Dietary Intake of c9,t11-Conjugated Linoleic Acid Correlates with Its Concentration in Plasma Lipid Fractions of Men but Not Women\textsuperscript{1,2}

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Abstract

The c9,t11–18:2 isomer of conjugated linoleic acid (c9,t11-CLA) represents the main dietary CLA form with putative health benefits. Whereas CLA intake influences the tissue CLA concentration, little is known about the association between dietary CLA and the CLA content of plasma lipid fractions. This study was designed to document fasting and nonfasting plasma c9,t11-CLA concentrations in a population of free-living adults (n = 94) and relate these concentrations to c9,t11-CLA intake. We also determined the c9,t11-CLA content of the primary plasma lipid fractions in a subset (n = 50) of our participants, related these to c9,t11-CLA intake, and determined whether c9,t11-CLA intake or plasma c9,t11-CLA was correlated with plasma cholesterol. Mean fasting plasma c9,t11-CLA concentrations were 0.48 ± 0.01 and 0.54 ± 0.01% (wt:wt) of total fatty acids for men and women, respectively (P < 0.05); nonfasting concentrations were 0.28 ± 0.01 and 0.38 ± 0.01% of total fatty acids, respectively (P < 0.001). All major esterified plasma lipid fractions contained c9,t11-CLA; TG had the highest percentages. In men, c9,t11-CLA intake correlated (r = 0.47; P < 0.05) with TG c9,t11-CLA content, suggesting that TG c9,t11-1CLA may serve as a biomarker for c9,t11-CLA intake. In females, there were no correlations between c9,t11-CLA intake and the c9,t11-CLA content of any esterified plasma lipid fraction. In neither sex was there a relation between dietary c9,t11-CLA or plasma c9,t11-CLA concentration and circulating lipoprotein cholesterol concentration. The influence of sex on circulating c9,t11-CLA content and further validation of biomarkers of c9,t11-CLA intake warrant further investigation. J. Nutr. 142: 1645–1651, 2012.

Introduction

CLA are a collection of positional and geometric isomers of linoleic acid (c9,c12–18:2) with c9,t11–18:2 being the main isomer. The diversity of health benefits associated with CLA has gained an increasing amount of attention in the past few years. In animals, CLA is a chemopreventive agent with possible protective associations with atherogenesis, diabetes, adiposity, and immune function (1). However, the link between CLA and human health has been less conclusive (2).

The lipid portion of food products derived from ruminant animals, such as dairy and beef, provides the majority of dietary CLA in the American diet, most of which is present in the form c9,t11–18:2 (3,4). Despite the availability of several methodologies designed to document dietary intake, such as semiquantitative FFQ, dietary records (DR)\textsuperscript{5}, and diet recalls, assessment of CLA intake of populations and individuals is challenging (4). In a large cross-sectional study in the northwest region of the United States, DR and FFQ methodologies were found to underestimate mean total CLA and c9,t11-CLA intakes compared with estimates obtained by biochemical analysis of 3-d food duplicates (FD) (4). Thus, neither DR nor FFQ may be appropriate to use for estimating CLA intakes of individuals, although they may be useful in estimating relative intakes among various populations. Therefore, more reliable and accurate methods to estimate dietary intakes of individuals, including biological markers thereof, should be investigated.

Because CLA is incorporated into various lipid fractions in the body, it is possible that biomarkers of CLA intake would

\textsuperscript{1} Supported by the Finnish Food Industry Research Program on Animal Fats, the National Cattlemen’s Beef Association, the Idaho Beef Council, and the United Dairymen of Idaho.


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\textsuperscript{5} Abbreviations used: CE, cholesterol ester; DR, dietary record; FD, food duplicate; HDL-C, HDL cholesterol; LDL-C, LDL cholesterol; MG-DG, monoglyceride/diglyceride; NEFA, nonesterified fatty acid; PL, phospholipid; TC, total cholesterol; VLDL-C, VLDL cholesterol.
include various plasma lipid fractions. For instance, the concentrations of 9,11-CLA found in plasma phospholipids (PL) and TG were associated with recent (7-d DR) and chronic (FFQ) c9, t11-CLA intake in a group of young German women (5). Moreover, recent investigations indicate a dose-dependent enrichment of the plasma lipid fractions of healthy individuals after intake of dietary or supplemental c9,t11-CLA (6–8).

The beneficial role of supplemental and dietary c9,t11-CLA intake on cardiovascular health has also been demonstrated in animals, specifically rabbits and hamsters (9–13). Although the literature related to its potential antiatherogenic effect in humans is growing, the beneficial effects of CLA remain controversial.

The study presented here provides data from a population for whom dietary CLA and c9,t11-CLA intakes were previously reported (4). We tested the following hypotheses: 1) concentrations of c9,t11-CLA when found esterified in plasma and plasma lipid fractions in this population fall within those previously reported; 2) a positive, linear relation exists between current c9,t11-CLA intake and nonfasting plasma esterified c9,t11-CLA concentration as well as between chronic c9,t11-CLA intake and fasting plasma esterified c9,t11-CLA concentration; 3) chronic c9,t11-CLA intake correlates with circulating indicators [i.e., cholesterol ester (CE) and PL] of medium-term (>2 wk) fatty acid intake, whereas current c9,t11-CLA intake correlates with indicators of short-term fatty acid intake (i.e., TG); and 4) neither c9,t11-CLA intake nor plasma c9,t11-CLA in any lipid fraction is associated with variation in selected cardiovascular disease risk factors.

### Participants and Methods

**Participants.** Men (n = 51) and women (n = 51) were recruited from the communities of Pullman, WA and Moscow, ID. To be eligible for inclusion, participants had to be between the ages of 18 and 60 years, self-reported healthy, and not suffering from eating disorders. A representative subsample of men (n = 21) and women (n = 28) was chosen for plasma lipid fraction analyses. Physical activity levels were assessed using defined categories (i.e., sedentary, slightly, moderately, very, and extremely active). The Institutional Review Board of Washington State University and the Human Assurances Committee at the University of Idaho approved all procedures used, and written informed consent was obtained.

**Study design.** Details concerning dietary and anthropometric assessment were previously reported (4). Briefly, current fatty acid intake was assessed using both FD and DR methodology over a 3-d period including 2 weekdays and one weekend day; chronic fatty acid intake was assessed using FFQ methodology. Data were analyzed using a computerized nutrient database (Food Processor, version 7.02; ESHA Research) modified to contain quantities of t10,c12-CLA and c9,t11-CLA. Composite FD were collected simultaneously with DR. On the last day of the FD data collection period between 1800 and 2130 h, nonfasting blood samples (20 mL) were collected into heparinized, vacuum-sealed tubes (VWR International) 30–90 min after the participant had consumed his/her evening meal. After centrifugation, plasma was harvested and stored at −70°C for later analysis. The following morning, fasting blood samples and anthropometric data were collected between 0600 and 0900 h. Anthropometric measurements included height, weight, and percent body fat as estimated by skin-fold measurements at 7 sites.

**Lipid extraction and fatty acid analyses.** Plasma lipids were extracted using a modified Folch procedure (14). An aliquot of the extracted samples was prepared for lipid fractionation by taking it to dryness under nitrogen; these were then weighed and stored in hexane/methyl tert-butyl ether (200:3, J.T. Baker). The remaining samples were prepared for total plasma esterified fatty acid analysis via methylation using a methanolic sodium methoxide solution (15).

### Plasma lipid fraction analyses.** The method developed by Hamilton and Comai (16) was used for separation of plasma lipid fractions. Due to limited column loading capacity, extracted lipid samples (1.5 mg) were fractionated in duplicate using silica Maxi-Clean cartridges (600 mg; Alltech Associates) and combined after fractionation. The following fractions were eluted in the order listed: CE, TG, nonesterified fatty acids (NEFA), monoglycerides/diglycerides (MG-DG), and PL. Due to difficulties in separation of the NEFA and MG-DG fractions, these 2 fractions were ultimately combined for analytical purposes. With the exception of CE, all fractions were methylated using sodium methoxide (Aldrich) (17). For complete methylation, the NEFA-containing fraction was further methylated using trimethylsilyldiazomethane (Aldrich) (18). CE were treated with sodium methoxide and methyl acetate in diethyl ether for 1 h to achieve complete methylation (15). Samples were analyzed using a gas chromatograph (HP 6890 Agilent Technologies) equipped with a capillary column (60 m, 0.25 mm i.d., with 0.25-μm film thickness; Cyanopropyl; Quadrex). Helium was used as carrier gas at a constant flow mode with linear velocity set at 20 cm/sec. The flame ionization detector was heated to 260°C and detector gas flows were set at 40, 450, and 49 mL/min for hydrogen, compressed air, and nitrogen, respectively. Samples (1 μL) were injected in the splitless mode (injection temperature 260°C), initial oven temperature of 50°C with 4 min hold time increased by 10°C/min to 200°C, and at 2°C/min to a final temperature of 240°C.

The identities of fatty acid peaks were established by comparing retention times to a 14-component C9-C24 FAME standard mixture (SigmaAldrich Chemical); CLA mixture containing 80% c9,t11-CLA, 17% c9,c11–18:2, and 1% t9,t11–18:2 (Matreya); pure t10,c12–18:2 (Matreya) and an anhydrous milk fat reference standard obtained from the Commission of the European Communities (CRM 164; European Community Bureau of Reference).

**Plasma TG and lipoprotein cholesterol.** Plasma TG, total cholesterol (TC), LDL cholesterol (LDL-C), HDL cholesterol (HDL-C), and VLDL cholesterol (VLDL-C) were determined using accepted clinical lipid chemistry methodologies. TC, HDL-C, and TG concentrations were quantified using an enzymatic, endpoint determination method utilizing an automated Roche Hitachi 917 analyzer (Roche Diagnostics). Concentrations of LDL-C and VLDL-C were estimated mathematically from TC and HDL-C concentrations (19).

**Statistical analyses.** Data analyses were performed using Minitab Statistical Software (release 13.0) and SAS (version 9.2). The statistical analysis was completed in 3 phases. First, descriptive statistics were compiled to estimate means, SE, and proportions where appropriate. Second, the statistical “effects” of nutritional state (fasting vs. nonfasting) and sex (male vs. female) on plasma lipid fraction fatty acid concentrations were examined using 2-way ANOVA; the interaction between nutritional state and sex was also evaluated.

Finally, to investigate the relations between c9,t11-CLA intake and plasma c9,t11-CLA concentration or plasma lipid fractions, Pearson correlation coefficients were calculated and multiple regression analyses conducted. Multiple regression analyses included the following independent variables: c9,t11-CLA intake (current, chronic), percent body fat, activity level, age, and sex. Similarly, to investigate the relation between c9,t11-CLA intake or plasma c9,t11-CLA concentration and risk factors of cardiovascular disease (i.e., TG, TC, HDL-C, LDL-C, VLDL-C, and LDL/HDL and TC/HDL ratios), the independent variables c9,t11-CLA intake (current, chronic) or plasma c9,t11-CLA (fasting vs. nonfasting), age, activity, saturated fat intake, and percent body fat were included in the model.

In all analyses, main effects were considered significant at P ≤ 0.05 and interaction terms were considered significant at P ≤ 0.1. Multiple comparisons were made using Tukey-Kramer tests. To meet assumptions of normality, data were log-transformed when necessary.

### Results

**Description of study population.** Detailed information concerning participants in this study was previously published.
(4). Briefly, the entire study population consisted of 47 participants (47 men and 47 women); however, dietary data were not available for 1 man (excluded from this paper) (Table 1). The demographic variables of the participants included in the lipid fraction subsample described here did not differ from those of the entire study population (data not shown). Whereas all men in this subsample were Caucasian, ethnicity among the women was as follows: 25 Caucasian, 1 Asian, and 2 Latina.

**Dietary intakes.** Detailed information concerning macronutrient and fatty acid intake of the entire population was previously published (4). Briefly, the mean, chronic, total CLA intake (c9, t11-CLA + t10,c12-CLA) estimated by FFQ was 197 ± 19 and 93 ± 11 mg/d for men and women, respectively, and the c9,t11-CLA intakes were 151 ± 15 and 72 ± 9 mg/d, respectively. Using FD, the current total CLA intake was estimated to be 212 and 151 mg/d and c9,t11-CLA intake 193 and 140 mg/d for men and women, respectively. No differences in intake of CLA intakes were 151 ± 6 mg/d for men and women, respectively. No differences in intake of BMI, height, and weight between men and women, respectively. No differences in intake of macronutrients, and individual fatty acids (estimated by 3-d DR or 3-d FD) were observed between the entire study population and the subsample (data not shown). 10

10 Please note that the term 18:2 will be used in the remainder of this manuscript to refer to linoleic acid (c9,c12-18:2) as opposed to other 18:2 isomers (e.g., c9, t11-CLA).

**Relations between nutritional state or sex and plasma fatty acids.** There were both independent and interactive effects of nutritional state and sex on several of the plasma fatty acids. Thus, the plasma fasting and nonfasting esterified fatty acid contents were summarized separately for each sex (Tables 2 and 3). For each sex, compared with nonfasting values, the concentration of fasting esterified c9,t11-CLA was ~30% higher (P < 0.05). In addition, the fasting and nonfasting concentrations of esterified c9,t11-CLA were higher in women than in men (P < 0.05). Although no consistent patterns among all the fatty acids were observed between men and women or fasting and nonfasting values, a positive correlation existed between fasting and nonfasting plasma esterified 10:0, 16:0, 17:0, 18:1, 18:2, 18:3, and c9,t11-CLA concentrations (r = 0.32–0.53; P < 0.05) in men and for esterified 14:0, 18:0, 18:2, and c9,t11-CLA (r = 0.41–0.53; P < 0.005) in women.

**Relations between nutritional state or sex and fatty acids in lipid fractions.** Except for the 14:0 concentration, which was lower in the subsample of men compared with the entire study population (1.0 vs. 1.3%, respectively; P < 0.05), no differences between the subset and entire study population were observed for the other fatty acids for either sex.

However, each plasma fraction exhibited distinct fatty acid patterns in both sexes in the fasting and nonfasting states (Table 3). The dominant fatty acid in CE was 18:2 followed by 18:1; in TG, the dominant fatty acid was 18:1. In the NEFA/MG-DG fraction, 16:0 and 18:0 represented the major fatty acids; for PL, 16:0 and 18:2 were predominant. Differences existed for both sexes in the concentration of c9,t11-CLA between the fasting and nonfasting states. In both sexes and nutritional states, TG contained the greatest enrichment of c9,t11-CLA, followed by CE and PL (P < 0.001), with the smallest amount in the NEFA/MG-DG fraction (P < 0.05). Fasting concentrations of c9,t11-CLA in the TG and NEFA/MG-DG fractions differed from those in the nonfasting state (P < 0.001).

Among women, plasma c9,t11-CLA concentrations were greater in the fasting state compared with the nonfasting state for CE (P < 0.001) and TG (P < 0.05) fractions, whereas the PL and NEFA/MG-DG fractions did not differ. Among men, plasma c9,t11-CLA concentrations were greater in fasting than nonfasting CE (P < 0.01) and PL (P < 0.05) fractions, with no difference observed in TG and NEFA/MG-DG fractions. For most fatty acids, differences existed between the fasting and nonfasting states, but the directionality was inconsistent.

**Relations between c9,t11-CLA intake and plasma/lipid fraction c9,t11-CLA content.** Multiple regression analyses indicated that neither current nor chronic c9,t11-CLA intake correlated with the c9,t11-CLA concentrations of fasting or nonfasting plasma or any fasting plasma lipid fraction. However, the results did suggest a statistical effect of sex (P < 0.05). As such, the results of the correlation analyses between c9,t11-CLA intake estimated by DR, FD, or FFQ and c9,t11-CLA content of fasting and nonfasting plasma lipid fractions are presented separately for each sex (Tables 4 and 5).

**Table 1** Demographic variables and anthropometric measurements

<table>
<thead>
<tr>
<th>Variable</th>
<th>Men</th>
<th>Women</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>32 ± 2</td>
<td>30 ± 2</td>
</tr>
<tr>
<td>Ethnicity, n</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Caucasian</td>
<td>43</td>
<td>40</td>
</tr>
<tr>
<td>Asian</td>
<td>3</td>
<td>5</td>
</tr>
<tr>
<td>Latino</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>81 ± 2</td>
<td>62 ± 2</td>
</tr>
<tr>
<td>Height, cm</td>
<td>178 ± 1</td>
<td>164 ± 1</td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td>25 ± 1</td>
<td>23 ± 1</td>
</tr>
<tr>
<td>Body fat, %</td>
<td>15 ± 1</td>
<td>21 ± 1</td>
</tr>
</tbody>
</table>

1 Values are mean ± SEM; n = 47.

**Table 2** Fasting and nonfasting plasma esterified fatty acids in men and women

<table>
<thead>
<tr>
<th>Fatty acid</th>
<th>Men</th>
<th>Women</th>
<th>% of identified FAME</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Fasting</td>
<td>Nonfasting</td>
<td>Fasting</td>
</tr>
<tr>
<td>10:0</td>
<td>0.19</td>
<td>0.17</td>
<td>0.12*</td>
</tr>
<tr>
<td>12:0</td>
<td>0.42</td>
<td>0.40</td>
<td>0.27*</td>
</tr>
<tr>
<td>14:0</td>
<td>1.33</td>
<td>1.22</td>
<td>1.03*</td>
</tr>
<tr>
<td>15:0</td>
<td>0.31</td>
<td>0.30</td>
<td>0.31</td>
</tr>
<tr>
<td>16:0</td>
<td>22.7</td>
<td>22.8</td>
<td>22.2</td>
</tr>
<tr>
<td>17:0</td>
<td>0.49</td>
<td>0.48</td>
<td>0.46</td>
</tr>
<tr>
<td>18:0</td>
<td>11.1</td>
<td>11.1</td>
<td>9.2*</td>
</tr>
<tr>
<td>18:1</td>
<td>14.9</td>
<td>14.8</td>
<td>15.0</td>
</tr>
<tr>
<td>18:2</td>
<td>18.3</td>
<td>20.3**</td>
<td>21.3*</td>
</tr>
<tr>
<td>c9,t11-CLA</td>
<td>0.46</td>
<td>0.28**</td>
<td>0.54*</td>
</tr>
<tr>
<td>c9,t11,15-18:3</td>
<td>0.63</td>
<td>0.70</td>
<td>0.79</td>
</tr>
</tbody>
</table>

1 Values are mean; n = 47. *Different from men, P < 0.05; **different from fasting, P < 0.05. 1 Interaction between sex and nutritional state, P < 0.01. 1 Interaction between sex and nutritional state, P < 0.05. 1 These fatty acids accounted for 70–75% of the esterified FAME identified in plasma.

Association between dietary and plasma CLA 1647

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TABLE 3  Fatty acid concentrations in fasting and nonfasting plasma lipid fractions from men and women

<table>
<thead>
<tr>
<th>Fatty acid</th>
<th>Men</th>
<th>Women</th>
</tr>
</thead>
<tbody>
<tr>
<td>CE</td>
<td></td>
<td></td>
</tr>
<tr>
<td>TG</td>
<td></td>
<td></td>
</tr>
<tr>
<td>MG-DG</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PL</td>
<td></td>
<td></td>
</tr>
<tr>
<td>NEFA/</td>
<td></td>
<td></td>
</tr>
<tr>
<td>MG-DG</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PL</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

% of identified FAME

10:0  
Fasting 0.17 0.59 1.30 0.07 0.13 0.38 0.70 0.06 0.14  
Nonfasting 0.12 0.07 0.16 0.08 0.03 0.11 0.18 0.13  

12:0  
Fasting 0.18 0.74 1.51 0.14 0.15 0.84 1.37 0.18 0.22  
Nonfasting 0.09 0.18 0.76 0.06 0.05 0.31 1.15 0.06  

14:0  
Fasting 0.67 2.41 3.70 0.61 0.85 3.11 4.37 0.88 0.59  
Nonfasting 0.59 1.23 2.13 0.22 0.55 1.60 2.43 0.27  

16:0  
Fasting 11.5 23.1 23.9 30.3 13.6 24.8 25.5 10.8 9.1  
Nonfasting 10.7 18.9 19.9 25.5 11.7 20.8 19.0 27.4  

18:1  
Fasting 2.26 2.71 1.58 0.46 3.25 3.64 1.35 0.73 0.52  
Nonfasting 2.05 1.81 0.69 0.39 3.00 3.09 0.81 0.64  

18:2  
Fasting 1.47 4.42 28.1 16.4 1.60 4.97 24.5 14.2 11.9  
Nonfasting 1.35 5.26 25.5 16.7 1.23 4.45 26.1 14.5  

18:3  
Fasting 17.9 33.7 10.5 10.8 19.5 30.7 13.7 12.0 9.1  
Nonfasting 17.7 31.5 9.24 10.8 18.5 29.6 9.22 10.6  

18:4  
Fasting 51.0 17.3 3.90 22.5 46.6 14.0 3.6 21.7 14.5  
Nonfasting 50.7 19.1 5.26 21.9 49.7 18.4 4.2 21.9  


c9,11-CLA intake (mg/d) estimated by FD was positively correlated with CE c9,11-CLA and PL c9,11-CLA. However, when c9,11-CLA intake was estimated using DR, it was best predicted by the c9,11-CLA content of CE in the fasting state. Intake of c9,11-CLA and plasma lipid fractions were not correlated in either nutritional state in women. When dietary c9, 11-CLA intake was estimated using FFQ, no correlations were present between any plasma lipid fraction and chronic c9,11-CLA intake for either sex.

Plasma TG, TC, and lipoprotein-C: relationships with plasma c9,11-CLA and c9,11-CLA intake. Plasma lipid and lipoprotein concentrations are shown in Table 6. Whereas women had higher HDL-C concentrations than men, male participants had higher ratios of LDL-C: HDL-C and TC: HDL-C. Multiple regression analyses indicated that the interaction between nonfasting c9,11-CLA concentration and sex was significant in explaining variations of HDL-C, LDL-C: HDL-C, and TC: HDL-C ratios (P < 0.1). There was no interactive effect of sex in any of the other models. Further results of multiple regression analyses indicated that current c9,11-CLA intake (estimated by FD) and age were independently and positively related with TC (P < 0.05). A weak positive relation (P = 0.08) was observed between current c9,11-CLA intake and plasma TG. Current and chronic c9,11-CLA intake did not correlate with any of the lipoprotein cholesterol concentrations or their associated ratios.

When fasting plasma c9,11-CLA was used as a predictor variable instead of c9,11-CLA intake, positive correlations (P < 0.05) existed between fasting plasma c9,11-CLA and TG, VLDL-C, and TC. In addition, age correlated positively (P < 0.001) with TC. No correlation existed between fasting plasma c9,11-CLA and HDL-C or LDL-C as well as their associated ratios. Furthermore, no correlations were found between nonfasting plasma c9,11-CLA and TC, TG, or LDL-C. The adjusted R² for the mentioned models ranged from 0.13 to 0.28.

Discussion

This study was designed to document the c9,11-CLA concentrations in plasma and plasma esterified lipid fractions (fasting and nonfasting) of healthy, free-living adults residing in North America and to evaluate the use of the c9,11-CLA concentrations of various plasma esterified lipid fractions as potential biomarkers of c9,11-CLA intake. Data from this study indicate that the mean fasting plasma c9,11-CLA concentrations were 0.46 and 0.54% of total fatty acids for men and women, respectively, and 0.28 and 0.38% of total fatty acids in nonfasting plasma, respectively. Overall, higher concentrations of c9,11-CLA were found in fasting compared with nonfasting plasma and men had lower circulating c9,11-CLA concentrations compared with women.

The mean fasting plasma c9,11-CLA concentrations of participants from different parts of the world have been documented in a number of studies, ranging from 0.16 to 0.40% of total fatty acids.
TABLE 5  Correlations between c9,t11-CLA intake (estimated by FD, DR, and FFQ) and c9,t11-CLA content of nonfasting lipid fractions in men and women

<table>
<thead>
<tr>
<th>Dietary intake methodology</th>
<th>Men</th>
<th>Women</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>CE</td>
<td>TG</td>
</tr>
<tr>
<td>3-d FD mg/d</td>
<td>0.57a</td>
<td>-0.31</td>
</tr>
<tr>
<td>% of total fatty acids</td>
<td>0.48a</td>
<td>-0.08</td>
</tr>
<tr>
<td>3-d DR mg/d</td>
<td>0.16</td>
<td>-0.20</td>
</tr>
<tr>
<td>% of total fatty acids</td>
<td>0.29</td>
<td>-0.06</td>
</tr>
<tr>
<td>FFQ mg/d</td>
<td>0.29</td>
<td>-0.06</td>
</tr>
</tbody>
</table>

1 Data are Pearson correlation coefficients for males (n = 22) and females (n = 28). Letters indicate significant correlations: *P < 0.05; **P < 0.01. CE, cholesterol ester; DR, dietary record; FFQ, food duplicate; PL, phospholipid.

TABLE 6  Plasma lipid and lipoprotein concentrations in men and women

<table>
<thead>
<tr>
<th>Lipid class</th>
<th>Men</th>
<th>Women</th>
</tr>
</thead>
<tbody>
<tr>
<td>TG, mmol/L</td>
<td>1.12 ± 0.07</td>
<td>1.02 ± 0.08</td>
</tr>
<tr>
<td>TC, mmol/L</td>
<td>4.74 ± 0.16</td>
<td>4.77 ± 0.15</td>
</tr>
<tr>
<td>HDL-C, mmol/L</td>
<td>0.91 ± 0.03</td>
<td>1.22 ± 0.05*</td>
</tr>
<tr>
<td>LDL-C, mmol/L</td>
<td>3.32 ± 0.16</td>
<td>3.11 ± 0.13</td>
</tr>
<tr>
<td>VLDL-C, mmol/L</td>
<td>0.52 ± 0.03</td>
<td>0.47 ± 0.03</td>
</tr>
<tr>
<td>LDL-C:HDL-C</td>
<td>3.8 ± 0.3</td>
<td>2.7 ± 0.2*</td>
</tr>
</tbody>
</table>

1 Values are mean ± SEM, n = 46. *Different from men, P < 0.05. HDL-C, HDL cholesterol; LDL-C, LDL cholesterol; TC, total cholesterol; VLDL-C, VLDL cholesterol.
that used CLA as part of a dairy-rich diet have shown no beneficial, but also no detrimental, effects of CLA on cardiovascular risk; our data generally support these findings (26,35,36). Epidemiological evidence also suggests no change in risk for coronary heart disease from ruminant trans fatty acid intake (37,38).

However, the results of our study indicate that current c9,t11-CLA intake and plasma esterified c9,t11-CLA concentrations are positively related to TC and that plasma esterified c9,t11-CLA is positively correlated with TG and VLDL-C. No correlation existed between c9,t11-CLA intake (current or chronic) or plasma (fasting and nonfasting) esterified c9,t11-CLA and HDL-C, LDL-C, or LDL-C:HDLC or TC:HDLC ratios. The possible link between c9,t11-CLA intake or plasma esterified c9,t11-CLA concentration and TC and TG concentrations requires further investigation. Consequently, our cross-sectional data are in agreement with human intervention studies, which generally do not support the notion that dietary or plasma esterified c9,t11-CLA is associated with variations in lipoprotein cholesterol concentrations and thus do not support the hypothesis that c9,t11-CLA consumption decreases the risk of cardiovascular disease in healthy adults.

The majority of research related to the impact of CLA intake on cardiovascular health in humans has focused on lipoprotein metabolism. However, evidence in animal models suggests that the antiatherogenic properties of CLA may be linked to its antiinflammatory properties (10,39,40). Consequently, future investigations require further investigation. Consequently, our cross-sectional data are in agreement with human intervention studies, which generally do not support the notion that dietary or plasma esterified c9,t11-CLA is associated with variations in lipoprotein cholesterol concentrations and thus do not support the hypothesis that c9,t11-CLA consumption decreases the risk of cardiovascular disease in healthy adults.

In summary, c9,t11-CLA enrichment of nonfasting plasma was lower than that of fasting samples in both men and women. All major plasma lipid fractions contained c9,t11-CLA, with the greatest enrichment observed in the TG fraction. Although human investigations indicate a weak relationship between c9,t11-CLA intake and total plasma c9,t11-CLA concentrations, no apparent relationship was observed in this cross-sectional study where FD (the “gold standard” of dietary intake methodology) rather than DR data were used to estimate intake in males. Nonetheless, our data suggest that TG c9,t11-CLA content is a potential biomarker of recent c9,t11-CLA intake, whereas the role of CE and PL c9,t11-CLA contents as biomarkers of c9,t11-CLA intake during previous weeks warrants further investigations. No potential biomarkers of c9,t11-CLA intake were identified for females. Moreover, our data do not support the notion that dietary c9,t11-CLA or plasma c9,t11-CLA concentrations are associated with variations in lipoprotein cholesterol concentrations. Further studies are necessary to investigate potential biomarkers and identify reliable and accurate methods to estimate c9,t11-CLA intake.

Acknowledgments
M.K.M., M.A.M., and T.D.S. designed the study; K.L.R. conducted the research; K.R.L. and A.M.S. performed sample analysis; N.D., K.L.R., and M.K.M. performed statistical analysis; and K.R.L., M.K.M., and M.A.M. primarily wrote the paper. All authors read and approved the final manuscript.

Literature Cited


