Genetic variation in fatty acid composition of subcutaneous fat in cattle

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Summary

The fatty acid composition of beef fat has been linked to flavour and consumer health. Our research demonstrated that those fatty acids individually constituting more than 2% of total fatty acids were moderately to highly heritable and genetically related to overall fatness. These results indicate that selective breeding could be used to change the composition of beef fat.

Abstract

Genetic parameters were estimated for fatty acid composition of subcutaneous beef fat of 1573 animals which were the progeny of 157 sires across seven breeds grown out on pasture and then finished on either grain or grass in northern NSW or in central Queensland. There was genetic variation in individual fatty acids with estimates of heritability for the proportions of C14:0, C14:1c9, C16:0, C16:1c9, C18:0 and C18:1c9 fatty acids in subcutaneous beef fat of the order of 0.4 or above. Also substantial correlations between some fatty acids were observed. Genetic correlations between fatty acids and fat depth at the P8 site suggested that much of the genetic variation in fatty acid composition was related to changes in fatness. Selection for decreased fatness resulted in decreased proportions of C18:1c9 with concomitant increases in C18:0, C14:0 and C16:0. This suggested that selection for decreased fatness at a given weight will result in a decrease in the proportions of mono-unsaturated fatty acids in the subcutaneous fat in the carcass with a corresponding increase in the proportions of saturated fatty acids.

Key Words: Genetic heritability, Genetic correlation, Fatty acids
Introduction

The manipulation of fatty acid composition of beef fat is seen as important for three reasons. Firstly to reduce saturated fatty acids that may contribute to increased serum cholesterol levels and coronary heart disease in humans, secondly the contribution of particular fatty acids to the flavour attributes of beef and lastly variation in fat softness (Smith et al. 2009). The latter trait can have different signals for the beef industry depending upon the specific market. Whilst softer fat is desirable in the Japanese market it does not attract a premium in Australian markets although there are indirect economic implications as hard fat can be a problem for workplace safety in boning rooms and result in additional costs involved with carcase re-warming as well as food safety and quality issues.

There are both production management and genetics options by which fatty acid composition may be manipulated. Management options via a change in diet are more difficult in ruminants than in monogastric animals, as rumen bacteria hydrogenate ingested unsaturated fat into more saturated fats (Harfoot 1981). Accordingly, St. John et al. (1987) concluded that genetic selection of cattle for changes in fatty acid composition might be more effective than nutritional manipulation. A more recent paper by Chung et al. (2006) suggested that that combining genetic selection and dietary manipulation together provided an effective way to modify carcass fatty acid composition.

The two avenues for making genetic changes in fatty acid composition include exploitation of breed differences via breed replacement or crossbreeding, or selection within breeds. There have been numerous reports of breed differences in fatty acid
composition of subcutaneous and intramuscular fat in cattle (Perry et al. 1998; Siebert et al. 1999; Xie et al. 1996b; Zemabyashi and Nishimura 1996). The most consistent demonstration of breed variation in fatty acid composition tends to be the Japanese black breed, which produce more unsaturated fat in intramuscular and subcutaneous depots than other breeds (Boyston et al. 1995; Siebert et al. 1999; Xie et al. 1996a) at a variety of weights and ages.

Pitchford et al. (2002) and Inoue et al. (2011) both reported heritability estimates for fatty acid composition. In the study by Inoue et al (2011) heritability estimates were extremely high for most fatty acids, (h² between 0.82 and 0.65 for all individual fatty acids except C18:2). By contrast, Pitchford et al. (2002) found heritability estimates that were much lower in crosses between beef, dairy and Japanese Black breeds (h² between 0.14 and 0.21). It is difficult to reconcile differences in these estimates as they comprised different breeds and production systems and were based on reasonably small populations.

The objective of this study was to estimate the heritability of fatty acid composition from a larger sample of purebred animals in a range of production systems representative of the diverse markets and finishes in Australia.

**Materials and Methods**

The dataset comprised fatty acid profiles from the subcutaneous fat depot taken from 1573 carcasses as part of the progeny test program set up by the Co-operative Research Centre (Upton et al. 2001). Briefly, carcasses were sampled from cattle grown out on pasture and finished on either grain or grass in northern NSW or in central Queensland. A total of seven breeds were represented including four *Bos Taurus* breeds and three tropically adapted breeds. These cattle represented progeny of 157 sires.
Cattle and management

The Cooperative Research Centre (CRC) straight-breeding experiment was designed to examine the effect of different finishing regimes and market weights on a variety of growth, carcass quantity and quality traits and to estimate genetic parameters for these traits (Bindon 2001). Breeds were chosen to represent the major breed types commercially used for beef production in Australia, although herd size requirements limited the choice of breeds. The temperate breeds included Angus, Murray Grey, Hereford and Shorthorn (Bos taurus), whilst the tropically adapted breeds Brahman, Santa Gertrudis (Bos indicus derived) and Belmont Red (Africander derived). Whilst the breeds were run in cohort groups from weaning they were bred on different properties and so between breeds comparisons were confounded by herd of origin.

Table 1 presents the number of cattle within each breed, number of sires and the average number of progeny per sire with data on fatty acid profiles. There was a total of 157 sires with an average of 9 progeny per sire (Table 1), although the number of progeny for individual sires ranged from 1 to 10.

A full description of the overall design and management procedures was described by Upton et al. (2001). Briefly, calves from a breeding program which ensured link sires between herds were purchased at weaning and transported to a CRC managed property for grow out. After arrival they were allocated to market and finishing groups based on breed, sex, property of origin and sire. The target markets endpoints were defined as domestic (400kg LW), Korean (520 kg LW) and Japanese (640 kg) market categories. Within these groups, half the cattle allocated to each market were finished on grain and the remainder on pasture. To reach these weights, domestic grain finished groups were fed for approximately 60 days, the Korean cattle for approximately 100 days, and the
Japanese cattle for approximately 150 days. The progression of cattle from
backgrounding to finishing and finally slaughter was based on the average liveweight of
animals within a market and finishing group (cohort). The range of weights within a
target market group was wide with domestic, Korean and Japanese target market groups
ranging between 124 and 296, 242 and 408, 182 and 393 kg, respectively.
Measurements at slaughter were those described in detail by Perry et al. (2001). This
included fat depth at the P8 site which is located at the intersection of a line parallel to
the spine from the tuber ischium and a line perpendicular to it from the spinous process
of the 3rd sacral vertebra.

*Fatty Acid composition*

Fatty acid composition was determined by gas-liquid chromatography (according to
the method of Smith et al. 1998). A 20 g sample of subcutaneous beef fat from the
12/13 rib junction was collected approximately 24 hours after slaughter and stored at -20°C. Fatty acids in the fat sub-sample (50-100 mg) were esterified in 3 mL of 0.25 M
sodium methoxide: diethyl ether (1:1, v/v) at 60°C for 2 min (Bannon et al. 1982).
After cooling to room temperature, 5 mL 5% NaCl was added and tubes re-capped and
vortexed. Fatty acid methyl esters were extracted with 5 mL petroleum ether (b.p. 60-
80°C). After the phases had separated a portion of the top phase was transferred to a 1.5
mL GC sample bottle. The fatty acid composition of the fat was determined by gas-
liquid chromatography (Shimadzu GC17 with AOC17 auto-injector) using a
50mx0.25mm fused silica capillary column (CP-Sil-88, ChromPak, Holland). A split
flow injection system was used (1:60 split ratio) with nitrogen as the carrier gas. A
column pressure of 111kPa was used giving a total flow of 57mL/min. The initial
temperature was 150°C incrementing at 5°C min⁻¹ until 235°C was reached. This
temperature was maintained for eight minutes. Individual fatty acids were identified by comparing the retention times with standards (Alltech, Sydney, Australia).

Mean percentage of fatty acids are presented in Table 2. When expressed as a proportion of the total fatty acids, those fatty acids with less than 0.25% were omitted from the data set and the proportions recalculated. This left a total of 15 fatty acids for analysis, but included an unknown fatty acid which comprised an average of 0.6% of total fatty acids.

Statistical Analyses

Initially single trait analyses were used to estimate heritability, genetic and phenotypic variances for individual fatty acids. Univariate models were fitted in ASREML (Gilmour et al. 2009). Fixed effects were modelled as:
nutrition x year + sex x herd x kill + hot carcass weight x (sex x herd x kill). The animal was effect was modelled as a random effect.

The sex x herd. x kill term accounted for the fixed effects of market category (domestic, Korean or Japanese slaughter weight), nutrition (feedlot or pasture finished), grow-out location (northern NSW or central (Queensland) and cohort (herd of origin). Hot standard carcass weight within sex x herd x kill was included as a covariate. The herd term accounted for breed effects as each herd only contained a single breed.

Multiple trait analyses were carried out in VCE (Groeneveld and Garcia-Cortez 1998) to estimate genetic and phenotypic correlations between fatty acid proportions and fat depth (mm) at the P8 site. The multiple trait analyses were restricted to the fatty acids with proportions greater than 2% and heritabilities that were significantly greater than zero. This was performed as including fatty acids with low heritabilities and or constituting low percentages of total fatty acids would have unstable correlation estimates.
Results

The mean proportions for the 15 individual fatty acids ranged from 0.26 to 38.61%. Three fatty acids, which included C16:0, C18:0 and C18:1c9 generally comprised 80 percent of the total fatty acids. The remaining fatty acids which included C18:3, C15:0, C18:2c9,t11, C19:0, C17:1, C18:2c9,c12, C14:1, C17:0 and C18:1c11 each comprised less than 2% of total fatty acids.

Proportions of most fatty acids in subcutaneous beef fat had heritabilities that were significantly different to zero and ranged from 0.21 for C18:1c11 to 0.56 for C18:1c9. Exceptions were C17:1, C18:1,t11, C18:2c9,c12 and C18:3 where the heritabilities were not significantly different from zero (P>0.05, Table 2).

The associated genetic and phenotypic correlations between the major fatty acids and hot carcase P8 fat depth are presented in Table 3. Generally the phenotypic correlations were of a similar magnitude to the genetic correlations, with notable exceptions being a higher positive genetic correlation between both C14:0 and C16:0 with C16:1c9 than the corresponding phenotypic correlation. In addition there was a low genetic correlation between C18:1c9 and C16:1c9, whereas the phenotypic correlation was moderately positive. It was also of note that the genetic correlations of P8 fat depth with the different fatty acid proportions bore little relationship to the phenotypic correlations between P8 fat depth and fatty acids proportions.

Genetic correlations between the short-chain fatty acids C14:0 and C16’s (C16:0 and C16:1c9) were positive. These fatty acids were negatively correlated with the C18 fatty acids. A negative correlation between C18:1c9 and C18:0 was also observed. All fatty acids were genetically correlated with P8 fat depth. At the same hot carcass weight,
genetically leaner cattle (ie decreased P8 fat depth) had lower percentages of C16:1c9 and C18:1c9 and higher proportions of C14:0, C16:0 and C18:0.

**Discussion**

Our analyses showed that the proportions of C14:0, C14:1c9, C16:0, C16:1c9, C18:0 and C18:1c9 fatty acids in subcutaneous beef fat had heritabilities of the order of 0.4 or above. These results were inbetween the results of Inoue *et al.* (2011) and Pitchford *et al.* (2002) who also reported heritabilities for fatty acid proportions. For example the heritability of C18:0 in the current study was 0.44 which compares with 0.71 and 0.14 in Inoue *et al.* (2011) and Pitchford *et al.* (2002), respectively. A similar trend was evident for a number of the other major fatty acids. Reasons for the differences between these two studies are unclear, however such variation in estimates of heritability between studies are not unusual, particularly if based on relatively low number of sires and progeny. In the meta analysis of Rios Utrera and Van Vleck (2004) heritability estimates varied between 0.15 and 0.92 for carcass weight (36 studies) whilst estimates varied between 0.03 and 0.84 for fat depth (34 studies).

The moderate to high heritabilities of many fatty acids and their correlation with other fatty acids and P8 fat depth has implications in current and future breeding programs. Australian beef breeding programs are currently focused on decreased fatness at a given weight or age. Our results suggest that breeding for decreased fat depth will increase the amount of C14:0, C16:0 and C18:0 at the expense of lower percentages of C18:1c9. As decreasing proportions of percentages of C18:1c9 have been associated with adverse human nutrition Yu and Etherton (1995) and beef flavour characteristics (Larick and Turner 1990; Melton *et al.* 1982; Yeo and Park 1983) the correlated responses to selection for decreased P8 fat depth are likely to be negative on these aspects of meat quality. Overall the correlations suggest that the resultant fat in
carcasses from cattle selected for low backfat would be more saturated and therefore harder, although its impact would be reduced because there would be less fat in the carcass.

While it is generally recommended that decreasing the intake of saturated fat and increasing unsaturated fat intake has a beneficial impact on human serum cholesterol (e.g. Adams et al. 2010), some studies have found that individual saturated fatty acids such as C18:0 have a neutral effect on serum cholesterol levels (Yu and Etherton 1995). Future breeding programs could include selection for improved fatty acid composition. The high heritabilities and the generally beneficial correlations between the major fatty acids (C14:0, C16:0, C18:0 and C18:1c9) could be used to improve the fatty acid composition of beef fat. The percentages of C18:1c9 which is beneficial for flavour, human nutrition and fat hardness could be increased at the expense of predominantly C14:0 and C16:0. CLAs may prevent the development of some forms of cancerous tumors (Chin et al. 1992), and the moderate heritability of this fatty acid suggested that selective breeding could be used to increase its proportion in beef fat. However the measurement of fatty acid composition for assessment of fat unsaturation is expensive, and the time delay in responses makes it impractical to include in any breeding objective, unless there were large premiums associated with the desired changes. In addition our results suggest that selection for increased unsaturated fatty acids would also result in increased P8 fat thickness.

A number of fatty acids (C18:1t11, C18:2c9,12c and C18:3) had heritabilities that were not significantly different to zero. This was not surprising given these fatty acids cannot be synthesised by the animal in vivo and are derived solely from the diet in the case of C18:2 and C18:3 (Harfoot 1981). Trans monounsaturated fatty acids are formed
in the rumen by hydrogenation of C18:2 and C18:3, and C17:1 is a product formed by desaturation of C17:0 (Fievez et al 2003) which originates from rumen bacteria. Regarding C15:0, C17:0, C19:0, we did not distinguish between the straight-chain odd-length or branched-chain fatty acids which can either originate from synthesis in the animal from propionyl-CoA, or from rumen bacteria respectively. Given the displayed high heritability it is suggestive that these may have been branched chain fatty acids. Also, whilst C18:2c9,t11 can be formed in the rumen, it is also readily synthesised by desaturation of C18:2t11 in adipose tissue (Bauman 1999) which could account for its apparent high heritability.

The correlated changes in fatty acids and fat depth may be suggestive of the mechanisms that are acting to cause changes in fatty acid composition since fat accumulation at any site results from the interplay of several physiological processes (Pothoven et al. 1974). These processes include deposition of fatty acids synthesised de novo, uptake of fatty acids from the bloodstream and lipolysis of stored fat (Pothoven et al. 1974). To extend this concept to changes in fatty acid composition in subcutaneous fat, de novo synthesis must be broken down into three stages, synthesis to C16:0, elongation of C16:0 to C18:0 and desaturation to C16:1c and C18:1c9. Genetic differences in any of these processes could lead to changes in the fatty acid composition of subcutaneous fat.

The negative correlations between C14:0, C16:0, C18:0 and C18:1c9 suggest that genetic differences in stearoyl-CoA desaturase (SCD) activities may exist between sires used in the present study. SCD is responsible for the conversion of C16:0 to C16:1c9 and C18:0 to C18:1c9. Genetic variation in SCD activity has been confirmed by a number of studies using genomic techniques. SNPs in SCD gene have been shown to
be associated with differences in fatty acid composition in a number of studies (Li et al. 2012; Maharani et al. 2012). Additionally, as time on a finishing diet (hence, carcass fatness) increases, stearoyl-CoA desaturase (SCD) activity increases (Chung et al. 2007; Brooks et al. 2011), which is in agreement with our observations where a positive genetic correlation was shown for P8 fat depth and C18:1c9 percentage and a negative correlation for P8 and C18:0 percentage. The correlations from our study suggested that there was substantial genetic variation in SCD activity. However, control mechanisms over SCD are diverse, and hence this would require validation of the importance of these metabolic pathways.

The same complexity applies to the regulation of elongase and fatty acid synthetase. Literature on elongase activity is sparse. There is some evidence for genetic differences in elongase activity in this study as C14:0 exhibited a positive correlation with C16:0, although there was little correlation between C14:0 with C18:0 which suggested that some sires exhibited a propensity to elongate specific but not all fatty acids.

**Conclusion**

This study showed that fatty acid proportions of many fatty acids were moderately to highly heritable. The three fatty acids (C16:0, C18:0 and C18:1c9) constituting 80% of total fatty acids all had high heritabilities (0.43, 0.44 and 0.56 respectively). However, before fatty acid composition could be included in a breeding program, more information on the relationship between fatty acid composition and other production traits such as fatness and fertility would be required, and economic weights would need to be calculated. Currently, many breeding programs aim to decrease fatness at a given weight. Our results showed that decreasing carcass fatness would result in increased proportions of C14:0, C16:0 and C18:0 and decreased proportions of C18:1c9 in
subcutaneous fat. Whilst this would result in more saturated harder fat the impact of this on fat hardness would be decreased as there would also be a reduction in the total amount of fat.

References


Li C, Aldai N, Vinsky M, Dugan MER, McAllister TA (2011) Association analyses of single nucleotide polymorphisms in bovine stearoyl-CoA desaturase and fatty acid synthase genes with fatty acid composition in commercial cross-bred beef steers. Animal Genetics 43, 93-97


Table 1. Number of cattle and sires per breed and average number of progeny per sire for the six breeds used in variance component estimation

<table>
<thead>
<tr>
<th>Breed</th>
<th>Number of animals</th>
<th>Number of sires</th>
<th>Average progeny per sire</th>
</tr>
</thead>
<tbody>
<tr>
<td>Angus</td>
<td>492</td>
<td>58</td>
<td>8</td>
</tr>
<tr>
<td>Brahman</td>
<td>178</td>
<td>18</td>
<td>10</td>
</tr>
<tr>
<td>Belmont Red</td>
<td>273</td>
<td>24</td>
<td>11</td>
</tr>
<tr>
<td>Murray Grey</td>
<td>119</td>
<td>17</td>
<td>7</td>
</tr>
<tr>
<td>Santa Gertrudis</td>
<td>392</td>
<td>20</td>
<td>6</td>
</tr>
<tr>
<td>Shorthorn</td>
<td>119</td>
<td>20</td>
<td>6</td>
</tr>
<tr>
<td>Total</td>
<td>1573</td>
<td>157</td>
<td>9</td>
</tr>
</tbody>
</table>
Table 2 Raw proportions, standard deviations, heritability ($h^2$), standard errors and phenotypic standard deviations of P8 fat depth and individual fatty acids in subcutaneous beef fat.

<table>
<thead>
<tr>
<th>Fatty Acid</th>
<th>Raw Mean</th>
<th>Standard Deviation</th>
<th>Heritability $\pm 0.12$</th>
<th>Phenotypic variance $\pm 0.01$</th>
</tr>
</thead>
<tbody>
<tr>
<td>C14:0</td>
<td>3.54</td>
<td>0.67</td>
<td>0.55 $\pm 0.12$</td>
<td>0.31 $\pm 0.01$</td>
</tr>
<tr>
<td>C14:1c9</td>
<td>1.05</td>
<td>0.49</td>
<td>0.51 $\pm 0.12$</td>
<td>0.10 $\pm 0.00$</td>
</tr>
<tr>
<td>C15:0</td>
<td>0.56</td>
<td>0.15</td>
<td>0.43 $\pm 0.11$</td>
<td>3.65 $\pm 0.17$</td>
</tr>
<tr>
<td>C16:0</td>
<td>27.71</td>
<td>2.62</td>
<td>0.43 $\pm 0.11$</td>
<td>3.65 $\pm 0.17$</td>
</tr>
<tr>
<td>C16:1c9</td>
<td>4.18</td>
<td>1.42</td>
<td>0.38 $\pm 0.11$</td>
<td>0.87 $\pm 0.04$</td>
</tr>
<tr>
<td>C17:0</td>
<td>1.41</td>
<td>0.45</td>
<td>0.28 $\pm 0.10$</td>
<td>0.06 $\pm 0.00$</td>
</tr>
<tr>
<td>C17:1c9</td>
<td>0.93</td>
<td>0.22</td>
<td>0.13 $\pm 0.08$</td>
<td>0.02 $\pm 0.00$</td>
</tr>
<tr>
<td>C18:0</td>
<td>13.97</td>
<td>3.65</td>
<td>0.44 $\pm 0.11$</td>
<td>5.97 $\pm 0.28$</td>
</tr>
<tr>
<td>C18:1t11</td>
<td>3.33</td>
<td>1.78</td>
<td>0.12 $\pm 0.08$</td>
<td>1.00 $\pm 0.04$</td>
</tr>
<tr>
<td>C18:1c9</td>
<td>38.61</td>
<td>4.19</td>
<td>0.56 $\pm 0.12$</td>
<td>7.60 $\pm 0.36$</td>
</tr>
<tr>
<td>C18:1c11</td>
<td>1.47</td>
<td>0.45</td>
<td>0.21 $\pm 0.09$</td>
<td>0.07 $\pm 0.00$</td>
</tr>
<tr>
<td>C19:0</td>
<td>0.59</td>
<td>0.24</td>
<td>0.22 $\pm 0.09$</td>
<td>0.02 $\pm 0.00$</td>
</tr>
<tr>
<td>C18:2c9,c12</td>
<td>1.00</td>
<td>0.43</td>
<td>0.06 $\pm 0.07$</td>
<td>0.03 $\pm 0.00$</td>
</tr>
<tr>
<td>C18:3</td>
<td>0.26</td>
<td>0.15</td>
<td>0.00 $\pm 0.00$</td>
<td>0.00 $\pm 0.00$</td>
</tr>
<tr>
<td>C18:2c9,t11</td>
<td>0.57</td>
<td>0.32</td>
<td>0.24 $\pm 0.09$</td>
<td>0.02 $\pm 0.00$</td>
</tr>
</tbody>
</table>

Common fatty acid indexes

<table>
<thead>
<tr>
<th></th>
<th>Raw Mean</th>
<th>Standard Deviation</th>
<th>Heritability $\pm 0.12$</th>
<th>Phenotypic variance $\pm 0.04$</th>
</tr>
</thead>
<tbody>
<tr>
<td>SAT</td>
<td>51.12</td>
<td>5.36</td>
<td>0.54 $\pm 0.12$</td>
<td>11.24 $\pm 0.54$</td>
</tr>
<tr>
<td>MONO</td>
<td>46.24</td>
<td>5.37</td>
<td>0.53 $\pm 0.12$</td>
<td>11.40 $\pm 0.54$</td>
</tr>
<tr>
<td>PUFA</td>
<td>1.26</td>
<td>0.38</td>
<td>0.05 $\pm 0.07$</td>
<td>0.04 $\pm 0.00$</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Hot P8 fat depth (mm)</th>
<th>Raw Mean</th>
<th>Standard Deviation</th>
<th>Heritability $\pm 0.10$</th>
<th>Phenotypic variance $\pm 0.58$</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>11.23</td>
<td>5.45</td>
<td>0.28 $\pm 0.10$</td>
<td>12.92 $\pm 0.58$</td>
</tr>
</tbody>
</table>

Note: SAT = total saturated fatty acids; MONO = total monosaturated fatty acids; PUFA = total polyunsaturated fatty acids (Note MONO and PUFA excluded trans fatty acids)
Table 3 Genetic (above diagonal) and phenotypic (below diagonal) correlations between P8 fat depth (HP8) and selected fatty acid proportions in subcutaneous beef fat.

<table>
<thead>
<tr>
<th></th>
<th>HP8</th>
<th>C14:0</th>
<th>C16:0</th>
<th>C16:1c9</th>
<th>C18:0</th>
<th>C18:1c9</th>
</tr>
</thead>
<tbody>
<tr>
<td>HP8</td>
<td>-</td>
<td>-0.19</td>
<td>-0.21</td>
<td>0.15</td>
<td>-0.46</td>
<td>0.56</td>
</tr>
<tr>
<td>C14:0</td>
<td>0.05</td>
<td>-</td>
<td>0.55</td>
<td>0.46</td>
<td>-0.09</td>
<td>-0.62</td>
</tr>
<tr>
<td>C16:0</td>
<td>0.06</td>
<td>0.68</td>
<td>-</td>
<td>0.56</td>
<td>-0.26</td>
<td>-0.68</td>
</tr>
<tr>
<td>C16:1c9</td>
<td>-0.04</td>
<td>0.08</td>
<td>0.16</td>
<td>-</td>
<td>-0.76</td>
<td>-0.04</td>
</tr>
<tr>
<td>C18:0</td>
<td>0.08</td>
<td>-0.10</td>
<td>-0.32</td>
<td>-0.82</td>
<td>-</td>
<td>-0.46</td>
</tr>
<tr>
<td>C18:1c9</td>
<td>-0.13</td>
<td>-0.70</td>
<td>-0.47</td>
<td>0.28</td>
<td>-0.36</td>
<td>-</td>
</tr>
</tbody>
</table>