activity throughout the intervention. Five subjects using antihypertensive medication maintained the same regimen throughout the study. All subjects completed a lifestyle questionnaire and gave their informed consent in writing after receiving oral and written information about the study. Twenty-three of the 53 subjects agreed to hand in two 24-h fecal collections at the end of both the cheese and butter periods.

### Study design and diets

The study was a randomized crossover intervention with a 2-wk run-in period of habitual diet. The 2 intervention periods lasted 6 wk each and were separated by a washout period of ≥2 wk on habitual diet. The participants were provided with the test diets, which substituted part of their daily diet and replaced ~13% of energy of their daily fat intake. Energy needs were calculated according to the Commission of the European communities (19) and WHO/FAO/UNU (20) prediction equations for basal energy expenditure based on age, sex, and weight. Basal energy expenditure was multiplied by physical activity level. The participants were grouped according to their energy level: low (≤9.8 MJ), medium (9.8–12.5 MJ), and high (≥12.5 MJ), which was then used to set the fat content in the test diets to substitute the 13% of energy. The amount of experimental foods to be consumed on the medium energy level was 143 g cheese and 47 g butter. The experimental foods were hard cheese “Samsø” (27 g fat/100 g) and salted butter, both produced from cow milk. The subjects were not allowed to heat the cheese or butter before consumption. The cheese was consumed 3–4 mo after the production day. The production strains used in the fermentation of Samsø cheese were as follows: *Lactococcus lactis* subsp. *cremoris*, *Lactococcus lactis* subsp. *lactis*, *Lactococcus lactis* subsp. *lactis biovar deacetylactis*, and *Leuconostoc*. The fatty acid composition and the calcium content of the cheese and butter were analyzed by Qlip (Leusden, Netherlands). The fatty acid composition and calcium content are listed in Table 1.

The subjects were instructed to not consume other dairy products apart from a maximum of 6 cl. low fat milk (0.5% fat for coffee, tea, etc) supplied by the department. They had to consume the same amount of milk throughout the entire intervention. They could choose to consume none, 1, 2, 3, 4, or 5, or 6 cl/d of the remaining diet was self-selected. All dairy products were provided by Arla Food, Denmark (the cheese from Taulov dairy, the butter from Holstebro dairy, and the milk from Esbjerg dairy).

Subjects completed a 3-d dietary record to provide information about dietary intake during the intervention and ensure stable weight. This was done during the run-in period and during the last 2 wk of each intervention period. Two weekdays and one weekend day have been included in the dietary record to take any differences in nutrient intake during weekdays and weekend days into account. The participants were given feedback and advice after delivering the first dietary record on how they could decrease fat intake during the intervention period. This was achieved by guidance from our dietitian. Dietary intake was estimated by using Dankost 3000 dietary assessment software (Dankost).

Subjects attended the department to collect the test food and for follow-up and weighing each or each second week. After 3 wk of intervention, the participants were interviewed about their well-being, physical activity level, and diet to make sure they adhered to the test diet and maintained the same weight and physical activity level. In addition, participants were supplied with recipes as inspiration for how the cheese and butter could be consumed.

The study was carried out according to the Helsinki Declaration and approved by the Danish National Committee on Biomedical Research Ethics (report no. H-B-2009–052).

### Blood samples

Blood samples were taken in duplicate on consecutive days, after the run-in period and after 3 and 6 wk of the intervention, from fasting subjects after 10 min of supine rest. The values from each set of the samples were averaged. In addition to fasting for 12 h, the participants were asked to refrain from smoking for 12 h, to refrain from performing any extreme sports for 36 h, and to not drink alcohol or take medicine for 24 h before the blood sampling.

Blood samples were drawn after 3 wk of intervention for the measurement of serum blood lipids (triacylglycerol and total, LDL, and HDL cholesterol) and after the run-in period and after 6 wk of intervention for the measurement of blood lipids, hsCRP, plasma glucose, and serum insulin. The blood samples for measurement of blood lipids, hsCRP, and insulin were collected into dry tubes, whereas the blood samples for the measurement of glucose were collected into tubes with a 1 x 3 mL fluoridecitrate mixture. Blood samples were kept at room temperature for 30 min to coagulate. Subsequently, all blood samples were centrifuged at 2200 x g for 20 min at 20°C and stored at −80°C until analyzed.

LDL and HDL cholesterol were assessed with an enzymatic colorimetric procedure (ABX Pentra LDL Direct CP and ABX Pentra HDL Direct CP). Cholesterol and triacylglycerol concentrations were assessed in serum by enzymatic procedures (CHOD-PAP and GPO-PAP, respectively). CHOD-PAP is an enzymatic photometric test, whereas GPO-PAP is an enzymatic colorimetric method. Blood lipids were analyzed with an ABX Pentra 400 Chemistry Analyzer (Horiba ABX). The interassay

### Table 1

Fatty acid composition and calcium content of the cheese and butter<sup>1</sup>

<table>
<thead>
<tr>
<th>Fatty acid</th>
<th>Cheese</th>
<th>Butter</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sum 4:0–12:0</td>
<td>14.7</td>
<td>14.3</td>
</tr>
<tr>
<td>14:0</td>
<td>10.5</td>
<td>10.7</td>
</tr>
<tr>
<td>16:0</td>
<td>27.1</td>
<td>29.2</td>
</tr>
<tr>
<td>18:0</td>
<td>11.6</td>
<td>10.7</td>
</tr>
<tr>
<td>18:1n–9&lt;sup&gt;2&lt;/sup&gt;</td>
<td>20.9</td>
<td>20.3</td>
</tr>
<tr>
<td>18:2n–6</td>
<td>1.8</td>
<td>1.7</td>
</tr>
<tr>
<td>18:3n–3</td>
<td>0.4</td>
<td>0.4</td>
</tr>
<tr>
<td>Others</td>
<td>10.9</td>
<td>10.5</td>
</tr>
<tr>
<td>Calcium (mg/100 g)</td>
<td>834</td>
<td>19</td>
</tr>
</tbody>
</table>

<sup>1</sup> Analyzed by Qlip, Leusden, Netherlands.
<sup>2</sup> Including 18:1trans12.
CVs for total, LDL, and HDL cholesterol and triacylglycerol were 1.9%, 3.3%, 2.8%, and 4.2%, respectively. The intraassay CVs for total, LDL, and HDL cholesterol and triacylglycerol were 0.9%, 0.9%, 1.2%, and 2.6%, respectively.

Serum CRP concentrations were measured by using a highsensitive immunometric assay (ABX Pentra CRP CP). The analysis was carried out with an ABX Pentra 400 Chemistry Analyzer (Horiba ABX). The detection limit was 0.1 mg/L. The interassay and intraassay CVs were 3.4% and 3.6%, respectively.

Glucose concentrations were measured with an enzymatic procedure (ABX Pentra Glucose HK CP) and were analyzed with an ABX Pentra 400 Chemistry Analyzer (Horiba ABX). The interassay and intraassay CVs for plasma glucose were 1.5% and 1.1%, respectively. Insulin was measured by chemiluminescent immunoassay with an Immulite 1000 (Siemens Medical Solution Diagnostics). The detection limit was 14.4 pmol/L. The inter- and intraassay CVs for serum insulin were 5.1% and 2.7%, respectively.

Insulin resistance was calculated by using HOMA with the following formula:

\[
\text{HOMA} = \frac{\text{Fasting serum insulin (μU/mL)} \times \text{fasting plasma glucose (mmol/L)}}{22.5}
\]

HOMA values have been shown to correlate well with values obtained by using the gold standard clamp technique (23, 24).

Blood pressure

Blood pressure was measured on 2 consecutive days after the run-in period and after 6 wk of each intervention by using an automatic blood pressure monitor (model UA-787Pplus; A&D Medical). One measurement made after 10 min of supine rest was not used. Subsequently, 2 new measurements were made and averaged.

Fecal samples

All feces excreted were collected into preweighed containers from 23 subjects during the last 2 d of each intervention period. The samples from the same intervention period were pooled and blended with Milli-Q water (1:1) (Millipore Corp) and frozen at −20°C. Before analysis, the samples were freeze-dried and homogenized. The samples were acid hydrolyzed with 3 mol HCl/L at 90°C for 1 h. Total fat content was measured by a method from Soxhlet (ANKOM Technology) with modifications (25).

Statistical analysis

The washout period resulted in total cholesterol values that were not significantly different from run-in values (5.3 compared with 5.2 mmol/L). The incorporation of the washout period allowed subjects to get a break between the 2 periods of cheese and butter intake with no other dairy products allowed.

The outcome variables (total, LDL, and HDL cholesterol; total:HDL cholesterol; triacylglycerol; CRP; insulin; glucose; HOMA values; systolic and diastolic blood pressure; and fecal fat content) were analyzed by using a linear mixed model incorporating systematic effects of period and treatment and their interaction, a carryover effect, and the explanatory variables age, sex, period, weight, and smoking, which were included, if significant, to adjust for possible differences between subjects. Model reduction was performed, and nonsignificant effects were removed one by one from the model.

Subject-specific random effects were included to account for intersubject variability and to adjust for any nonspecific differences that could not be explained by the explanatory variables included. In the model for the outcome variables measured at both 3 and 6 wk (the blood lipids), individual series of measurements for a given treatment were included in the random statement to allow a higher correlation of outcome variables in the same treatment period than in different periods. In addition, the effect of weeks was included in the model to investigate the effect over time (3 and 6 wk). No time-by-treatment interactions existed; therefore, the data for 3 and 6 wk were pooled.

The outcome variables were Bonferroni-corrected to adjust for multiple comparisons. Because of a detection limit of 0.1 mg/L for CRP and of 14.4 pmol/L for insulin, the results below these concentrations were not included in the statistical analyses. Model validation was based on graphic assessment using normal probability plots and residual plots. Model-based least-squares means were used to quantify the treatment effects. In cases in which deviations from the model assumptions were detected, outcome variables were transformed by using appropriate transformations. All P values were evaluated at a 5% significant level. The analyses were made by using PROC MIXED in Statistical Analysis System version 9.1 (SAS Institute).

RESULTS

Subjects

Fifty-three subjects entered the study. Four subjects dropped out for various reasons. This left 49 subjects to complete the intervention. One subject completed only the first 3 wk in each period. The baseline characteristics of the subjects who completed the study (n = 49) are listed in Table 2. No differences in weight were observed between the cheese and butter periods.

Blood lipids

The blood lipids were measured after the run-in period and after 3 and 6 wk of intervention. The changes in serum total, LDL-, and HDL-cholesterol concentrations after the run-in, cheese, and butter periods are shown in Figure 1. The cheese period resulted in a 5.7% lower total cholesterol concentration (P < 0.0001) and a 6.9% lower LDL-cholesterol concentration.

Table 2

<table>
<thead>
<tr>
<th>TABLE 2</th>
<th>Baseline characteristics of the 49 subjects who completed the intervention</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Value</td>
</tr>
<tr>
<td>Sex [n (%)]</td>
<td></td>
</tr>
<tr>
<td>Men</td>
<td>28 (57)</td>
</tr>
<tr>
<td>Women</td>
<td>21 (43)</td>
</tr>
<tr>
<td>Postmenopausal women [n (%)]</td>
<td></td>
</tr>
<tr>
<td>Age (y)</td>
<td>55.5 ± 12.41</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>172.4 ± 9.1</td>
</tr>
<tr>
<td>BMI (kg/m2)</td>
<td>25.3 ± 3.3</td>
</tr>
<tr>
<td>Waist circumference (cm)</td>
<td>90.9 ± 11.2</td>
</tr>
<tr>
<td>Smoking [n (%)]</td>
<td>10 (20)</td>
</tr>
</tbody>
</table>

1 Mean ± SD (all such values).
(P < 0.0001) compared with the butter period. In addition, the cheese period resulted in a decrease in HDL cholesterol compared with the butter period (P < 0.005).

The cheese period did not result in higher total and LDL-cholesterol concentrations or lower HDL-cholesterol concentrations when compared with run-in. The butter period resulted in higher total and LDL-cholesterol concentrations than did the run-in period (P < 0.0005 and P < 0.05, respectively).

As listed in Table 3, the ratio of total to HDL cholesterol did not differ significantly between the run-in and cheese periods or between the cheese and butter periods. Correspondingly, plasma triacylglycerol concentrations did not differ between the cheese and butter periods; however, triacylglycerol concentrations were greater in the butter period than in the run-in period (P < 0.05).

**TABLE 3**
Results after the run-in, cheese, and butter periods

<table>
<thead>
<tr>
<th></th>
<th>Run-in</th>
<th>Cheese</th>
<th>Butter</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total:HDL cholesterol</td>
<td>3.57 ± 0.03</td>
<td>3.59 ± 0.03</td>
<td>3.68 ± 0.03</td>
</tr>
<tr>
<td>Triacylglycerol (mmol/L)</td>
<td>1.06 ± 0.06</td>
<td>1.15 ± 0.06</td>
<td>1.17 ± 0.06</td>
</tr>
<tr>
<td>hsCRP (mg/L)²</td>
<td>1.15 ± 1.21</td>
<td>0.93 ± 1.21</td>
<td>0.92 ± 1.21</td>
</tr>
<tr>
<td>Glucose (mmol/L)</td>
<td>5.59 ± 0.06</td>
<td>5.63 ± 0.06</td>
<td>5.53 ± 0.06</td>
</tr>
<tr>
<td>Insulin (pmol/L)²</td>
<td>46.5 ± 3.04</td>
<td>46.7 ± 3.05</td>
<td>45.2 ± 3.05</td>
</tr>
<tr>
<td>HOMA²</td>
<td>1.69 ± 0.12</td>
<td>1.71 ± 0.12</td>
<td>1.62 ± 0.12</td>
</tr>
<tr>
<td>Systolic BP (mm Hg)</td>
<td>127 ± 1.02</td>
<td>125 ± 1.02</td>
<td>127 ± 1.02</td>
</tr>
<tr>
<td>Diastolic BP (mm Hg)</td>
<td>79.4 ± 1.04</td>
<td>78.7 ± 1.05</td>
<td>79.8 ± 1.05</td>
</tr>
<tr>
<td>Fat content in feces (%)³</td>
<td>—</td>
<td>22.6 ± 1.45</td>
<td>19.9 ± 1.45</td>
</tr>
</tbody>
</table>

¹ All values are least-squares means ± SEMs. BP, blood pressure; HOMA, homeostasis model assessment; hsCRP, high-sensitivity C-reactive protein. ²Significantly different from run-in, P < 0.05 (linear mixed model with Bonferroni correction). ³Significantly different from butter period, P < 0.05 (linear mixed model with Bonferroni correction).

CRP, insulin, glucose, blood pressure, and fat in feces

CRP, glucose, insulin, and blood pressure were measured after the run-in period and after 6 wk of intervention (Table 3). The cheese period resulted in higher glucose concentrations than did the butter period (P < 0.05). No differences in hsCRP concentrations, insulin concentrations, and blood pressure were observed between the run-in, cheese, and butter periods. Feces were collected during the last 2 d of each intervention period from 23 of the subjects. Fecal fat excretion did not differ between the cheese and butter periods (P = 0.1035).

**Dietary records**

Results from the dietary records are listed in Table 4. No differences were observed in energy intake. The amount of SFAs, PUFAs, and MUFAs did not differ between the butter and cheese period, but the amount of fat intake appeared significantly higher when measured as a percentage of energy (P < 0.05) during the butter period than during the cheese period. However, this difference was not significant when fat intake was calculated in grams (P = 0.0517). Fat intake in grams and SFA intake were both lower during the run-in period than during the cheese and butter periods (P < 0.05). The intake of protein was lower and of carbohydrates was greater in the butter period than in the cheese period (P < 0.05). As expected, the calcium intake differed across the interventions (P < 0.05).

**DISCUSSION**

This study was designed to compare the effect of cheese intake with butter intake with the same fat content, and with habitual diet (run-in). Cheese intake resulted in LDL cholesterol concentrations that were 6.9% lower compared with intake of butter, with the same fat content. This is in accordance with 3 other intervention studies where subjects consumed cheese and butter in a crossover design (15–17). Results from these smaller studies suggested a significant or borderline significant lowering
different from run-in period, $P < 0.05$ (linear mixed model). *Significantly different from butter period, $P < 0.05$ (linear mixed model). $^\dagger$Significantly different from run-in period, $P < 0.05$ (linear mixed model).

Of particular interest was that cheese intake did not increase LDL-cholesterol concentrations compared with the run-in period, despite data from the dietary records indicating a significantly higher total fat (15.2 g) and 11.1 g higher saturated fat intake during the cheese period than during the run-in period. These findings are supported by others who have found that a high daily intake of cheese does not increase LDL-cholesterol concentrations significantly compared with a lower saturated fat intake during a run-in period (16).

Cheese intake resulted in lower HDL-cholesterol concentrations than did butter intake. These findings are in contrast with the findings of cross-sectional analyses from the Oslo Health Study, which showed a positive association between cheese consumption and HDL cholesterol (14). Strong evidence indicates that HDL cholesterol is protective against CVD (24, 25). However, the total: HDL cholesterol ratio has been suggested to be an even better predictor of the development of CVD (26), but no difference in this ratio was observed between treatments.

In the study by Tholstrup et al (17) and the study by Biong et al (15), ~20% of energy came from dairy fat from the test foods; ~13% of energy came from dairy fat in the current study. This finding indicates that results can be achieved with the use of smaller amounts of the test foods, and would thus be more comparable with normal intakes. These findings are supported by the study by Nestel et al (16), in which the intake of fat was similar to the medium energy level in our study. Although our results support previous findings indicating that cheese does not increase total cholesterol and LDL-cholesterol concentrations, the reasons for this neutral effect of cheese on blood lipids remains unknown. One possible explanation may be the effect of the high content of calcium in cheese, because both animal and human studies have shown an increased excretion of fat in feces with intake of calcium (27–30). Dairy calcium seems to have a greater effect in this respect (18). The effect of calcium may be exerted in the intestine, where it forms insoluble calcium soaps with free fatty acids or by being able to bind to bile acids (27).

We found that the cheese period resulted in a mean 11.6% higher excretion of fecal fat compared with the butter period; however, the difference was not statistically significant. The dietary records and results of the analyses of the calcium content of the cheese and butter suggested a significantly higher calcium intake during the cheese period. Therefore, the high calcium content of cheese, per se, did not appear to explain the neutral effect of cheese on serum cholesterol in our study; thus, it is important to consider other possible mechanisms.

Cheese has a high content of protein. The dietary records suggested different protein intakes during the run-in, cheese, and butter periods; the largest intake occurred during the cheese period. Therefore, it seems likely that the high protein content of cheese could be a possible explanation for the neutral effect of cheese on cholesterol. However, Tholstrup et al (17) adjusted for the casein content in their study and found significantly lower LDL-cholesterol concentrations with cheese intake than with butter intake. Therefore, the higher protein intake was unlikely to be a reason for the neutral effect of cheese compared with butter. However, it could be speculated that some matrix effect might exist because the fat globules in cheese are trapped within the casein matrix formed from aggregated micelles (31).

Fermentation is in itself a strong candidate for an explanation of the effect. In our study the Samso cheese consumed was produced mostly by using various L. lactis species. Although no consistent hypocholesterolemic effect of Lactococcus bacteria has yet been established, it is probable that the fermentation of cheese accounts, at least in part, for the reduction in LDL cholesterol.

St-Onge et al (32) reviewed the possible mechanisms of fermented dairy products on cholesterol metabolism. It was suggested that bacteria in the large intestine produce short-chain fatty acids from unabsorbed carbohydrates, which may alter cholesterol synthesis. In addition, bacteria in the intestine may bind to bile acids, which will increase its excretion and thereby result in a decreased bile acid recycling in the enterohepatic circulation.

The lower glucose concentration after butter consumption than after cheese consumption was unexpected, and we cannot explain these findings. Insulin resistance values were subsequently calculated by using HOMA, but no differences were observed.

A minor limitation of this study was that the 3-d dietary records indicated that the percentage of energy as fat in the diet during the cheese period was lower than that during the butter period. This, however, was not the case when fat intake was estimated in grams. The high protein content of cheese has to be compensated for by something else in the distribution of macronutrients (as a percentage of energy). Calculations of data from the dietary records by using Mensink and Katan’s (33) predictive equation for changes in serum cholesterol, when changing dietary fatty acid composition, showed that the intake during the cheese period should have the same or even a small cholesterol-increasing effect compared with the butter period.

In the current larger intervention study of longer duration, we confirm previous findings that cheese does not increase cholesterol concentrations compared with butter. Our results indicate that even a high intake of a full-fat cheese may not affect CVD risk markers compared with a habitual diet (run-in) with a lower content of total and saturated fat. Thus, dietary advice regarding the intake of full-fat hard cheese by persons with hypercholesterolemia may need to be revised. Because the fecal fat content did not differ significantly between the butter and cheese diet...
periods, we suggest that the slightly lower fat absorption with cheese intake played only a minor role. Therefore, the underlying mechanisms behind this neutral effect of cheese intake on cholesterol concentrations still need to be elucidated.

We thank our biomedical laboratory technicians Anette Hansen and Hanne Lysdal Petersen for technical assistance, our dietitian Hanne Jensen, and the staff of our kitchen for their excellent work. We also thank Tina Cuthbertson for efficient language editing of the final manuscript, Ylva Ardø for sharing her knowledge, and Lars Ove Dragsted for valuable advice during the planning of the study.

The authors’ responsibilities were as follows—JH: participated in the research design, conducted the research, performed the statistical analysis, and wrote the first manuscript; EL: participated in conducting the research; TT designed the research and collaborated on writing the manuscript. All authors approved the final version of the manuscript. JH is a PhD student at the Department of Human Nutrition, Faculty of Life Science, University of Copenhagen; EL was a master student at the same department during the study period and TT is an Associate Professor. None of the authors had a conflict of interest to declare. The sponsors had no influence on the manuscript or its conclusions.

REFERENCES