# Effect of dietary fats on the Δ6- and Δ5-desaturation of fatty acids in rat liver microsomes

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1. Rats were given diets containing (% dietary energy): 46 arachis oil (AO), 36 partially-hydrogenated arachis oil (HAO)+10 AO, 36 partially-hydrogenated marine oil (HMO)+10 AO, or 46 of a combination of rape-seed oils high and low in erucic acid (RSO+LERSO).

2. In the liver microsomes the content of arachidonic acid (20:4ω6) was reduced in the groups given HAO+AO and HMO+AO.

3. The rates of Δ6-desaturation of linoleic acid into γ-linolenic acid (18:3ω6) and of Δ5-desaturation of dihomo-γ-linolenic acid into arachidonic acid were studied in vitro at two substrate levels: a high substrate level reflecting maximal microsomal desaturase activity in rat liver and a low substrate level reflecting desaturase activity under physiological conditions.

4. Dietary HAO, rich in 18:1 isomers, suppressed the Δ6-desaturase activity but not the Δ5-desaturase activity. Dietary HMO, rich in 18:1, 20:1 and 22:1 isomers, reduced both Δ6- and Δ5-desaturase activities.

The metabolic pathway for the formation of arachidonic acid is as follows:

| Δ6-Desaturation | | Elongation | | Δ5-Desaturation | |
|---|---|---|---|---|---|
| 18:2ω6 | → | 18:3ω6 | → | 20:3ω6 | → 20:4ω6 |
| Linoleic acid | | γ-Linolenic acid | | Dihomo-γ-linolenic acid | Arachidonic acid |

The rate-limiting steps are the desaturations, especially the Δ6-desaturation (Marcel *et al.* 1968, Hassam *et al.* 1975). It has recently been demonstrated that in rats given partially-hydrogenated fats, in particular fats of marine origin plus a supplement of linoleic acid, the content of arachidonic acid in liver lipids is lower than in rats given unhydrogenated fats (Hølmer *et al.* 1982). The reduced content of 20:4ω6 may result from a decreased formation of 20:4ω6 due to the presence of isomeric fatty acids in the diet. Recently, it has thus been reported that several *cis*- and *trans*-octadecenoic acids inhibit the Δ6- and Δ5-desaturases in vitro (Mahfouz *et al.* 1980, 1981) when incubated with liver microsomes obtained from rats raised on diets containing neither essential fatty acids (EFA) nor isomeric octadecenoic acids. Alternatively, the lower level of 20:4ω6 may reflect an increased catabolism of 20:4ω6 by the peroxisomal pathway induced by $C_{22:1}$ fatty acids or high-fat diets (Christiansen *et al.* 1979; Neat *et al.* 1980, 1981), whereas the mitochondrial retroconversion, i.e. chain-shortening, is not active with 20:4ω6 as substrate (Sprecher & James, 1979).

The present paper describes an experiment in which rats were maintained on diets sufficient in EFA and containing either partially-hydrogenated arachis oil (HAO), rich

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Table 1. *Fatty acids (weight % ((mg/g) × $10^{-1}$) total fatty acids) in dietary fat mixtures* determined by gas–liquid chromatography (GLC)*

| Dietary fat (% dietary energy)... | 46 AO | 36 HAO+ 10 AO | 36 HMO+ 10 AO | 46 RSO+ LERSO |
|---|---|---|---|---|
| Fatty acid | | | | |
| 14:0 | Trace | Trace | 5·9 | 0·1 |
| 14:1: *Trans* | — | — | 0·2 | — |
| *Cis* | — | — | 0·2 | — |
| 14:2: *Cis, trans* | — | — | 0·4 | — |
| 16:0 | 12·5 | 11·7 | 16·4 | 4·6 |
| 16:1: *Trans* | — | 0·2 | 3·4 | — |
| *Cis* | Trace | Trace | 1·4 | 0·3 |
| 16:2 *Cis, trans* | — | — | 0·7 | — |
| *Cis, cis* | — | — | 0·3 | — |
| 18:0 | 3·5 | 14·1 | 6·3 | 1·5 |
| 18:1: *Trans* | — | 35·9 | 8·6 | — |
| *Cis* | 38·9 | 21·4 | 11·6 | 41·9 |
| 18:2: *Cis, trans* | — | 1·9 | 1·2 | — |
| *Cis, cis* | 38·7 | 9·2 | 8·7 | 19·2 |
| 18:3 | 0·8 | 0·2 | 0·2 | 11·8 |
| 20:0 | 1·3 | 1·4 | 3·8 | 0·5 |
| 20:1: *Trans* | — | 0·7 | 7·6 | — |
| *Cis* | 1·0 | 0·5 | 3·6 | 4·3 |
| 20:2: *Cis, trans* | — | — | 1·7 | — |
| *Cis, cis* | — | — | 0·4 | 0·2 |
| 22:0 | 3·0 | 2·8 | 3·7 | Trace |
| 22:1: *Trans* | — | 0·2 | 7·5 | — |
| *Cis* | — | 0·1 | 3·6 | 15·4 |
| 22:2: *Cis, trans* | — | — | 1·3 | — |
| *Cis, cis* | — | — | 0·5 | — |
| *Trans* fatty acids by: | | | | |
| GLC | — | 38·9 | 32·6 | — |
| IR | — | 37·8 | 37·3 | — |

AO, arachis oil; HAO, partially-hydrogenated arachis oil; HMO, partially-hydrogenated marine oil; RSO, rape-seed oil; LERSO, low-erucic-acid rape-seed oil; IR, infrared spectroscopy.

* For details of diets, see p. 751.

in 18:1, or partially-hydrogenated marine oil (HMO), rich in 22:1, 20:1 and 18:1 *cis-trans* as well as positional isomeric fatty acids. The activities of Δ6-desaturase as well as Δ5-desaturase were determined in vitro at two substrate levels and correlated with the fatty acid compositions of the microsomal membranes.

## MATERIALS AND METHODS

### *Dietary fats*

The following dietary fats were used: arachis oil (AO) (peanut oil) and HAO (Aarhus Oliefabrik A/S, Denmark); HMO (Jahres Fabrikker A/S, Norway); high-erucic-acid rape-seed oil (RSO) and low-erucic-acid rape-seed oil (LERSO) (AB Karlshamn Oljefabrikker, Sweden).

### *Animal experiments*

Two animal experiments were performed. In each experiment, forty weanling male Wistar rats (Specific Pathogen-Free) (Møllegaard Laboratory, Denmark) were divided into four groups, each of ten rats, with similar average weight, and given a basal diet (Aaes-Jørgensen

Table 2. *Effect of diet on weight and food intake*
(Mean values with their standard errors)

| Dietary fat (% dietary energy)... | 46 AO | | 36 HAO+ 10 AO | | 36 HMO 10 AO | | 46 RSO+LERSO | |
|---|---|---|---|---|---|---|---|---|
| | Mean | SE | Mean | SE | Mean | SE | Mean | SE |
| Body-wt at: Start of experiment (g) | 61·2 | 1·3 | 61·2 | 0·9 | 60·3 | 1·0 | 61·2 | 1·4 |
| End of experiment (g)† | 420·3 | 11·2 | 462·2 | 7·8 | 441·3 | 16·2 | 407·9 | 14·4 |
| Body-wt gain (g) / Food intake (g)‡ | 0·20 | 0·02 | 0·25 | 0·02 | 0·19 | 0·01 | 0·16 | 0·02 |

AO, arachis oil; HAO, partially-hydrogenated arachis oil; HMO, partially-hydrogenated marine oil; RSO, rape-seed oil; LERSO, low-erucic-acid rape-seed oil.

† Significant differences (Student's *t* test): AO *v.* HAO+AO ($P < 0·005$).

‡ During 1 week of the experiment. Significant differences (Student's *t* test): HAO+AO *v.* HMO+AO ($P < 0·05$), HAO+AO *v.* RSO+LERSO ($P < 0·02$).

& Hølmer, 1969) in which the dietary fats were included instead of the corresponding weight of sucrose. The four diets contained (% dietary energy): 46 AO, 36 HAO+10 AO, 36 HMO+10 AO and 46 (RSO+LERSO) respectively (Table 1). Rats were caged in pairs at 25° and a relative humidity of 45%. Diets and water were provided *ad lib.* The rats were examined and weighed weekly. During 1 week of the experiment the rats were weighed daily and their food intake determind (Table 2). At the end of the experimental period the rats were fasted overnight before being killed by decapitation. The experimental periods were 13 or 16 weeks.

### *Microsomal Δ6- and Δ5-desaturase activities*

From each group of ten rats the livers were divided into five pools, each containing equal amounts (3 g) from two livers. The microsomal fraction was prepared as described previously (Hølmer *et al.* 1982). Protein was determined according to Lowry *et al.* (1951).

Enzyme assays were performed immediately as described previously (Larsson & Brimer, 1979; Hølmer *et al.* 1982). [1-$^{14}$C]linoleic acid and [8, 9, 11, 12, 14, 15-$^{3}$H]eicosatrienoic acid were from Amersham International, Amersham, Bucks. The maximal Δ6- and Δ5-desaturase activities, at saturating substrate level (Mahfouz *et al.* 1980, 1981), were assayed separately in volumes of 1 ml containing 2 mg microsomal protein following 16 weeks of feeding; the quantities of linoleic acid or dihomo-γ-linolenic acid added were 100 nmol (0·2 μCi) or 20 nmol (1 μCi) in 25 μl propylene glycol respectively. Following 10 weeks of feeding, the Δ6- and Δ5-desaturase activities were assayed at low exogenous substrate levels, non-saturating substrate levels, in volumes of 1·75 ml containing either 10 mg microsomal protein and 1 nmol (0·05 μCi) linoleic acid or 5 mg microsomal protein and 1 pmol (0·2 μCi) dihomo-γ-linolenic acid.

The conversion of labelled fatty acids was determined essentially as previously described (Hølmer *et al.* 1982) with the following modifications: after addition of appropriate amounts of carriers, fatty acids were separated on silica gel G plates impregnated with 100 g silver nitrate/kg, developed twice in hexane–diethyl ether (1:1, v/v). Δ5-Desaturase activity was calculated after quench correction by the external standard ratio method.

*Fatty acid analysis*

The fatty acid composition and the *trans*-fatty acid contents of the dietary fats were analysed by gas–liquid chromatography (GLC; Høy & Hølmer, 1979). The distribution between *cis*- and *trans*-fatty acids was determined by GLC on a 6·10 m × 3·18 mm o.d. stainless-steel column with 15% OV 275 on 100/120 mesh Chromosorb P (AW-DMCS) (Supelco, Inc., Bellefonte, Pa., USA) at 220° with a helium flow of 10 ml/min. The total content of *trans*-fatty acids was determined by infrared spectroscopy (IR; Hølmer & Aaes-Jørgensen, 1969). The fatty acid composition of liver microsomal lipids was analysed by GLC after saponification (Hølmer *et al.* 1982).

## RESULTS AND DISCUSSION

*Dietary fats*

The HAO+AO and the HMO+AO diets had the same contents of total *trans*-fatty acids (Table 1). In the HAO+AO the *trans*-fatty acids were primarily 18:1 acids, mainly 18:1$\omega$11 to 18:1$\omega$8 (Høy & Hølmer, 1979), whereas the HMO+AO diet had a large content of 20:1 and 22:1 isomers, mainly 20:1$\omega$13 to 20:1$\omega$5 and 22:1$\omega$13 to 22:1$\omega$6 (Lund & Hølmer, 1976). The RSO+LERSO diet contained erucic acid *cis* 22:1$\omega$9 at a level comparable to the total content of 22:1 fatty acids in HMO+AO. All diets provided adequate levels of linoleic acid to prevent EFA-deficiency.

The combinations of dietary fats used in the present experiment allowed the following comparisons: (1) effects of 18:1 isomers (group HAO+AO) *v.* 18:1+20:1+22:1 isomers (group HMO+AO),fed at the same level, when expressed as *trans*-fatty acids, (2) effects of 22:1 isomers (primarily *trans*-fatty acids) (group HMO+AO) *v.* erucic acid *cis* 22:1$\omega$9 (group RSO+LERSO), (3) effects of partially hydrogenated fats (groups HAO+AO and HMO+AO) *v.* unhydrogenated fats (group AO).

*Animal experiments*

During the experimental period the rats did not develop any gross symptoms of deficiency. The growth of the HAO+AO group was significantly higher than that of the AO group (Table 2), whereas the ratios of growth: food consumption were not significantly different from that of the AO group.

*Microsomal lipids*

In the total lipids of the microsomal fractions (Table 3) the contents of 20:4$\omega$6 and its conversion product 22:5$\omega$6 decreased in the group given HAO+AO, and especially in the HMO+AO group relative to the AO group. This finding is in agreement with previous results for rat liver mitochondria (Hølmer *et al.* 1982), although the changes observed in microsomes were substantially smaller. In the HMO+AO group, 18:2$\omega$6 and 20:3$\omega$6 accumulated, whereas in the HAO+AO group only 20:3$\omega$6 was increased relative to the AO group. The microsomal fatty acid profiles reflected the in vivo fatty acid metabolism, during which the dietary isomeric fatty acids were present as free fatty acids or CoA esters to exert inhibitory action on the desaturases. Thus, HMO disturbed the formation of 20:4$\omega$6 more than HAO, probably due to the content of 20:1 and 22:1 isomers. This difference was not exerted by incorporation of 20:1 and 22:1 into the microsomal membrane (Table 3), nor did it result from the total content of isomers incorporated into the membrane lipids, expressed as the *trans*-fatty acid content.

A similar decrease in the conversion of 18:2$\omega$6 to 20:4$\omega$6 was observed in the RSO+LERSO group, due to the presence of 18:3$\omega$3 in the diet (Brenner, 1974). This decrease was balanced by an increased amount of $\omega$3 fatty acids.

Table 3. *Fatty acids (weight % ((mg/g) × $10^{-1}$) total fatty acids) in liver microsomal lipids*
(Mean values with their standard errors for four to five microsomal pools)

| Dietary fat (% dietary energy)... | 46 AO | | 36 HAO+ 10 AO | | 36 HMO+ 10 AO | | 46 RSO+ LERSO | |
|---|---|---|---|---|---|---|---|---|
| Fatty acid | Mean | SE | Mean | SE | Mean | SE | Mean | SE |
| 14:0 | Trace | — | Trace | — | 0·2 | 0·1 | 0·2 | 0·1 |
| 16:0 | 15·6 | 0·3 | 11·5 | 0·6 | 14·3 | 0·7 | 16·4 | 0·7 |
| 16:1: *Trans* | — | — | 0·7 | 0·1 | 1·3 | 0·1 | — | — |
| *Cis* | 0·3 | 0·1 | 0·5 | 0·1 | 1·0 | 0·1 | 0·7 | 0·1 |
| 16:2 | 0·2 | 0·1 | Trace | — | 0·3 | 0·1 | 0·3 | 0·1 |
| 18:0 | 27·3 | 0·4 | 19·7 | 0·9 | 19·3 | 0·7 | 26·9 | 0·6 |
| 18:1: *Trans* | — | — | 13·6 | 0·5 | 9·0 | 0·4 | — | — |
| *Cis* | 7·7 | 0·1 | 9·4 | 0·3 | 8·7 | 0·4 | 11·7 | 0·4 |
| 18:2: *Cis, trans* | — | — | 0·4 | 0·1 | 0·6 | 0·1 | — | — |
| *Cis, cis* | 8·3 | 0·2 | 7·1 | 0·2 | 11·2 | 0·4 | 8·3 | 0·3 |
| 18:3ω6 | 0·2 | 0·1 | Trace | — | 0·3 | 0·1 | Trace | 0·1 |
| 18:3ω3 | Trace | — | 0·2 | 0·1 | Trace | — | 0·6 | 0·1 |
| 20:1 | 0·3 | 0·1 | 0·3 | 0·1 | 0·7 | 0·1 | 1·0 | 0·1 |
| 20:3ω9 | Trace | — | 0·6 | 0·1 | 0·7 | 0·1 | Trace | — |
| 20:3ω6 | 0·2 | 0·1 | 0·7 | 0·1 | 1·4 | 0·1 | 0·6 | 0·1 |
| 20:4ω6† | 32·4 | 0·3 | 29·6 | 0·9 | 26·6 | 0·9 | 25·3 | 0·6 |
| 22:1 | — | — | Trace | — | Trace | — | 0·3 | 0·1 |
| 22:4ω6 | 0·8 | 0·1 | 0·4 | 0·1 | 0·2 | 0·1 | Trace | — |
| 22:5ω6 | 3·1 | 0·1 | 1·8 | 0·2 | 1·2 | 0·1 | Trace | — |
| 22:5ω3 | 0·2 | 0·1 | Trace | — | 0·2 | 0·1 | 0·9 | 0·1 |
| 22:6ω3 | 2·9 | 0·2 | 2·2 | 0·1 | 1·4 | 0·2 | 5·8 | 0·3 |
| 24:0 | 0·2 | 0·1 | 0·2 | 0·1 | Trace | — | Trace | — |
| *Trans*-fatty acids | — | — | 14·8‡ | 0·5 | 11·0 | 0·4 | — | — |

AO, arachis oil; HAO, partially-hydrogenated arachis oil; HMO, partially-hydrogenated marine oil; RSO, rape-seed oil; LERSO, low-erucic-acid rape-seed oil.

† Group mean values were significantly different ($P < 0·001$) as tested by one-way analysis of variance. Significant differences (Student's $t$ test): AO *v.* HAO+AO ($P < 0·05$), AO *v.* HMO+AO ($P < 0·001$), AO *v.* RSO+LERSO ($P < 0·001$), HAO+AO *v.* RSO+LERSO ($P < 0·01$).

‡ Primarily *trans* 18:1.

### Δ6- *and* Δ5-*desaturase activities*

Rat liver microsomal Δ6- and Δ5-desaturase activities were measured in vitro at two substrate levels (Tables 4 and 5). In these incubations the labelled substrates were diluted with microsomal lipids containing approximately 5% free fatty acids (Peluffo *et al.* 1976). Therefore, the conversions of substrates into products were expressed as percentage conversion instead of nmol products formed, as used conventionally.

The desaturations measured involve several reactions: fatty acid activation, desaturation and esterification. Since these reactions depend on the substrate levels, the incubations performed at the two substrate levels will be considered separately. At saturating substrate level (Table 4), the maximal desaturase activity was measured. The Δ6-desaturase activity was lower in the HAO+AO group and in the HMO+AO group compared with the AO group, whereas the activity in the RSO+LERSO group was similar to that of the AO group. The Δ5-desaturase activity was reduced in the HMO+AO group but not in the HAO+AO group. At non-saturating substrate level (Table 5) the conversion of the endogenous microsomal pool of fatty acids was measured (Pollard *et al.* 1980). Also under these circumstances the Δ6-desaturase activity was lower in the HAO+AO group and in the HMO+AO group compared with the AO group. The Δ5-desaturase activity was only

Table 4. *Liver microsomal desaturase activities (% conversion of substrate/2 mg protein per 20 min) at saturating substrate level*

(Mean values with their standard errors for four to five microsomal pools)

| Dietary fat (% dietary energy)... | 46 AO | | 36 HAO+ 10 AO | | 36 HMO+ 10 AO | | 46 RSO+ LERSO | |
|---|---|---|---|---|---|---|---|---|
| | Mean | SE | Mean | SE | Mean | SE | Mean | SE |
| Δ6-Desaturase | 5·06 | 0·15 | 3·04 | 0·40 | 3·64 | 0·27 | 6·14 | 0·16 |
| Δ5-Desaturase | 48·3 | 2·6 | 45·8 | 2·6 | 33·9 | 2·1 | 45·4 | 2·5 |

AO, arachis oil; HAO, partially-hydrogenated arachis oil; HMO, partially-hydrogenated marine oil; RSO, rape-seed oil; LERSO, low-erucic-acid rape-seed oil.

Group mean values were significantly different (Δ6-desaturase, $P < 0·001$; Δ5-desaturase, $P < 0·01$) as tested by one-way analysis of variance.

Significant differences (Student's *t* test), Δ6-desaturase: AO *v.* HAO+AO ($P < 0·01$), AO *v.* HMO+AO ($P < 0·01$), AO *v.* RSO+LERSO ($P < 0·01$), HAO+AO *v.* RSO+LERSO ($P < 0·001$), HMO+AO *v.* RSO+LERSO ($P < 0·001$); Δ5-desaturase: AO *v.* HMO+AO ($P < 0·01$), HAO+AO *v.* HMO+AO ($P < 0·01$), HMO+AO *v.* RSO+LERSO ($P < 0·01$).

Table 5. *Liver microsomal desaturase activities (% conversion of substrate/10 mg protein per 20 min for Δ6-desaturase; % conversion of substrate/5 mg protein per 20 min for Δ5-desaturase) at non-saturating substrate levels*

(Mean values with their standard errors for four to five microsomal pools)

| Dietary fat (% dietary energy)... | 46 AO | | 36 HAO+ 10 AO | | 36 HMO+ 10 AO | | 46 RSO+ LERSO | |
|---|---|---|---|---|---|---|---|---|
| | Mean | SE | Mean | SE | Mean | SE | Mean | SE |
| Δ6-Desaturase | 4·78 | 0·50 | 3·24 | 0·32 | 2·07 | 0·35 | 4·07 | 0·23 |
| Δ5-Desaturase | 57·7 | 4·3 | 51·2 | 3·6 | 36·6 | 4·0 | 50·6 | 5·7 |

AO, arachis oil; HAO, partially-hydrogenated arachis oil; HMO, partially-hydrogenated marine oil; RSO, rape-seed oil; LERSO, low-erucic-acid rape-seed oil.

Group mean values were significantly different (Δ6-desaturase, $P < 0·001$; Δ5-desaturase, $P < 0·025$) as tested by one-way analysis of variance.

Significant differences (Student's *t* test), Δ6-desaturase: AO *v.* HAO+AO ($P < 0·05$), AO *v.* HMO+AO ($P < 0·01$), HAO+AO *v.* HMO+AO ($P < 0·05$), HMO+AO *v.* RSO+LERSO ($P < 0·01$); Δ5-desaturase: AO *v.* HMO+AO ($P < 0·01$), HAO+AO *v.* HMO+AO ($P < 0·05$).

reduced in the HMO+AO group, but not in the HAO+AO group, relative to the reference group (AO).

The observed desaturase activities were in general agreement with the fatty acid profiles of the microsomal lipids (Table 3), except for the RSO+LERSO group, in which the microsomal level of 20:4ω6 was reduced, whereas the desaturase activities were increased compared with the AO group.

Reduced conversions of polyunsaturated fatty acids in the microsomal membrane may arise from an inhibitory effect of other polyunsaturated fatty acids present (Brenner, 1974). It has also been demonstrated in vitro that *cis*- and *trans*-18:1 fatty acids inhibit the Δ6- and Δ5-desaturases of EFA-deficient rats (Mahfouz *et al.* 1980, 1981) at incubation conditions comparable to the saturating substrate levels used in our experiment. The diets used for the rats in the experiments by Mahfouz *et al.* (1980, 1981) did not contain isomeric

fatty acids. It has also been shown (Hill *et al.* 1982) that marginally EFA-deficient rats given partially-hydrogenated soya-bean oil rich in 18:1 isomers have depressed Δ6-desaturase activity but not depressed Δ5-desaturase activity, compared with rats given either fully-hydrogenated fats or low-fat diets. The effects of EFA status on the activity of Δ6-desaturase have been debated. Castuma *et al.* (1972) found that EFA deficiency increased the Δ6-desaturase activity, whereas Sprecher & James (1979) found no effect. No previous publications have mentioned the effects of long-chain isomeric fatty acids of partially-hydrogenated marine oils demonstrated in the present paper.

In addition to a competitive effect on desaturation, it is possible that the incorporation of isomeric fatty acids into the microsomal membranes *per se*, evidenced by the *trans* contents (Table 3), affects the activities of the enzymes. It has been found that desaturase activity depends on a lipid environment (Jeffcoat, 1979), and a reduced activity of mitochondrial respiratory enzymes following intake of *trans* fatty acids has been reported (Svensson, 1982).

Feeding *trans*, *trans* 18:2 at high levels to rats reduced the formation of liver 20:4ω6 (Anderson *et al.* 1975) in vivo, and diets containing *trans* 18:1 + *trans*, *trans* 18:2 + *cis*, *trans* 18:2 depressed the Δ6-desaturase activity in vitro (de Schrijver & Privett, 1982) which was attributed to the incorporation of 3% *trans*, *trans* 18:2 in the microsomal membrane. No accumulation of 20:3ω6 was observed by these authors, indicating that primarily the Δ6-desaturase was affected. In agreement with this, Mahfouz (1981) found that intake of marginally EFA-deficient diets containing $C_{18}$-*trans* fatty acids was followed by a deposition of several *trans* 18:1 isomers as well as *trans*, *trans* 18:2 isomers in the microsomal membrane and a lowered Δ6-desaturase activity.

The Δ5-desaturase activity of the HAO + AO group was unaffected, in agreement with Hill *et al.* (1982). Therefore, the reduction of Δ5-desaturase activity in the microsomes from the HMO + AO groups points to a special effect of the dietary HMO. In vivo, dietary 20:1 and 22:1 isomers may influence the desaturase activities as reflected in the fatty acid profile of the microsomes. In vitro, at saturating substrate levels, the concentrations of 20:1 and 22:1 were very low, however, due to the procedure for preparation of microsomes as evidenced by the fatty acid composition of the microsomes (Table 3). Therefore, the in vitro retardation of Δ5-desaturation probably resulted from the changed fatty acid profile of the microsomal membranes of HMO + AO-fed rats.

In the RSO + LERSO group, the maximal Δ6-desaturase activity was increased relative to the AO group. This may reflect an induction of Δ6-desaturation in order to overcome the competitive inhibitory effect of 18:3ω3 (Brenner & Peluffo, 1966; Pollard *et al.* 1980). Furthermore, this effect may be accentuated, since neither 18:3ω3 nor 22:1ω9 from RSO + LERSO were present to any significant extent in the microsomal membrane to exert influence on the desaturation, contrary to the *trans* fatty acids from HAO and HMO that were significantly incorporated into the microsomal membrane.

From the experiments described in the present paper it appears that partially-hydrogenated oils given at high levels (36% dietary energy), even with a supply of 4·2% dietary energy as 18:2, can affect the fatty acid composition of the tissue lipids through inhibitory effects on the microsomal Δ6- and Δ5-desaturases. These effects are more pronounced with partially-hydrogenated marine oils than with partially-hydrogenated vegetable oils, pointing to a specific influence of $C_{20:1}$ and $C_{22:1}$ isomeric fatty acids. This agrees with the previous observation that HMO and HAO aggravate EFA-deficiency in the rat (Aaes-Jørgensen & Hølmer, 1969), HMO being the most potent aggravator. In human consumption, these effects may not be observed due to the normally much lower intake of partially-hydrogenated oils in proportion to EFA.

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