Incorporation of cis-9, trans-11 conjugated linoleic acid and vaccenic acid (trans-11 18:1) into plasma and leucocyte lipids in healthy men consuming dairy products naturally enriched in these fatty acids

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(Received 5 January 2005 – Revised 13 April 2005 – Accepted 20 April 2005)

The present study investigated whether consuming dairy products naturally enriched in cis-9, trans-11 (c9,t11) conjugated linoleic acid (CLA) by modification of cattle feed increases the concentration of this isomer in plasma and cellular lipids in healthy men. The study had a double-blind cross-over design. Subjects aged 34–60 years consumed dairy products available from food retailers for 1 week and then either control (0·17 g c9,t11 CLA/d; 0·31 g trans-vaccenic acid (tVA)/d) or CLA-enriched (1·43 g c9,t11 CLA/d; 4·71 g tVA/d) dairy products for 6 weeks. After 7 weeks washout, this was repeated with the alternate products. c9,t11 CLA concentration in plasma lipids was lower after consuming the control products, which may reflect the two-fold greater c9,t11 CLA content of the commercial products. Consuming the CLA-enriched dairy products increased the c9,t11 CLA concentration in plasma phosphatidylcholine (PC) (38 %; P=0·035), triacylglycerol (TAG) (22 %; P<0·0001) and cholesterol esters (205 %; P<0·0001), and in peripheral blood mononuclear cells (PBMC) (238 %; P<0·0001), while tVA concentration was greater in plasma PC (65 %; P=0·035), TAG (98 %; P=0·001) and PBMC (84 %; P=0·004). Overall, the present study shows that consumption of naturally enriched dairy products in amounts similar to habitual intakes of these foods increased the c9,t11 CLA content of plasma and cellular lipids.

Conjugated linoleic acid: Blood lipids: Human diet: Dairy products

Conjugated linoleic acid (CLA) describes a group of eighteen-carbon fatty acids, which differ in the geometry and position of the two conjugated double bonds. The principal sources of CLA in the human diet are dairy products and meat from ruminants (Lawson et al. 2001), with cis-9, trans-11 (c9,t11) CLA being by far the major isomer consumed (Lawson et al. 2001). This isomer is formed as a result of biohydrogenation reactions carried out by bacteria in the rumen, which produce the precursor trans-11 octadecenoic acid (trans-vaccenic acid; tVA) and by Δ9 desaturase, which converts tVA to c9,t11 CLA, primarily in the mammary gland (Lawson et al. 2001).

Studies in animal models in which CLA intakes were increased show anti-tumorigenic activity (Ip et al. 1991; Belury, 2002), decreased atherogenesis (Lee et al. 1994; Nicolosi et al. 1997; Munday et al. 1999), decreased adiposity and increased lean body mass (Park et al. 1997; West et al. 1998; DeLany et al. 1999). Thus, greater CLA consumption may have beneficial effects on human health (Roche et al. 2001). However, the positive outcomes indicated by the animal studies have not been replicated in human studies (Calder, 2002; Kelley & Erickson, 2003; Watkins et al. 2004). This may reflect the type of the CLA preparations used in human studies. Some studies in human subjects have used synthetic mixtures of CLA isolomers, mainly c9,t11 CLA and t10,c12 CLA, with smaller amounts of other isomers, in which the amount of each isomer consumed is low relative to the total CLA intake (Benito et al. 2001; Kelley et al. 2001; Von Loefelholz et al. 2003). Other authors have reported the effects of 50:50 and 80:20 preparations of c9,t11 and t10,c12 CLA (Mougins et al. 2001; Masters et al. 2002; Noone et al. 2002; Albers et al. 2003) and others have used encapsulated triacylglycerols (TAG) highly enriched (≥80 %, w/w) in c9,t11 or t10,c12 CLA (Burdge et al. 2004). While low intakes of individual isomers in CLA mixtures may account for the limited effectiveness in altering biological outcomes in human subjects, consumption of approximately 2·5 g c9,t11 or t10,c12 CLA per day in highly enriched preparations also produced only modest effects on HDL- and LDL-cholesterol concentrations (Tricon et al. 2004b) and immune function (Tricon et al. 2004a), although the results suggested divergent outcomes for c9,t11 CLA and t10,c12 CLA with a relatively beneficial effect of the c9,t11 CLA isomer (Tricon et al. 2004b). One possible alternative

Abbreviations: c9, t11, cis-9, trans-11; CE, cholesterol ester; CLA, conjugated linoleic acid; FAME, fatty acid methyl esters; PBMC, peripheral blood mononuclear cells; PC, plasma phosphatidylcholine; TAG, triacylglycerol; tVA, trans-vaccenic acid; UHT, ultra-high temperature processed.

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DOI: 10.1079/BJN20051506
Explanation may be low bioavailability of CLA from synthetic TAG preparations. This is supported by the observation that when c9,t11 and t10,c12 CLA were consumed at 2.5 g/d for 8 weeks the increment in the concentrations of these isomers in plasma phosphatidylcholine (PC), cholesteryl esters (CE) and NEFA, although significant, was <0.5% and in leucocytes <0.2% (Burdge et al. 2004). Similar changes in the concentrations of these isomers in plasma or cellular lipids have been reported in other studies in human subjects where CLA intakes were increased (Benito et al. 2001; Masters et al. 2002; Noone et al. 2002; Petridou et al. 2003). Although direct comparisons between studies in human subjects and rodents are difficult, a similar magnitude of incorporation of c9,t11 CLA (0.4% total fatty acids) has been reported in the liver of rats fed diets containing chemically synthesised c9,t11 CLA (Sebedio et al. 2001; Alasnier et al. 2002). This suggests that a difference between species in the activity of CLA may account for differences in biological outcomes.

Even if CLA has a lower biological activity in man, its effectiveness may be enhanced by optimising the amount of CLA available to the body. It is possible that consumption of foods enriched in CLA by modifying fatty acid metabolism in ruminants may increase the bioavailability of CLA from human diets, which will be important if human consumers are to gain health benefits from increased intakes of specific CLA isomers. Chardigny et al. (2003) showed that TAG structure is an important determinant of CLA bioavailability in rats. Incorporation of CLA into TAG synthesised naturally may enhance bioavailability by presenting the fatty acid to the body in a structure more readily hydrolysed by lipase enzymes. In the present study, we describe the effect of consuming dairy products naturally enriched in c9,t11 CLA on the concentrations of c9,t11 CLA and t11 FA in plasma and cellular lipids using a double-blind, cross-over design. We report the incorporation of these fatty acids in plasma PC, CE, TAG and NEFA fractions and into peripheral blood mononuclear cells (PBMC).

Experimental procedures

Materials

Solvents were purchased from Fisher Scientific UK Ltd (Loughborough, Leicestershire, UK), and all other reagents were from Sigma (Poole, Dorset, UK). Fatty acid standards were from Sigma and Nu-Chek-Prep (Elysian, MN, USA).

Production of dairy products

A detailed description of the dietary interventions in the cattle to produce the control and CLA-enriched dairy products, the preparation of ultra-high temperature processed (UHT) milk, cheese and butter and the organoleptic properties of these foods will be published elsewhere (Jones et al. 2005). In brief, forty-nine early lactation Holstein–British Friesian cows were fed total mixed rations containing 0 (control) and 45 g/kg (on DM basis) of a mixture (1:2, w/w) of fish oil and sunflower-seed oil to standard practices. Triangle tests conducted on UHT milk (1 week after processing), butter (2 weeks after manufacture), and cheese (1 week after maturation) by a panel (n 20) of consumer volunteers showed that there were no significant differences in the flavour or visual properties of the products.

Subjects and study design

The study was carried out at two centres (about 60 miles apart) in the south of the UK and had a double-blind, cross-over design. Inclusion criteria were: male sex; BMI >18 kg/m² and <32 kg/m²; absence of diagnosed CVD, diabetes, liver or endocrine dysfunction, chronic inflammatory disease; no medication use; omnivorous eating habits; smoking ten or less cigarettes per d; alcohol consumption twenty-one units or less per week; no supplement (such as vitamins, fish oils or evening primrose oil) use. Volunteers fitting these criteria were screened for fasting plasma concentrations of cholesterol (3·0–8·0 mmol/l), TAG (0·5–3·0 mmol/l), and glucose (3·9–6·8 mmol/l). Ultimately thirty-two healthy men aged 34–60 years were recruited into the study. One subject withdrew after the first phase of the intervention for reasons not directly related to the study. Thirty-one subjects completed the study (eleven in Southampton, UK and twenty in Reading, UK). Identical protocols were used at both sites for administering the intervention trial and for the collection and storage of samples. The study was approved by the University of Reading Ethics and Research Committee and the South and West Hampshire Local Research Ethics Committee. Subjects gave written consent.

Subjects were assigned to consume either the control or CLA-enriched dairy products in random order, and asked to substitute their habitual dairy products for the experimental ones. The study took the following format. Subjects consumed commercially prepared milk, cheese and butter during a 7 d run-in period before consuming the experimental dairy products. The daily provision and consumption of individual dairy products is summarised in Table 1. The subjects then consumed the experimental products in amounts designed to provide 0·17 g c9,t11 CLA/d (control) or 1·43 g c9,t11 CLA/d (CLA-enriched) (Table 2). These products also provided 0·31 g or 4·71 g VA/d, respectively (Table 2). Daily provision of cheese was greater during the period when subjects consumed the CLA-enriched products in order to provide the same total intake of fat. This was because the fat content of the CLA-enriched milk was lower than that of the control milk. Subjects consumed either the control or CLA-enriched products for 6 weeks and then returned to consuming their habitual dairy products for a 7-week washout period. Subjects then consumed the commercial products for a second run-in period of 7 d before consuming alternate experimental products for a further 6 weeks.

After an overnight fast, blood samples (40 ml) were collected at baseline, after the run-in period, and after the 6 weeks intervention period. Lithium heparin was used as an anticoagulant. Blood was separated into plasma and cellular fractions by centrifugation using Histopaque mononuclear cell separation media (Sigma) (Kew et al. 2003). Plasma was collected and frozen in tubes containing butylated hydroxytoluene and stored at −20°C. PBMC were aspirated from the interface, washed with PBS and frozen at −20°C.

Analysis of food intakes

Subjects completed food diaries for 3 d, including two weekdays and one weekend day, during the recruitment and screening period before commencing the study as a marker of habitual intakes, and during each of the periods of consumption of the

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Table 1. Provision of dairy foods and intakes of dairy products before the run-in period of the study and during the period of consumption of the control or conjugated linoleic acid (CLA)-enriched foods

<table>
<thead>
<tr>
<th>Dairy products group</th>
<th>Habitual (mean and SD)</th>
<th>Provision (mean and SD)</th>
<th>Control</th>
<th>CLA-enriched</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Milk (ml/d)</td>
<td>212 23.8 500</td>
<td>500 470.2 14.4</td>
<td>500</td>
<td>491.4 8.6</td>
</tr>
<tr>
<td>Butter (g/d)</td>
<td>14.1 1.9 12.5</td>
<td>12.5 15.7 0.9</td>
<td>12.5</td>
<td>15.7 1.3</td>
</tr>
<tr>
<td>Cheese (g/d)</td>
<td>28.3 6.5 28.0</td>
<td>28.0 32.6 2.1</td>
<td>36.6</td>
<td>40.4 2.8</td>
</tr>
</tbody>
</table>

Mean values were significantly different from those during the pre-study phase: *P < 0.05, **P < 0.001 (Student’s paired t test). Mean value was significantly different from that for the control phase: †P < 0.001 (Student’s paired t test).
Concentration of cis-9, trans-11 conjugated linoleic acid in plasma and peripheral blood mononuclear cell lipids

There were no significant differences in the concentrations of the fatty acids routinely detected in each lipid fraction apart from c9,t11 CLA and rVA.

c9,t11 CLA was detected in all of the plasma and PBMC samples collected at the start and end of the intervention period. There were significant differences between plasma lipid classes and PBMC in the proportion of c9,t11 CLA present at the start of the intervention period (ANOVA; P<0.0001). The proportion of c9,t11 CLA in plasma TAG was significantly greater (P<0.0001) than in PBMC, NEFA, and CE, which did not differ significantly, while PBMC contained the least amount (P<0.0001 compared with plasma lipids) of this isomer (Table 2). The distribution of c9,t11 CLA between these lipid pools did not change when the subjects consumed the control or CLA-enriched dairy products, although the absolute concentrations varied according to the dietary intervention. There were no significant differences in the concentration of c9,t11 CLA between the start of the control intervention and the start of the intervention with the CLA-enriched dairy products (Table 3).

There was no significant difference between dietary regimens in the concentration of c9,t11 CLA within each lipid pool at the start of the intervention period (Table 3). Between the start and end of the intervention period when subjects consumed the control products there was a decrease in c9,t11 CLA concentration in plasma PC (38 %), TAG (22 %), NEFA (37 %) and CE (25 %), although there was no change in the c9,t11 CLA content of PBMC (Table 3). Consumption of the CLA-enriched dairy products resulted in an increase in the fractional concentration of c9,t11 CLA in plasma PC (42 %), TAG (161 %), CE (205 %) and PBMC (238 %), while there was no significant change in the concentration of this isomer in plasma NEFA (Table 3). These effects were reflected in significant differences at the end of the intervention period between consuming the control or CLA-enriched dairy products (Table 3).

There was no significant relationship between the proportion of c9,t11 CLA in any of the lipid pools measured at the start of the intervention or after consuming the control products. However, after consumption of the CLA-enriched dairy products there was a positive relationship between the concentration of c9,t11 CLA in plasma PC and in plasma TAG (r 0.36; P=0.0002) and PBMC (r 0.36; P=0.047). The proportion of c9,t11 CLA in plasma TAG was positively related to the concentration of this isomer in the CE fraction (r 0.7; P<0.0001).

Concentration of trans-vaccenic acid in plasma and peripheral blood mononuclear cell lipids

rVA was detected in plasma PC, TAG and NEFA fractions and PBMC at baseline and after the intervention periods. However, rVA was not detected in the CE fraction (Table 4). There was no significant difference in the fractional concentration of rVA between plasma PC, TAG and NEFA, and PBMC at the start of the intervention period. There were no significant differences in

| Table 2. cis-9, trans-11 (c9,t11) conjugated linoleic acid (CLA) and trans-vaccenic acid (rVA) content of dairy products and provision of these fatty acids during the periods of consuming the run-in, control and CLA-enriched dairy products. |  |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | c9,t11 CLA | rVA |  |
| | Run-in | Control | CLA-enriched | Run-in | Control | CLA-enriched |  |
| Content in dairy products |  |  |  |  |  |  |  |
| Milk (g/100 ml) | 0.02 | 0.02 | 0.14 | 0.03 | 0.03 | 0.45 |  |
| Butter (g/100 g) | 1.07 | 0.34 | 2.92 | 0.49 | 0.67 | 9.88 |  |
| Cheese (g/100 g) | 0.39 | 0.12 | 0.98 | 0.23 | 0.26 | 3.33 |  |
| Intake (g/d) |  |  |  |  |  |  |  |
| Milk | 0.1 | 0.1 | 0.70 | 0.15 | 0.16 | 2.25 |  |
| Butter | 0.13 | 0.04 | 0.37 | 0.06 | 0.08 | 1.24 |  |
| Cheese | 0.11 | 0.03 | 0.36 | 0.06 | 0.07 | 1.22 |  |
| Total | 0.34 | 0.17 | 1.43 | 0.27 | 0.31 | 4.71 |  |

| Table 3. Fractional cis-9, trans-11 conjugated linoleic acid (CLA) concentration (g/100 g) in plasma lipids and peripheral blood mononuclear cells (PBMC) (Mean values and standard deviations). |  |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | Control (n 31) | CLA-enriched (n 31) |  |
| | Start | End | Start v. end: P* | Start | End | Start v. end: P* | Comparison between food products |  |
| | Mean | SD | Mean | SD | Mean | SD | Mean | SD | Start v. start: P | End v. end: P |  |
| PC | 0.26 | 0.09 | 0.16 | 0.06 | <0.0001 | 0.24 | 0.07 | 0.34 | 0.12 | 0.035 | NS | <0.0001 |
| TAG | 0.51 | 0.18 | 0.40 | 0.16 | 0.017 | 0.46 | 0.19 | 0.20 | 0.26 | <0.0001 | NS | <0.0001 |
| NEFA | 0.27 | 0.07 | 0.17 | 0.12 | 0.018 | 0.24 | 0.25 | 0.35 | 0.25 | NS | NS | <0.001 |
| CE | 0.24 | 0.11 | 0.28 | 0.08 | 0.016 | 0.21 | 0.07 | 0.64 | 0.23 | <0.0001 | NS | <0.0001 |
| PBMC | 0.07 | 0.05 | 0.08 | 0.05 | NS | 0.08 | 0.06 | 0.27 | 0.19 | <0.0001 | NS | <0.0001 |

PC, phosphatidylcholine; TAG, triacylglycerol; CE, cholesteryl ester.

* Statistical comparisons between samples collected at the start and end of the study were by Student’s paired t test.
the concentration of rVA between the start of the control intervention and the start of the intervention with the CLA-enriched dairy products (Table 4). rVA concentration in plasma PC was lower (36%) at the end of the intervention period compared with the start after consumption of the control products, but there was no significant change in the rVA content of TAG, NEFA or PBMC (Table 4). Consumption of the CLA-enriched products, which contained more than 10-fold higher rVA than the control products, was associated with an increase in the proportion of rVA in plasma PC (65%), TAG (98%) and PBMC (84%), but there was no significant difference in the rVA content of the NEFA fraction (Table 4). These effects were reflected in significant differences at the end of the intervention period between consuming the control or CLA-enriched dairy products (Table 4).

There was no significant relationship between the proportion of rVA in any of the lipid pools measured at the start of the intervention or after consuming the control foods. However, after consuming the CLA-enriched dairy products the rVA content of plasma PC was positively related to the concentration of this isomer in plasma TAG (\( r = 0.42; P = 0.0195 \)) and in PBMC (\( r = 0.54; P = 0.0019 \)). The proportion of rVA in plasma TAG was also positively related to the concentration of this isomer in the NEFA fraction (\( r = 0.42; P = 0.019 \)).

**Discussion**

The present study shows that consuming dairy products naturally enriched in CLA, especially \( 9,11 \text{CLA} \), increases the concentration of the \( 9,11 \text{CLA} \) isomer in plasma and cellular lipids. This was accompanied by an increase in the concentration of rVA plasma and cellular lipids. One aim of the present study was to determine whether CLA status could be altered by consuming dairy products in amounts that would be achievable in the general population. The habitual consumption of milk was about half that required by the study intervention, while the intakes of cheese and butter were comparable. Milk provided approximately 50% of the \( 9,11 \text{CLA} \) consumed per d during the intervention period, with an equal contribution being made by the butter and cheese combined. Thus it is possible that consuming CLA-enriched products in amounts typical of the UK population may provide a means for raising \( 9,11 \text{CLA} \) intakes. For example, compared with commercially prepared products used during the run-in period, CLA-enriched cheese and butter alone would double the daily intake of this isomer.

c\( 9,11 \text{CLA} \) was present in all four plasma lipid classes and in PBMC total lipids at baseline, which would reflect intakes in the background diet. The concentration of this isomer was lower in plasma lipids after consumption of the control products compared with baseline, although there was no difference in the concentration of \( 9,11 \text{CLA} \) in PBMC. One possible explanation is that the intake of \( 9,11 \text{CLA} \) during the two run-in periods was greater than when subjects consumed the control products. This is consistent with previous findings that the concentration of \( 9,11 \text{CLA} \) in plasma lipids is highly dependent upon dietary intakes (Noone et al. 2002; Burdge et al. 2004).

Consumption of 1.4 g \( 9,11 \text{CLA} \) per d in the CLA-enriched products resulted in significant increases in the concentration of this isomer in plasma PC, CE, TAG and in PBMC total lipids, and a trend towards an increase in \( 9,11 \text{CLA} \) concentration in the NEFA fraction. \( 9,11 \text{CLA} \) was present in highest concentrations in the TAG fraction at baseline and after consuming the control or CLA-enriched foods, which suggests that, as in the rat (Banni et al. 2001), \( 9,11 \text{CLA} \) is preferentially incorporated into this lipid pool. One previous report described the use of dairy products fortified with \( 9,11 \text{CLA} \) from a synthetic source, although the concentration of this isomer in plasma or tissue lipid pools was not reported (Malpuech-Brugere et al. 2004). The present study shows for the first time that substituting dairy products naturally enriched in \( 9,11 \text{CLA} \) significantly increases the concentration of this isomer in plasma and cellular lipids. The concentrations achieved in plasma PC and CE were comparable with those obtained when subjects consumed encapsulated preparations containing 1.2 g \( 9,11 \text{CLA} \) per d (Burdge et al. 2004). The concentration of \( 9,11 \text{CLA} \) in PBMC total lipids was also similar to that reported previously using synthetic CLA preparations (Kelley et al. 2001; Burdge et al. 2004). Overall, these data suggest that provision of \( 9,11 \text{CLA} \) naturally incorporated into a food matrix does not appear to increase its bioavailability in man above synthetic sources of \( 9,11 \text{CLA} \).

Intakes of the \( 9,11 \text{CLA} \) precursor rVA were similar during the run-in period and when subjects consumed the control dairy foods. However, the daily intake of rVA was 17-fold greater when subjects consumed the CLA-enriched dairy products. The greater rVA content of the CLA-enriched products reflects increased ruminal biohydrogenation required to increase synthesis of \( 9,11 \text{CLA} \). There was no indication of differential partitioning of rVA between plasma PC, TAG and NEFA fractions. There was a significant decrease in rVA concentration in plasma lipids, although this was only significant for PC, when

**Table 4.** Fractional trans-vaccenic acid concentration (g/100 g) in plasma lipids and peripheral blood mononuclear cells (PBMC)

<table>
<thead>
<tr>
<th></th>
<th>Control (n 31)</th>
<th></th>
<th>CLA-enriched (n 31)</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Start</td>
<td>End</td>
<td>Start</td>
<td>End</td>
</tr>
<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
<td>SD</td>
</tr>
<tr>
<td>PC</td>
<td>0.28</td>
<td>0.12</td>
<td>0.18</td>
<td>0.08</td>
</tr>
<tr>
<td>TAG</td>
<td>0.36</td>
<td>0.24</td>
<td>0.26</td>
<td>0.16</td>
</tr>
<tr>
<td>NEFA</td>
<td>0.35</td>
<td>0.38</td>
<td>0.16</td>
<td>0.16</td>
</tr>
<tr>
<td>CE</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
</tr>
<tr>
<td>PBMC</td>
<td>0.49</td>
<td>0.35</td>
<td>0.58</td>
<td>0.37</td>
</tr>
</tbody>
</table>

*Statistical comparisons between samples collected at the start and end of the study were by Student’s paired t test.*

CLA, conjugated linoleic acid; PC, phosphatidylcholine; TAG, triacylglycerol; CE, cholesteryl ester; ND, not detected.
subjects consumed the control products despite similar intakes to the run-in periods. The explanation for this decrease is not clear. Consumption of the CLA-enriched products resulted in increased rVA concentration in plasma PC and TAG, and in PBMC, and a non-significant trend towards a higher rVA concentration in the NEFA fraction. Thus, intakes of dairy products naturally enriched in c9,t11 CLA that are sufficient to increase the concentration of this isomer in plasma and cellular lipid also increase the levels of rVA. This may be a possible cause for concern because increased intakes of the trans fatty acid elaidic acid (trans-9 18:1) are associated with greater risk of CVD (Zock & Katan, 1992). However, there is insufficient evidence to indicate whether similar effects are produced by increased consumption of rVA. Man is able to convert rVA to c9,t11 CLA (Turpeinen et al. 2002). Thus, enrichment of dairy products with rVA may represent an additional means of increasing availability of c9,t11 CLA within the body.

Overall, the present study indicates that it is feasible to increase c9,t11 CLA intakes in human consumers by the intake of dairy products naturally enriched in this isomer at intakes of these foods that do not greatly exceed those of the UK population. However, one of the drawbacks of natural enrichment of dairy products with CLA by the dairy feeding regimen used here is that there is also an increase in the level of rVA in milk and this fatty acid is also readily incorporated into plasma and cellular lipid pools. Thus strategies to enrich milk with c9,t11 CLA without the accompanying increase in rVA may be important for the future development of CLA-enriched dairy products. Together these data suggest that there is no obvious advantage in terms of the concentration of c9,t11 CLA in blood lipids and PBMC in attempting to increase c9,t11 CLA intake by providing modified dairy products against highly enriched encapsulated preparations.

Acknowledgements

The present study was funded by a grant (number EFH/16) to P. C. C., P. Y., C. M. W. and R. F. G. from the Biotechnology and Biological Sciences Research Council, Scottish Executive Environment and Rural Affairs Department, Department of Food and Rural Affairs and the Milk Development Council under the Eating, Food and Health LINK scheme.

References

Conjugated linoleic acid in blood and cell lipids


