

## ORIGINAL COMMUNICATION

# Dairy fat in cheese raises LDL cholesterol less than that in butter in mildly hypercholesterolaemic subjects

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**Objective:** To determine whether dairy fat in cheese raises low-density lipoprotein (LDL) cholesterol as much as in butter, since epidemiology suggests a different impact on cardiovascular disease.

**Design:** A randomised crossover trial testing the daily consumption of 40 g dairy fat as butter or as matured cheddar cheese, each of 4 weeks duration, was preceded by and separated by 2-week periods when dietary fat was less saturated.

**Setting:** Free-living volunteers.

**Subjects:** A total of 14 men and five women of mean age  $56 \pm 8$  y, with mean total cholesterol of  $5.6 \pm 0.8$  mmol/l.

**Main outcome measures:** Plasma cholesterol, LDL cholesterol (LDL-C), HDL cholesterol (HDL-C), triacylglycerol and glucose.

**Results:** Saturated fat intake was significantly lower during the run-in than during the cheese and butter periods. Mean lipid values did not differ significantly between the cheese and run-in periods, but total cholesterol and LDL-C were significantly higher with butter: total cholesterol (mmol/l): butter  $6.1 \pm 0.7$ ; run-in  $5.6 \pm 0.8$  ( $P < 0.05$ ; ANOVA with Bonferroni adjustment); vs cheese  $5.8 \pm 0.6$  ( $P > 0.05$ ); median LDL-C (mmol/l): butter  $3.9$  ( $3.5$ – $4.1$ ) vs run-in  $3.4$  ( $3.0$ – $4.1$ ) ( $P < 0.05$ ; Tukey test); vs cheese  $3.7$  ( $3.3$ – $3.9$ ) ( $P > 0.05$ ). Among 13 subjects whose initial LDL-C was  $> 4$  mmol/l, the difference between butter ( $4.4 \pm 0.3$  mmol/l) and cheese ( $3.9 \pm 0.3$  mmol/l) was significant ( $P = 0.014$ ). HDL-C was highest with butter and triacylglycerol with cheese (neither was significant).

**Conclusion:** A total of 40 g dairy fat eaten daily for 4 weeks as butter, but not as cheese, raised total and LDL cholesterol significantly compared with a diet containing significantly less saturated fat. Dietary advice regarding cheese consumption may require modification.

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### Introduction

Butter fat is generally recognised to consistently raise plasma cholesterol concentrations, especially in hypercholesterolaemic subjects (Chisholm *et al*, 1996; Noakes *et al*, 1996; Zock & Katan, 1997; Matthan *et al*, 2004). This appears to be a function of its fatty acid composition since the substitution of palmitic and stearic acids by oleic and linoleic acids in

butter from cows fed protected oils led to lower cholesterol values than butter derived from conventionally fed cows (Noakes *et al*, 1996). Some evidence suggests that the increase in middle-aged men is predominantly that of low-density lipoprotein (LDL-C), whereas in women high-density lipoprotein (HDL-C) increases as well as LDL-C (Clifton & Nestel, 1992). The epidemiological association between national consumption of butter fat and cardiovascular mortality has been advanced as a key reason for reducing intake of full-cream dairy fat (Artaud-Wild *et al*, 1993). Earlier Turpeinen (1979) had also demonstrated a strong correlation ( $r = 0.75$ ) between dairy fat consumption in 22 countries and mortality from coronary heart disease (CHD). However, the possibility that not all full-cream fat dairy products influence plasma cholesterol identically has been rarely investigated. The French and possibly the Swiss, although consuming

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large amounts of cheese, appear to have experienced less CHD mortality relative to butter fat intakes (Artaud-Wild *et al*, 1993). In fact, in an analysis of macronutrient consumption in 40 countries of varying socio-economic affluence in which the intake of dairy fats correlated strongly with the national mortality data from CHD, Artaud-Wild *et al* (1993) observed that cheese was the exception, at least in those with the highest saturated fat-cholesterol 'score', showing a significant negative correlation of  $-0.69$ . Renaud and de Lorgeril (1989) reported a significant correlation between consumption of dairy fat and CHD mortality when cheese was excluded.

Cheese is a fermented dairy product, and yoghurt rich in probiotic microorganisms has been claimed at least not to increase the plasma cholesterol concentration (McNamara *et al*, 1989). Data with whole milk appear to be less consistent than those with butter, suggesting additional components in milk that may modify the cholesterol-raising effect of the fat (Elwood *et al*, 2004).

Very recently, Tholstrup *et al* (2004) reported a controlled, crossover design comparison of milk, cheese and butter, providing almost identical amounts of dietary fat on plasma lipids. In 13 normocholesterolaemic young men, milk and butter, but not cheese, raised LDL-C significantly compared with the baseline diet. The LDL-C concentration was significantly lower with cheese than with butter. We had been undertaking a similar comparison of cheese vs butter in middle-aged men and women whose average plasma cholesterol concentration was moderately elevated at 5.6 mmol/l. The amount of fat required to be consumed daily in our trial was less than in the experiment by Tholstrup *et al* (2004). The results are reported in this paper.

## Methods

### Subjects

A total of 20 middle-aged healthy men and women were recruited on the basis of an LDL-C of above 3.6 mmol/l but <6 mmol/l at the initial screen. A total of 19, five women and 14 men completed the study and their characteristics are shown in Table 1. Mean age was  $56.3 \pm 7.8$  y and mean body weight was  $85.88 \pm 18.64$  kg. Although the subjects were overweight, the mean plasma glucose concentration was normal at  $5.1 \pm 0.6$  mmol/l. Subjects were normotensive,

**Table 1** Characteristics of the 20 subjects in the study

Variable	Mean (s.d.)
Men: Women	14:6
Age (y)	56.3 (7.8)
Weight (kg)	85.9 (18.6)
BMI (kg/m <sup>2</sup> )	27.7 (4.6)
Systolic blood pressure (mmHg)	124 (16.2)
Diastolic blood pressure (mmHg)	76 (11)
Plasma glucose (mmol/l)	5.1 (0.6)

nonsmokers and their habitual daily alcohol intake was <4 standard drinks for the men and <2 drinks for the women. None was treated with drugs that affect plasma lipid values, and liver and renal functions and full blood examination were normal in all. The five women were postmenopausal and not taking hormone replacement. Exclusion criteria were diabetes or other metabolic disturbances other than mild hypercholesterolaemia and unusual habitual patterns of eating or living. One woman who failed to complete the trial was unable to eat the required amount of cheese. Of the 19 subjects completing the trial (14 men and five women), 13 had an LDL-C concentration of at least 4 mmol/l (mean 4.2) at the end of the reduced saturated fat run-in. In the remaining six, the screening LDL-C had fallen below 4 mmol/l at the conclusion of the run-in period.

### Design

The two intervention dietary periods, with either butter or cheese, were of 4 weeks duration, preceded by a 2-week run-in during which a low-fat dairy, moderately increased carbohydrate background diet was instituted (that did not differ substantially from the subjects' habitual pattern of eating since the majority had known that their plasma cholesterol concentration was borderline). The initial test period, the run-in that was moderately reduced in fat, was followed by the two higher fat diets in a crossover design to which subjects were randomised (random numbers system). A similar 2-week wash-out took place between interventions. Similar amounts of fat were eaten during the cheese and butter periods. The remaining diet was self-selected but similar in energy intake throughout, with body weights at the end of run-in, intervention 1, wash-out and intervention 2 varying by an average of less than 0.5 kg (85.5, 85.9, 85.8 and 85.7 kg, respectively). The background diet was self-selected from a carefully constructed set of foods within specified food groups. Choices could be made within those groups of food.

Blood was taken on two occasions at the end of each period after the subjects had fasted at least 12 h overnight. Measurements included plasma cholesterol and triacylglycerol concentrations, LDL-C, HDL-C and glucose by standard enzyme-based methods (Cobas-Bio automated analyser, Roche, Basel, Switzerland). The values from each set of two samples were averaged. Blood pressures were measured at each visit by automated means; several measurements were made over 5 min and averaged.

The study was approved by the Human Ethics Committee of the Alfred Hospital, Melbourne, where the study was conducted.

### Dietary interventions and analysis

Initial and subsequent analyses of energy and macronutrient consumption at the end of each period were evaluated with mid-week 3-day food frequency questionnaires devised by

the Anti-Cancer Council of Victoria (Melbourne, Australia). This food frequency questionnaire has been validated for use in major prospective trials in Australia. It is characterised by care in design of questions, inclusion of many foods, setting an order of questions that allows ease of answering, obtaining information on choices within food groups and qualitative measures of food preparation and flexibility with serving sizes. Low-fat dairy products were the only dairy food allowed in the run-in and wash-out periods and the increased amount of saturated fat that was scheduled during the cheese and butter periods was partly substituted with carbohydrates from cereal products and vegetables. The latter was reduced during the interventions. Cheese was a 1-y matured cheddar type with a fat content of 33% wet weight (Murray Goulburn, Brunswick, Australia); 40 g daily fat consumption was achieved through 120 g cheese distributed over several meals during the day. A similar amount of butter fat was eaten from preweighed portions of butter and one butter-rich muffin, also distributed over three meals. Dietary counselling was provided at each visit. The macronutrient distributions during the three periods included in the statistical analysis are shown in Table 2.

### Statistical analysis

Since the hypothesis was that eating butter but not cheese would result in a statistically significant difference in LDL-C compared with the run-in period, those three periods were entered into the analysis. (The wash-out period, although resulting in a total cholesterol value that was not dissimilar from run-in value (5.8 vs 5.6 mmol/l), was not considered part of the hypothesis. Its incorporation allowed participants respite between two periods of high fat intake that was unusual given that their habitual diet was relatively low in

fat.) Normality was established initially for total cholesterol and HDL-C; for triacylglycerol and LDL-C, normality failed and the data were analysed as medians. Order of randomisation and period effect were initially shown not to be confounders. Analyses of variance with dietary periods as study factor and subjects as repeated measures were carried out for each outcome variable. For LDL-C and triacylglycerol, repeated measures ANOVA on ranks was used. Since these analyses showed significant heterogeneity among the three periods for total cholesterol and LDL-C, further comparisons were made between treatments and run-in values using all pairwise multiple comparison procedures (Bonferroni for total cholesterol and Tukey test for LDL-C). A two-tailed  $P < 0.05$  was regarded as significant. The numbers of subjects provided power to detect differences of 5% in the total cholesterol concentration and in LDL-C with a power of 80%. A secondary hypothesis related to the responses based on initial LDL-C concentrations. The number of subjects with LDL-C above 4 mmol/l had adequate power but was inadequate for those below 4 mmol/l; differences were nevertheless analysed, including both groups, by two-way ANOVA with interaction (groups  $\times$  time). Macronutrient intakes were analysed by one-way ANOVA (SAS Version 8.2; SAS Institute Inc., Cary, NC, USA). We thank Dr Michael Bailey (Monash University, Melbourne, Australia) for assistance.

### Results

The mean body weights at the end of each of the four periods and the macronutrient consumption analyses showed that energy intake did not differ significantly across the trial (Table 2). Consumption of total fat was greater during the cheese and butter periods than during the run-in period, although the difference was not statistically significant. However, saturated fat intake was significantly greater with cheese and butter than during the run-in period ( $P < 0.05$ ), whereas consumption of polyunsaturated and monounsaturated fatty acids did not differ across the three periods. Carbohydrate intake tended to be higher during the run-in period. Eating relatively large amounts of cheese and butter led to only one subject experiencing dislike and inability to continue.

Table 3 shows the plasma lipid data for the run-in and two interventions. The values during the wash-out are included for completeness. Total cholesterol and LDL-C concentrations with butter were 9 and 15% greater, respectively, than at the end of the run-in ( $P < 0.05$  for total cholesterol and  $< 0.05$  for LDL-C after Bonferroni and Tukey adjustments, respectively). By contrast, the corresponding lipid values at the end of the cheese and run-in periods did not differ significantly. Neither the total nor the LDL cholesterol concentrations differed significantly between the cheese and butter periods ( $P = 0.054$  for total cholesterol and 0.07 for LDL-C), although the values with butter tended to be

**Table 2** Average consumption of macronutrients during the run-in, butter and cheese periods

Nutrient	Run-in	Butter	Cheese
Energy (kJ)	7240 (1994)	7106 (2106)	7398 (2744)
Fat total (g)	63.3 (27.2)	73.8 (26.5)	74.5 (36.1)
(%)	31.5	37.4	36.2
Saturated fat <sup>a</sup>	25.2 (12.9)	33.9 (13.9)	33.0 (15.4)
(%)	12.5	17.2	16.7
Polyunsaturated fat <sup>b</sup>	9.3 (4.9)	8.2 (3.0)	9.7 (5.4)
(%)	4.6	5.2	4.7
Monounsaturated fat	28.5 (9.8)	29.2 (9.3)	29.9 (13.5)
(%)	14.2	14.8	14.6
Carbohydrate (g)	204 (54)	185 (51)	187 (70)
(%)	45.1	41.7	40.4
Protein (g)	82 (70–103)	80 (69–95)	86 (70–101)
(%)	18.1	19.4	18.6
Cholesterol (mg)	208 (172–292)	253 (209–342)	239 (196–274)

<sup>a</sup>ANOVA: differences between treatments  $P < 0.001$ ; Tukey test  $P < 0.05$  between the run-in and either butter or cheese.

<sup>b</sup>n-6 PUFA only.

Values are mean (s.d.) except for those of cholesterol, which are median (25–75%).

**Table 3** Plasma lipid concentrations (mmol/l) at the end of the run-in, butter and cheese ( $n=19$ )

Plasma lipid	Run-in	Butter	Cheese
Total cholesterol <sup>a</sup>	5.6 (0.8)	6.1 (0.7)	5.8 (0.6)
LDL-C <sup>b</sup>	3.4 (3.0–4.1)	3.9 (3.5–4.1)	3.7 (3.3–3.9)
HDL-C	1.5 (0.4)	1.6 (0.4)	1.5 (0.4)
Triacylglycerol	1.1 (0.7–1.4)	1.1 (0.9–1.4)	1.5 (0.8–1.6)

<sup>a</sup>RM ANOVA: difference between treatments  $P<0.002$ ; butter vs run-in  $P<0.05$  with Bonferroni adjustment.

<sup>b</sup>RM ANOVA on ranks: difference between treatments  $P=0.007$ ; butter vs run-in  $P<0.05$  with Tukey test.

Values are mean (s.d.) or median (25–75%).

higher than with cheese. The values for cheese were intermediate between run-in and butter, LDL-C being 5% lower with cheese than with butter. When the data for the 13 subjects whose LDL-C remained clearly elevated ( $>4$  mmol/l) after the reduced saturated fat run-in period were considered separately, the difference between the cheese period ( $3.9\pm 0.4$  mmol/l) and butter period ( $4.4\pm 0.4$  mmol/l) was significant ( $P=0.014$ ).

Plasma triacylglycerol concentrations were highest with cheese ( $P=0.052$  across the three treatments), median concentration at the end of the cheese period being approximately 50% higher than at the end of the butter period and the run-in. HDL-C was highest after butter but not significantly so.

Plasma glucose concentrations at the end of each period were on average not significantly dissimilar (5.09, 5.04 and 5.05 mmol/l after the run-in, cheese and butter, respectively). Plasma insulin concentrations were correspondingly not significantly dissimilar between the three dietary periods (data not shown). Average blood pressures (systolic, diastolic and median) and heart rates changed little over the entire period (data not shown).

## Discussion

The major finding of this study was that, compared with a moderately reduced fat diet (run-in), the LDL-C-raising effect of cheese was on average 6% less than that of butter at comparable intakes of total fat and saturated fat. Although not significant for the group as a whole, there was a strong trend for butter to raise total and LDL cholesterol more than cheese ( $P=0.054$  for the former and 0.07 for the latter). Energy consumption and body weights were not significantly different across the three dietary periods, but consumption of saturated fat was significantly greater with butter and cheese than during the run-in period (Table 2). However LDL-C was significantly higher than during the run-in only with butter intervention, by 15%. The LDL-C values with cheese consumption were intermediate between the reduced-fat run-in and butter values. (The difference between eating cheese and eating a Western diet might have

been even less.) Among the 13 subjects whose LDL-C concentrations were clearly raised ( $>4$  mmol/l), eating butter led to significantly higher LDL-C than when eating cheese (12%).

Since butter has been generally found to raise total and LDL cholesterol when compared with a diet containing less saturated fat (Chisholm *et al*, 1996; Noakes *et al*, 1996), it has been assumed that similar amounts of dairy fat in cheese would lead to a similar outcome. However, a recent publication by Tholstrup *et al* (2004), in which larger amounts of fat than in the present study were compared when butter and cheese were eaten, reported findings similar to ours. LDL-C concentrations were significantly higher after butter than after cheese in a study in which casein and lactose were approximately similar with the two diets. A third period during which milk was consumed resulted in LDL-C levels not different from that with butter.

Although HDL-C concentrations were highest with butter, the differences were not significant ( $P=0.22$  across treatments). That butter might raise HDL-C has been noted previously and in a recent study in which HDL-C was significantly higher with butter fat than with unsaturated fat, the metabolic reason appeared to lie in a faster rate of removal of HDL particles with the unsaturated fat (Matthan *et al*, 2004). The reason for the higher triacylglycerol concentration with cheese compared with the other two periods, although nonsignificant, is uncertain and likely to be a chance finding. The dietary data during the butter and cheese periods showed similar carbohydrate intake with no qualitative differences in the nature of the carbohydrate. A minor limitation of the study was that these were free-living individuals and although strongly motivated and closely supervised, the possibility of unreported deviation from the protocol exists.

The mechanisms underlying the different responses to butter fat and unsaturated fat appear to include down-regulation of LDL removal from the circulation, as measured by apolipoprotein B kinetics (Matthan *et al*, 2004). In that study, the LDL-C concentration was 14% higher after butter than when part of the saturated fatty acid content was replaced by linoleic acid.

Nutrition-related epidemiological associations have shown significant direct correlations between dairy fat consumption and cardiovascular outcomes (Turpeinen, 1979; Renaud & de Lorgeril, 1989; Artaud-Wild *et al*, 1993). Of particular interest are the comments regarding cheese that appear to differentiate its effect on plasma cholesterol from other high-fat dairy foods. In the 40-country study that showed a clear association between total dairy fat consumption and mortality from coronary disease, there was a paradoxical significant inverse correlation with cheese consumption, at least in those countries where saturated fat and cholesterol consumption were highest (Artaud-Wild *et al*, 1993). Renaud and de Lorgeril (1989), whose analysis of nutrients that affect CHD showed a positive association with dairy fats, suggested that cheese appeared to be an exception.

There are no ready explanations for our findings and those by Tholstrup *et al* (2004). The possible effect of fermentation, the physical state of fat globules and potential cholesterol-lowering components in cheese are worth considering. A meta-analysis of short-term trials of fermented dairy products such as yoghurt has suggested a possible cholesterol-lowering property through the high content of probiotic bacteria (Agerholm-Larsen *et al*, 2000). Others have found the effect to be inconsistent (McNamara *et al*, 1989). Fat globules in cheese that are encased in casein and phospholipid lose their envelope within butter. To what extent the higher phospholipid content of cheese might affect the transport and re-esterification of mono- and diacylglycerols in the enterocyte and whether positional changes in the triacylglycerol structure follow fermentation need exploring. The phospholipid concentration in the cheese was approximately 3/100 g or about 4 g in the amount of cheese eaten; however, since this did not induce a fatty acid profile different from that of butter, at least the linoleic acid content of phospholipid was unlikely to have had an effect. The fatty acid composition of the cheese and butter was similar (62 and 61%, respectively, for saturated fatty acids and 29 and 31% as monounsaturated fatty acids as proportions of total fat; total analysis not shown). Interesterification of fatty acids to produce new triacylglycerol structures does not itself influence the LDL-C response (Nestel *et al*, 1995). The issue of potential cholesterol-lowering constituents in milk has been much debated with one consensus meeting supporting such a possibility (Berner, 1993). On the other hand, Roberts *et al* (1982) showed that under metabolic unit controlled conditions, 38 g of milk fat or butter fat led to similar elevations in serum cholesterol. Casein *per se* does not appear to influence LDL-C differently from that of soy protein, at least in humans (Meinertz *et al*, 1988). Nevertheless, as shown in the DASH trial, the incorporation of low-fat dairy foods into a diet designed to lower blood pressure augmented the overall decline in cardiovascular risk (Appel *et al*, 1997) and raised the possibility of dietary calcium in the dairy foods contributing to the lower cholesterol concentration.

We have confirmed the recent findings from Denmark that eating cheese fat confers a lesser cholesterol-raising effect than eating a similar amount of butter. Subsequent to the completion of this study, Biong *et al* (2004) have reported similar findings in a study from Norway. The difference applies to the kind of cheese tested in this study, a matured cheddar and to subjects with modestly raised LDL-C concentrations. The latter finding suggests that inclusion of moderate amounts of cheese in the diet may need to be re-evaluated even for subjects with elevated plasma cholesterol.

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