Acute postprandial effect of hydrogenated fish oil, palm oil and lard on plasma cholesterol, triacylglycerol and non-esterified fatty acid metabolism in normocholesterolaemic males

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(Received 31 May 2005 – Revised 8 December 2005 – Accepted 9 December 2005)

The majority of research has focused on the association between trans unsaturated fatty acids (TUFAs) from hydrogenated vegetable oils and heart disease even though TUFAs are also produced from hydrogenated fish oil. We compared the acute effect of three solid fats on postprandial cholesterol, triacylglycerol (TAG) and NEFA concentrations in normocholesterolaemic males. Eight healthy male volunteers consumed each of the three 40 g fat meals (partially hydrogenated fish oil (PHFO), palm oil and lard) in random order and blood samples were drawn at 2, 4, 6 and 8 h thereafter for lipid analysis. The postprandial response in plasma TAG, TAG-rich lipoprotein-TAG (TRL-TAG), total cholesterol and plasma NEFA, measured as the area under the postprandial curve, was not significantly different between the three meals (P>0·05), which varied in MUFA, PUFA and TUFAs content. There was no marked elevation of longer-chain fatty acids (C20–22, cis or trans isomers) into the TRL-TAG fraction following the PHFO meal even though they provided 40 % of the total fatty acids in the PHFO meal. The postprandial TRL-TAG response to PHFO was expected to be higher, as it is higher in TUFAs, lower in PUFA and similar in saturated fatty acid composition compared with the lard and palm oil test meals. The absence of a higher postprandial response following ingestion of PHFO could be as a result of reduced absorption and increased oxidation of long-chain fatty acids (both cis and trans isomers).

Hydrogenated fish oil: Trans unsaturated fatty acids: Palm oil: Lard: Postprandial response

Postprandial lipaemia refers to the series of events which occurs following ingestion, absorption and metabolism of a fat-rich meal and it is well understood that a prolonged and elevated response precipitates a number of adverse metabolic events. These include the production of atherogenic chylomicron remnants, small dense LDL particles, a reduction in beneficial HDL-cholesterol, and an adverse effect on the process of thrombosis. It is therefore of benefit to identify fats which produce a lower postprandial response and therefore a lower risk of developing CHD (Zilversmit, 1979; Patsch et al. 1992; Karpe et al. 1993). The acute postprandial response to varying amounts and types of fat has been studied extensively with the exception of fats high in trans unsaturated fatty acid (TUFAs).

Animal fats such as lard, and partially hydrogenated oils, are commonly used in the food industry as components of margarine and processed foods because of their solidity and higher melting points. There is a consensus, however, that saturated fatty acids (SFA), from animal fats, and TUFAs, from partially hydrogenated oils, should be reduced in the diet of the general population (Expert Panel on Detection, Evaluation and Treatment of High Blood Cholesterol in Adults, 2001). It is recommended that SFA and TUFAs should provide no more than 7 % (Expert Panel on Detection, Evaluation and Treatment of High Blood Cholesterol in Adults, 2001) and 2 % of energy intake respectively (Department of Health, 1991). Dietary reference intakes are not available for SFA or TUFAs intake, as increased risk exists at levels above zero. The Institute of Medicine therefore does not provide an upper limit of intake and recommends eating as little as possible of these fats (National Academy of Sciences, 2002).

Habitual intake of TUFAs from partially hydrogenated vegetable oils has been shown to increase levels of plasma total and LDL-cholesterol levels (Mensink & Katan, 1990; Nestel et al. 1992), lower plasma HDL-cholesterol (Zock & Katan, 1992) and increase lipoprotein(a) (Mensink et al. 1992; Zilversmit, 1979).

Abbreviations: AUC, area under the postprandial curve; Cmax, maximum postprandial concentration; Cmin, minimum postprandial concentration; PHFO, partially hydrogenated fish oil; PHSO, partially hydrogenated soybean oil; SFA, saturated fatty acid; TAG, triacylglycerol; T max, time to maximum postprandial concentration; TRL, triacylglycerol-rich lipoprotein; TUFAs, trans unsaturated fatty acid.

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Almendingen et al. 1995). Recommendations to reduce dietary
TUFA intake have been made based on these lipid studies
along with evidence from epidemiological studies that have
shown an increased risk of CVD (Ascherio et al. 1994) and
acute myocardial infarction (Willett et al. 1993) with an
increased TUFA intake. Studies which have examined the
effects of TUFA on plasma lipids and lipoproteins have
used hydrogenated vegetable oils, and there is currently very
little information regarding TUFA from hydrogenated fish
oil, fats which are also used by the food industry. Of the
few studies which have examined the effect of TUFA from
partially hydrogenated fish oils (PHFO), results indicate that
PHFO is at least as potent as butterfat, and significantly
more potent than partially hydrogenated soyabean oil
(PHSO) in raising both plasma total and LDL-cholesterol
levels (Almendingen et al. 1995). In addition, Muller et al.
(1998) reported LDL-cholesterol concentrations which were
19 % higher in participants after 2 weeks of consuming a mar-
garine based on PHFO compared with a margarine based on
vegetable oils. The LDL:HDL ratio was 12.6 % higher in par-
ticipants on the PHFO margarine compared with those on the
vegetable oil margarine.

The issue arises as to whether there are benefits to replacing a
product which is rich in SFA, such as lard, with a product which
is not only rich in SFA, but which is also rich in TUFA, such as
PHFO. The effect of TUFA on blood lipids and lipoprotein con-
centrations has been studied following long-term (21 d) inges-
tion of TUFA compared with oleic acid (18 : 1; Judd et al.
1994), linoleic acid (18 : 2n-6; Lichtenstein et al. 1993), or
PHSO and butter (Almendingen et al. 1995). The reported
effects of TUFA, in these long-term experiments, represent a
steady state of production and catabolism. They do not provide
insight into the mechanism by which TUFA may interfere with
the complex process of assembly, secretion and metabolism of
lipoproteins. The acute postprandial lipaemic effects of TUFA
on lipid and lipoprotein levels is therefore of interest but has
not been studied extensively.

The aim of the present study was two-fold. Since it is
known that a prolonged and elevated acute postprandial
response is associated with adverse metabolic events, we
wanted to compare the lipaemic response to PHFO, lard and
palm oil, three textured fats commonly used by the food indus-
try. This may help to assess whether there are benefits to
replacing a product which is rich in SFA, such as lard, with a
product which is not only rich in SFA, but which is also
rich in TUFA, such as PHFO. In addition we were interested
in knowing how long-chain TUFA from PHFO are absorbed
and cleared from plasma.

Methods

Study design

The participants were eight healthy men with a mean age of
26-1 (SD 3-1) years, mean weight of 84-6 (SD 9-6) kg and a
mean BMI of 25-7 (SD 2-2) kg/m² and were recruited from the
personnel of St James’s Hospital, Dublin, Republic of
Ireland. The study was approved by the Ethics Committee of
the Federated Dublin Voluntary Hospital and all
participants gave informed consent. The following inclusion
criteria were used: BMI 20–30 kg/m², fasting plasma
cholesterol < 6.5 mmol/l, fasting plasma triacylglycerol (TAG)
< 2.0 mmol/l, Hb > 13.0 g/dl and γ-glutamyl transferase < 50
units, <90 min strenuous exercise per week and participants
could not be habitual consumers of any fatty acid supplement
or medication known to affect lipid metabolism.

Participants were asked to refrain from eating oily fish or
from doing strenuous exercise for 24 h and to fast for 12 h
before the test day. A 21 gauge, 32 mm venous catheter
(Abbott Ireland Ltd, Dublin, Republic of Ireland) was inserted
into the antecubital vein of the forearm and a fasting sample
was collected. One of the three test meals was then taken by
each volunteer and blood samples were drawn at 2, 4, 6 and
8 h for lipid analysis. Each volunteer ingested each of three
test meals using a Latin square design and a 2-week wash-out period between test meals.

Test meal composition

The test meals were liquid blends of 40 g of one of the test fats
(lard, PHFO (Trilyb Trading Ltd, Republic of Ireland) or palm
oil) gently melted and whisked with 150 ml skimmed milk,
15 g skimmed milk powder (Tescos) and 15 g chocolate fla-
vouring (Nestlé, Vevey, Switzerland) to form a homogeneous
drink. Pasteurised egg-yolk powder (Lactosan UK Ltd,
Brantree, Essex, UK) was added to the lard (2.83 g) and
palm oil (4.2 g) test meals to equalise the cholesterol com-
position of the test meals (Tables 1 and 2).

Biochemical analysis

Following collection, blood samples were immediately cen-
trifuged at 2500 rpm for 15 min, then the plasma was harvested,
sampled and stored at −20°C. Enzymic colourimetric assays
were used to determine plasma TAG, TAG-rich lipoprotein
(TRL)-TAG (TAG PAP; Biomerieux, Lyon, France), plasma
cholesterol (Chol PAP; Biomerieux) and plasma NEFA concen-
trations (acyl Co A synthase-acyl Co A oxidase; Wako Chemi-
cals, GmbH, Neuss, Germany) on a RA-XT clinical chemistry
analyzer (Technicon Inc., Tarrytown, NY, USA). The NEFA
enzymic colourimetric assay may underestimate the concen-
tration of longer-chain fatty acids as the enzyme acyl-CoA
synthetase does not measure C20 and C22 fatty acid composition
accurately (Shimizu et al. 1980). The inter-assay CV were 2.5 %
for plasma TAG, 2.4 % for TRL-TAG, 1.48 % for cholesterol
and 3.84 % for NEFA.

Plasma for TRL separation was stored overnight (2–5°C).
The plasma TRL fraction (chylomicrons) was prepared using a
modified version of Grundy & Mok (1976), as follows. Two
4.7 ml Optiseal polyallomer centrifugation tubes (Beckman
Instruments Inc., Palo Alto, CA, USA) were required for each
eight sample’s TRL separation. Plasma (1-6 ml) was placed
into each tube and overlaid with 1-6 ml saline (density 1-006 g/ml).
The TRL fraction was isolated by ultracentrifugation
(100 000g for 24 min (2-2 × 10⁶g/min), at 4°C) (Beckman
Optima TLX ultracentrifuge, Beckman Instruments Inc.),
harvested and stored (−20°C) for subsequent analysis.

The procedure of Folch et al. (1957) was used to extract TRL-
TAG and the lipid component of plasma was extracted using the
procedure of Dole (1956), for NEFA analysis. The TAG fraction of
TRL samples and the NEFA fraction of plasma samples were

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https://www.cambridge.org/core/terms. https://doi.org/10.1079/BJN20051723
isolated using TLC on silica 60LKD 19-lane TLC plates (Whatman, Clifton, NJ, USA) using a solvent system of light petroleum (40–60°C), diethyl ether and formic acid (80:20:2, by vol.) (Gibney & Bolton-Smith, 1988).

Component fatty acids were methylated using BF₃ in methanol. GLC was used to identify fatty acid methyl esters of TRL-TAG and plasma NEFA fractions taken at 0, 4 and 8 h postprandially, using a 100 m capillary column (SP 2560, 0.25 mm, 0.2 m) and the following temperature program; starting temperature 175°C, held for 35 min, increased by 2.5°C/min to 220°C, held for 15 min, increased by 2.5°C/min to 240°C, and held for 14 min (Hodgson et al. 1996). A split ratio of 1:50, and a flow rate of 0.7 ml/min and an injection volume of 1 μl were used.

The molar % of fatty acids was calculated in both the PHFO test meal and in the 4 h TRL-TAG samples following consumption of the PHFO meal as follows. The number of moles of all fatty acids in the PHFO meal and in the TRL-TAG sample was divided by the total number of moles of all fatty acids in the PHFO meal and in the TRL-TAG sample respectively, and multiplied by 100.

**Statistical analysis**

All statistical analysis was completed using Data Desk 4.1 (Data Description Inc., New York, NY, USA). Repeated-measures ANOVA, using meal as the independent variable, investigated changes in the postprandial variations of plasma TAG, TRL-TAG, cholesterol, and NEFA concentrations, and for TRL-TAG and NEFA, fatty acid composition. The postprandial data were expressed in summary form, i.e. area under the postprandial curve (AUC), incremental AUC, maximum postprandial concentration (Cmax) and time to maximal postprandial concentrations (Tmax) for each individual for each of the three meals. Time to minimal postprandial concentration (Cmin) was calculated for plasma NEFA concentrations. The AUC was calculated using the trapezium rule, as recommended by Matthews et al. (1990). Two-way ANOVA, using subject and meal as the independent variables, was used to investigate significant differences of these summary variables. Post hoc statistical analysis was completed using the least significant difference, which determines the criterion to identify a significant difference between group means (Snedecor & Cochran, 1989). Pearson correlation coefficients were calculated for fasting concentrations of TAG, TRL-TAG, cholesterol and NEFA with the postprandial AUC and Cmax. The level of statistical significance was set at α = 0.05 and the P values quoted are two-sided. TRL-TAG and NEFA composition data were log-transformed to give data a normal Gaussian distribution.

**Results**

Baseline characteristics of the eight participants are shown in Table 3.

Postprandial plasma triacylglycerol, triacylglycerol-rich lipoprotein-triacylglycerol, cholesterol and non-esterified fatty acid response

All three test meals elicited significant postprandial responses in plasma total TAG, TRL-TAG, cholesterol and NEFA (P < 0.05). Peak plasma TAG and TRL-TAG concentrations occurred 2 h after consumption of the test meals and returned to fasting

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**Table 1. Test meal composition (g)**

<table>
<thead>
<tr>
<th>Meal</th>
<th>PHFO</th>
<th>Lard*</th>
<th>Palm oil†</th>
</tr>
</thead>
<tbody>
<tr>
<td>Energy (kJ)</td>
<td>2596.4</td>
<td>2675.8</td>
<td>2714.04</td>
</tr>
<tr>
<td>Protein</td>
<td>17.5</td>
<td>11.3</td>
<td>11.5</td>
</tr>
<tr>
<td>Carbohydrate</td>
<td>38.1</td>
<td>24.6</td>
<td>23.9</td>
</tr>
<tr>
<td>Fat</td>
<td>45.0</td>
<td>65.5</td>
<td>64.5</td>
</tr>
<tr>
<td>SFA</td>
<td>18.9</td>
<td>27.5</td>
<td>21.2</td>
</tr>
<tr>
<td>MUFA</td>
<td>6.2</td>
<td>9.1</td>
<td>17.8</td>
</tr>
<tr>
<td>PUFA</td>
<td>2.0</td>
<td>2.9</td>
<td>3.3</td>
</tr>
<tr>
<td>Trans unsaturated fatty acids</td>
<td>15.8</td>
<td>23.0</td>
<td>0.0</td>
</tr>
<tr>
<td>Cholesterol (mg)</td>
<td>106.8</td>
<td>110.8</td>
<td>110.8</td>
</tr>
</tbody>
</table>

PHFO, partially hydrogenated fish oil; SFA, saturated fatty acids.

* Pasteurised egg-yolk powder added (2.83 g).
† Pasteurised egg-yolk powder added (4.2 g).

**Table 2. Fatty acid composition of the three test fats used (g/100 g fatty acids)**

<table>
<thead>
<tr>
<th>Meal</th>
<th>PHFO</th>
<th>Lard</th>
<th>Palm oil</th>
</tr>
</thead>
<tbody>
<tr>
<td>12:0</td>
<td>0.1</td>
<td>0.0</td>
<td>0.2</td>
</tr>
<tr>
<td>14:0</td>
<td>7.7</td>
<td>2.8</td>
<td>1.1</td>
</tr>
<tr>
<td>16:0</td>
<td>19.1</td>
<td>26.2</td>
<td>41.6</td>
</tr>
<tr>
<td>16:1cis</td>
<td>1.8</td>
<td>4.0</td>
<td>0.3</td>
</tr>
<tr>
<td>16:1trans</td>
<td>4.6</td>
<td>0.9</td>
<td>0.0</td>
</tr>
<tr>
<td>18:0</td>
<td>6.6</td>
<td>17.0</td>
<td>4.3</td>
</tr>
<tr>
<td>18:1cis</td>
<td>5.1</td>
<td>37.2</td>
<td>43.4</td>
</tr>
<tr>
<td>18:1trans</td>
<td>9.9</td>
<td>2.1</td>
<td>0.0</td>
</tr>
<tr>
<td>18:2cis</td>
<td>0.7</td>
<td>1.1</td>
<td>8.4</td>
</tr>
<tr>
<td>18:2trans</td>
<td>2.3</td>
<td>0.2</td>
<td>0.0</td>
</tr>
<tr>
<td>18:3</td>
<td>0.0</td>
<td>0.3</td>
<td>0.3</td>
</tr>
<tr>
<td>20:0</td>
<td>2.9</td>
<td>0.2</td>
<td>0.0</td>
</tr>
<tr>
<td>20:1cis</td>
<td>3.7</td>
<td>0.0</td>
<td>0.0</td>
</tr>
<tr>
<td>20:1trans</td>
<td>5.7</td>
<td>0.0</td>
<td>0.0</td>
</tr>
<tr>
<td>20:2cis</td>
<td>1.9</td>
<td>0.0</td>
<td>0.0</td>
</tr>
<tr>
<td>20:2trans</td>
<td>3.1</td>
<td>0.0</td>
<td>0.0</td>
</tr>
<tr>
<td>22:0</td>
<td>3.6</td>
<td>0.0</td>
<td>0.0</td>
</tr>
<tr>
<td>22:1cis</td>
<td>4.8</td>
<td>0.0</td>
<td>0.0</td>
</tr>
<tr>
<td>22:1trans</td>
<td>7.8</td>
<td>0.0</td>
<td>0.0</td>
</tr>
<tr>
<td>20 and C22trans PUFA†</td>
<td>7.5</td>
<td>0.0</td>
<td>0.0</td>
</tr>
<tr>
<td>15:0 + C17:0</td>
<td>1.2</td>
<td>0.0</td>
<td>0.0</td>
</tr>
</tbody>
</table>

PHFO, partially hydrogenated fish oil.
* Analysed in the study laboratory using GLC.
† Trans fatty acid isomers of C20–22 PUFA.
concentrations by 8 h (Table 4, Fig. 1 and Fig. 2). There was a marked variability in the postprandial response between individuals for TAG, TRL-TAG, cholesterol \((P<0.001)\) and NEFA \((P<0.0001)\) and also a statistically significant postprandial time effect (TAG, TRL-TAG, NEFA, \(P<0.0001\); cholesterol, \(P=0.004\)). There was no difference in the postprandial plasma TAG, TRL-TAG, cholesterol or NEFA responses following the three test meals. As fasting plasma TAG concentrations were correlated with TAG-C\(_{\text{max}}\) \((r=0.77; P<0.0001)\) and TAG-AUC \((r=0.87; P<0.0001)\), the incremental AUC was calculated. There was no difference in any of the summary variables (AUC, incremental AUC, T\(_{\text{max}}\), C\(_{\text{max}}\)) between meals (Table 5). NEFA concentrations were depressed at 2 h following all three test meals (Table 4; Fig. 3).

**Triacylglycerol-rich lipoprotein-triacylglycerol fatty acid composition**

Partially hydrogenated fish oil test meal. The following fatty acids showed a marked elevation in TRL-TAG 4 h postprandially following ingestion of the PHFO test meal; 18:0, 16:1\(trans\), and 18:1\(trans\) and returned to fasting levels 8 h postprandially. There was also a non-significant increase in 14:0, 16:0, 16:1\(cis\), 18:1\(cis\), and 20:1\(cis\) following the PHFO test meal. The fatty acid composition of the 4 h TRL-TAG samples, expressed as a percentage of total TRL-TAG fatty acids, is shown in Fig. 4. The following fatty acids were over-represented in the chylomicron fraction; 16:0, 16:1\(cis\), 18:1\(cis\) and 18:2\(cis, cis\), while 16:1\(trans\), 18:1\(trans\) and 18:2\(trans, trans\) were under-represented. Longer-chain fatty acids (C20–22) were either not present, or present in very small quantities, in the chylomicron fraction (TRL-TAG) despite their large contribution in the PHFO meal.

**Lard test meal.** A statistically significant meal x time interaction was demonstrated after the lard test meal was consumed, as 16:0, 18:0, and 18:1\(cis\) increased 4 h postprandially. In addition 14:0 and 16:1\(cis\) showed a statistically non-significant increase.

**Palm oil test meal.** After the palm oil test meal was consumed, the increase in 10:0 and 16:0 was statistically significant.

### Table 3. Age, body mass index, fasting total cholesterol, triacylglycerol, glucose and \(\gamma\)-glutamyl transferase (GT) of the study participants

<table>
<thead>
<tr>
<th>Participant</th>
<th>Age (years)</th>
<th>BMI (kg/m(^2))</th>
<th>Cholesterol (mmol/l)</th>
<th>Triacylglycerol (mmol/l)</th>
<th>Glucose (mmol/l)</th>
<th>(\gamma)-GT (U/l)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>27</td>
<td>23.25</td>
<td>4.86</td>
<td>0.98</td>
<td>3.58</td>
<td>16.0</td>
</tr>
<tr>
<td>2</td>
<td>21</td>
<td>25.18</td>
<td>4.34</td>
<td>0.67</td>
<td>5.13</td>
<td>11.0</td>
</tr>
<tr>
<td>3</td>
<td>23</td>
<td>26.69</td>
<td>5.27</td>
<td>0.97</td>
<td>6.06</td>
<td>13.0</td>
</tr>
<tr>
<td>4</td>
<td>30</td>
<td>22.46</td>
<td>4.80</td>
<td>1.21</td>
<td>5.29</td>
<td>6.0</td>
</tr>
<tr>
<td>5</td>
<td>30</td>
<td>27.33</td>
<td>5.07</td>
<td>1.50</td>
<td>6.34</td>
<td>13.0</td>
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<tr>
<td>6</td>
<td>27</td>
<td>27.90</td>
<td>4.78</td>
<td>2.07</td>
<td>4.40</td>
<td>23.5</td>
</tr>
<tr>
<td>7</td>
<td>26</td>
<td>25.60</td>
<td>3.74</td>
<td>1.30</td>
<td>5.14</td>
<td>22.0</td>
</tr>
<tr>
<td>8</td>
<td>25</td>
<td>25.14</td>
<td>5.50</td>
<td>1.99</td>
<td>4.50</td>
<td>25.0</td>
</tr>
<tr>
<td>Mean</td>
<td>26.1</td>
<td>25.7</td>
<td>4.8</td>
<td>1.33</td>
<td>5.1</td>
<td>14.9</td>
</tr>
<tr>
<td>SD</td>
<td>3.1</td>
<td>2.2</td>
<td>0.5</td>
<td>0.9</td>
<td>1.0</td>
<td>8.1</td>
</tr>
</tbody>
</table>

### Table 4. Postprandial plasma triacylglycerol (TAG), TAG-rich lipoprotein-TAG (TRL-TAG), cholesterol and non-esterified fatty acid responses to the ingestion of a partially hydrogenated fish oil (PHFO), lard or palm oil test meal by healthy volunteers

<table>
<thead>
<tr>
<th>Time after test meal (h)</th>
<th>0</th>
<th>2</th>
<th>4</th>
<th>6</th>
<th>8</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Plasma TAG (mmol/l)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PHFO</td>
<td>1.24</td>
<td>0.34</td>
<td>1.60</td>
<td>0.45</td>
<td>1.59</td>
</tr>
<tr>
<td>Lard</td>
<td>1.37</td>
<td>0.31</td>
<td>1.86</td>
<td>0.45</td>
<td>1.73</td>
</tr>
<tr>
<td>Palm oil</td>
<td>1.36</td>
<td>0.43</td>
<td>1.86</td>
<td>0.62</td>
<td>1.54</td>
</tr>
<tr>
<td><strong>TRL-TAG (mmol/l)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PHFO</td>
<td>0.51</td>
<td>0.22</td>
<td>0.87</td>
<td>0.37</td>
<td>0.76</td>
</tr>
<tr>
<td>Lard</td>
<td>0.57</td>
<td>0.24</td>
<td>1.05</td>
<td>0.41</td>
<td>0.92</td>
</tr>
<tr>
<td>Palm oil</td>
<td>0.50</td>
<td>0.30</td>
<td>0.98</td>
<td>0.51</td>
<td>0.69</td>
</tr>
<tr>
<td><strong>Cholesterol (mmol/l)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PHFO</td>
<td>4.87</td>
<td>0.59</td>
<td>4.93</td>
<td>0.61</td>
<td>4.94</td>
</tr>
<tr>
<td>Lard</td>
<td>5.11†</td>
<td>0.69</td>
<td>5.03</td>
<td>0.67</td>
<td>5.04</td>
</tr>
<tr>
<td>Palm oil</td>
<td>4.98‡</td>
<td>0.66</td>
<td>5.00</td>
<td>0.68</td>
<td>4.95</td>
</tr>
<tr>
<td><strong>NEFA (mmol/l)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PHFO</td>
<td>0.27</td>
<td>0.19</td>
<td>0.19</td>
<td>0.11</td>
<td>0.37</td>
</tr>
<tr>
<td>Lard</td>
<td>0.34</td>
<td>0.18</td>
<td>0.26</td>
<td>0.17</td>
<td>0.45</td>
</tr>
<tr>
<td>Palm oil</td>
<td>0.35</td>
<td>0.21</td>
<td>0.29</td>
<td>0.21</td>
<td>0.49</td>
</tr>
</tbody>
</table>

*Mean value was significantly different from that at fasting (time 0) \((P<0.05)\).
†Mean value was significantly different from that of the PHFO meal at the same time point \((P<0.05)\).
‡Mean value was significantly different from that of the lard meal at the same time point \((P<0.05)\).
significant 4 h postprandially; 18:1cis also increased, but this increase was not significant.

**Plasma non-esterified fatty acid composition**

The concentrations of individual fatty acids in the plasma NEFA pool at 0, 4 and 8 h following ingestions of the PHFO, lard and palm oil test meals were derived from applying concentrations (% w/w) of fatty acids to plasma NEFA concentrations (mmol/l). Significant meal × time interactions occurred after eating all three test meals.

**Partially hydrogenated fish oil test meal.** The following fatty acids were significantly increased in plasma NEFA, 4 and 8 h after the PHFO meal; 8:0, 10:0, 12:0, 14:0, 16:0, 18:0, 16:1cis, 16:1trans, 18:2cis, and 18:1cis and 18:1trans concentrations were statistically significantly increased at 8 h.

**Lard test meal.** Concentrations of 8:0, and 18:0 were significantly increased 4 and 8 h postprandially, and 16:0, 16:1cis, 18:1cis, and 18:2cis were significantly increased at 8 h.

**Palm oil test meal.** Concentrations of 16:0, and 18:2cis were significantly increased 4 and 8 h postprandially and 18:1cis increased at 8 h.

**Discussion**

Habitual fat intake and its effect on LDL, HDL and TAG concentrations have been studied extensively, including habitual TUFAs intake. However, the acute postprandial effect of TUFAs of marine origin on plasma lipids has not been widely investigated. In addition, since it has been well established that a prolonged and elevated acute postprandial response is associated with adverse metabolic events, we wanted to compare the acute postprandial lipaemic response to PHFO, which is high in TUFAs, with lard and palm oil. These fats were chosen for the test meals because of their similar solubility and because they are all

![Figure 1](https://www.cambridge.org/core/terms). **Fig. 1.** Postprandial response of total plasma triacylglycerol (TAG) for each of the test meals: partially hydrogenated fish oil (–□–), lard (–■–) and palm oil (–●–).

![Figure 2](https://www.cambridge.org/core/terms). **Fig. 2.** Postprandial response of triacylglycerol-rich lipoprotein-triacylglycerol (TRL-TAG) for each of the test meals: partially hydrogenated fish oil (–□–), lard (–■–) and palm oil (–●–).

**Table 5.** Summary variables for the postprandial response of plasma triacylglycerol (TAG), cholesterol and non-esterified fatty acids

<table>
<thead>
<tr>
<th></th>
<th>PHFO</th>
<th>Lard</th>
<th>Palm oil</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>TAG</strong></td>
<td>Mean</td>
<td>sd</td>
<td>Mean</td>
</tr>
<tr>
<td>AUC (mmol/l × 8 h)</td>
<td>11.15</td>
<td>2.98</td>
<td>12.56</td>
</tr>
<tr>
<td>IAUC (mmol/l × 8 h)</td>
<td>9.91</td>
<td>2.71</td>
<td>11.18</td>
</tr>
<tr>
<td>TAG Cmax (mmol/l)</td>
<td>1.74</td>
<td>0.56</td>
<td>1.95</td>
</tr>
<tr>
<td><strong>Cholesterol</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AUC (mmol/l × 8 h)</td>
<td>39.59</td>
<td>4.81</td>
<td>40.61</td>
</tr>
<tr>
<td>IAUC (mmol/l × 8 h)</td>
<td>34.72</td>
<td>4.23</td>
<td>35.50</td>
</tr>
<tr>
<td>Cholesterol Cmax (mmol/l)</td>
<td>7.25</td>
<td>1.04</td>
<td>5.50</td>
</tr>
<tr>
<td><strong>NEFA</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AUC (mmol/l × 8 h)</td>
<td>5.08</td>
<td>0.65</td>
<td>5.19</td>
</tr>
<tr>
<td>NEFA Cmin (mmol/l)</td>
<td>0.17</td>
<td>0.11</td>
<td>0.23</td>
</tr>
</tbody>
</table>

PHFO, partially hydrogenated fish oil; AUC, area under the curve; IAUC, incremental area under the curve; Cmax, maximum concentration; Tmax, time to reach maximum concentration; Cmin, minimum concentration.

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used by the food industry. Consumption of all three high-fat test meals resulted, as expected, in pronounced postprandial lipaemia. There was, however, no significant difference in the postprandial response in plasma TAG, TRL-TAG, total cholesterol or plasma NEFA, measured as the AUC, between the three test meals. A previous study (Shishebor, 1997) also showed no difference in the postprandial lipaemic response to PHSO compared with native soyabean oil, in healthy participants. A lower incremental AUC of TRL-TAG was shown, however, following ingestion of the PHSO meal in that study and a statistically non-significant lower TRL-TAG incremental AUC was shown following the PHFO meal in the present study. This difference could be due to preferential oxidation of TUFA, and can be supported by studies which have shown lower human tissue levels of TUFA compared with dietary intake (London et al. 1991; Cantwell et al. 2005).

We were specifically interested in examining how fatty acids specific to each test meal increased in TRL-TAG during the 8 h following consumption of each meal. The concentration of 18:0 and 18:1cis fatty acids showed marked elevations in TRL-TAG 4 h following ingestion of the lard meal, which mirrored the fatty acid composition of the meal, as 55% of the total fatty acids were from 18:0 and 18:1cis. Similarly, there was a significant increase in palmitic acid (16:0) in the TRL-TAG 4 h postprandially after the palm oil meal, as 42% of the total fatty acid composition was contributed by 16:0.

In contrast, 18:0, 16:1trans and 18:1trans fatty acids showed marked elevations in TRL-TAG 4 h after the PHFO meal even though these fatty acids provided only 21% of the total fatty acids. In addition, there was no marked elevation of the longer-chain fatty acids (C20–22, cis or trans isomers) even though they provided 40% of the total fatty acids in the PHFO meal. Long-chain fatty acids (>20 carbons) were clearly not incorporated into TRL-TAG to the same extent as fatty acids with carbon chain lengths between 16 and 18 due to decreased absorption. Clearly there was a decreased absorption of fatty acids with increasing chain length, a finding that is consistent with previous studies (Filer et al. 1969; Peters et al. 1991).

Studies in rats have indicated that the absorption of the long-chain SFA behenic acid (22:0) is highly dependent on the TAG source. The amount of 22:0 absorbed from groundnut oil (55%) was significantly greater than that absorbed from caprenin (11%); also there was reduced absorption with increasing 22:0 concentrations (3.6% fatty acids in groundnut oil and 46.6% of the fatty acids in caprenin). However, Webb & Sanders (1991) have shown that low-melting fatty acids co-ingested with 22:0 act as solvents, thereby increasing uptake of 22:0 into the mixed micellar phase. The PHFO test meal in the present study had a low concentration of 22:0 (3.6% of total fatty acids) similar to groundnut oil, but in addition had almost no low-melting fatty acids (0.1%; 12:0) compared with groundnut oil (46%; 12:0). Therefore, a low absorption of 22:0 in PHFO, similar to the absorption of behenic acid in caprenin (11%), would be expected. Whether this explanation can be extended to other

\[ \text{M. M. Cantwell et al.} \]
long-chain fatty acids (both cis and trans unsaturated geometric isomers) is questionable, although this mechanism may also explain the decreased recovery of other long-chain fatty acids in TRL-TAG following ingestion of the PHFO meal. In total long-chain fatty acids constituted only 3% of total fatty acids in the TRL-TAG 4 h postprandially, compared with 40% of total fatty acids in the PHFO test meal. The decreased absorption of long-chain fatty acids may explain why there was no difference in the postprandial TRL-TAG response shown following ingestion of a test meal which has a very high TUFAs content, a similar SFA content and a lower PUFA content compared with the palm oil and lard test meals.

In general, plasma NEFA concentrations reflected the major fatty acid constituents of the test meals, a finding that is consistent with previous studies (Frayn et al. 1996). However, the increase in long-chain fatty acids (18–22 cis and trans) following consumption of the PHFO test meal was not statistically significant. It has been estimated that almost 90% of the fatty acids found in NEFA in the late postprandial period are as a result of ‘spillover’ from lipoprotein lipase-derived fatty acids into the plasma (Frayn et al. 1997). It has also been shown that SFA and trans fatty acid isomers are preferentially incorporated into positions 1 and 3 of the TAG and PUFA at the sn-2 position (Holmer, 1998). Since lipoprotein lipase preferentially hydrolyses fatty acids at position 1 and 3 (Deckelbaum et al. 1990), it seems possible that trans fatty acids from TRL-TAG could be more rapidly hydrolysed than their cis isomers. However, in a study by Summers et al. (1999), subjects who were fed TAG with specific fatty acids enriched at positions 1 and 3 showed no specificity of lipoprotein lipase on palmitic, stearic and oleic acids. In addition, Summers et al. (2000) found no difference in adipose tissue extraction (lipoprotein lipase-mediated hydrolysis) of specific fatty acids. However, they did note that EPA (20 : 5 n-3), but not DHA (22 : 6 n-3), was under-represented in chylomicrons compared with the composition of a test meal.

In summary, there was no significant difference in the postprandial response in plasma TAG, TRL-TAG, total cholesterol or plasma NEFA, measured as the AUC, between the PHFO, lard or palm oil test meals. The postprandial TRL-TAG response to PHFO was expected to be higher than the response following lard and palm oil, as the fatty acid composition of PHFO was significantly higher in TUFAs, lower in MUFA, slightly lower in PUFA and similar in SFA content compared with lard and palm oil test meals. The absence of a higher postprandial response following ingestion of PHFO compared with lard and palm oil could be as a result of reduced absorption and increased oxidation of long-chain fatty acids (both cis and trans isomers). Although the acute postprandial effect of PHFO was no worse than the response to lard and palm oil in this group of healthy males, further studies should be carried out in other groups who could respond differently; for example, those with diabetes or heart disease.

Acknowledgements
Support from the Strategic Research and Development Fund, Dublin Institute of Technology, Kevin Street, Dublin 8, the Non-Commissioned Food Research Programme administered by the Department of Agriculture, Food and Rural Development and the National Dairy Council, Ireland, is gratefully acknowledged. We would like to thank Dr Anne-Marie Tully and Dr Enda Noone for their advice with laboratory analysis. We are grateful for the editorial assistance of the NCI CCR Fellows’ Editorial Board.

References


