lar species to be very abundant, especially in phosphatidyl-
ethanolamine (PE) and phosphatidylserine (PS).

A characteristic of marine food chains is that C16 and C18
polyunsaturated fatty acids (PUFA) are synthesized by
phytoplankton and these are then elongated to C20 PUFA in
the zooplankton and finally to C22 PUFA in fish. Thus EPA
can be expected to be more important in animals from lower
trophic levels. Neutral tissue from a marine crustacean was
therefore examined to find whether the main PUFA was EPA
or DHA.

Pandalus borealis was collected from Isfjord, Svalbard, in
September 1989. The eyes from 17 animals were placed in 4
ml of chloroform–methanol (2:1 by vol.) and stored at
−20°C under N2, until used. Lipid was then extracted by
the method of Folch et al. [6].

The lipid class composition was determined by high-
performance t.l.c. using the solvent system of [7], followed by
charring and densitometry. The phospholipid class composi-
tion was 4.8% sphingomyelin, 33.6% phosphatidylcholine
(PC), 6.0% PS, 4.2% phosphatidylinositol (PI), 4.3% cardio-
ine alcohol (FAA) found in the four phospholipid classes, but
from neural tissue of a marine crustacean therefore contain
the very low amount of EPA and are markedly different from those of
vertebrates.

We are very grateful to Dr Andrew Clarke, British Antarctic
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Comparative effects of n-9, n-6 and n-3 dietary fatty acids on mammary tumour incidence and membrane phospholipid fatty acid compositions

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Experimental diets high in fat have been shown to enhance
mammary tumorigenesis in spontaneous, carcinogen-

induced and transplantable mammary tumour models in rats
and mice [1]. The type of fat fed also influences mammary tumour incidence; Ip [2] has shown that tumour incidence is
linearly related to dietary linoleate (C18:2, n-6) content, up to a
maximum level which lies between 4 and 5%. More recent
studies have shown protective effects of long-chain poly-
unsaturated fatty acids of the n-3 class, found in high con-
centrations in fish oils. Protective effects operate at low-dose
levels [3] and remain even when the total fat content of the

Table 1. Fatty acid composition of the diacyl classes of phospho-

lipids from the eyes of Pandalus borealis

<table>
<thead>
<tr>
<th>Fatty acid composition (wt. %)</th>
</tr>
</thead>
<tbody>
<tr>
<td>PC</td>
</tr>
<tr>
<td>------</td>
</tr>
<tr>
<td>14:0</td>
</tr>
<tr>
<td>16:0</td>
</tr>
<tr>
<td>16:1</td>
</tr>
<tr>
<td>17:0</td>
</tr>
<tr>
<td>18:1</td>
</tr>
<tr>
<td>18:1</td>
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<tr>
<td>18:1</td>
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<tr>
<td>18:2</td>
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<tr>
<td>18:3</td>
</tr>
<tr>
<td>18:4</td>
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<td>20:1</td>
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<td>20:1</td>
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<td>20:4</td>
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<td>20:4</td>
</tr>
<tr>
<td>20:5</td>
</tr>
<tr>
<td>22:5</td>
</tr>
<tr>
<td>22:6</td>
</tr>
<tr>
<td>Total saturates</td>
</tr>
<tr>
<td>Total monoenes</td>
</tr>
<tr>
<td>Total n-3 PUFA</td>
</tr>
<tr>
<td>Total n-6 PUFA</td>
</tr>
</tbody>
</table>

Abbreviations used: PE, PI, PC, PS, PE, and PI. choline, ethanolamine and inositol phospholipid, respectively.
Fish-rich diets have been shown to reduce fasting triacylglycerol concentrations, found in high concentrations in fish and fish-oil fatty acid composition [6].

The phosphoinositide fatty acid composition of the platelet membrane has been shown to be resistant to alterations in dietary fatty acid intake [7, 8]. No studies have been undertaken to determine the susceptibility of mammary epithelial membrane phosphoinositide fatty acid composition to altered fatty acid intake. In the present study, tumour incidence and growth rates of carcinogen-treated animals, and the fatty acid compositions of mammary membrane choline (PC), ethanolamine (PE) and inositol (PI) phospholipids, of tissue from control and carcinogen-treated animals, have been studied in corn-oil, olive-oil and MaxEPA (fish-oil)-fed animals.

Twenty-four, 30-day-old female Sprague-Dawley rats weighing approximately 80 g were treated with the carcinogen ENU (ethylnitrosourea; 5 mg of ENU administered by gastric gavage). Treated animals were randomly assigned to corn-oil, olive-oil or MaxEPA dietary groups. Twenty-four control (non-ENU-treated) animals were also randomly assigned to each of the three dietary groups. Food intake and body weight were measured weekly and food pots changed daily. Animals in the ENU-treated group were killed when palpable mammary tumours could be felt at weekly examinations (between 16 and 20 weeks post-carcinogen). A control animal from the same dietary group was killed at the same time as the tumour-bearing animal.

At 20 weeks after carcinogen administration, tumour incidence rates were significantly lower in MaxEPA (7.19 ± 0.25; P < 0.05) -fed animals compared with corn-oil (13.27 ± 0.57; P < 0.05) -fed animals. Docosahexaenoic acid (C22:6, ω-3) was higher in MaxEPA (4.39 ± 0.66) than corn-oil (2.19 ± 0.25; P < 0.01) and olive-oil (2.63 ± 0.35; P < 0.05) -fed animals. Similar, but less marked, differences were found in the PE fraction of MaxEPA-fed animals, but no significant differences in phosphoinositide fatty acid composition were found between any of the three dietary groups. In tumour tissue no differences in the fatty acid composition of any of the phospholipid classes were found in animals fed diets of different fatty acid composition.

This study has confirmed the protective effects of n-3 fatty acids against mammary tumour development in carcinogen-treated animals. Failure to detect differences in phosphoinositide fatty acid composition of normal mammary tissue obtained from animals fed diets of different fatty acid composition suggests that the composition of this membrane component is resistant to dietary fatty acid modification. The findings do not support the suggestion that dietary fatty acid-induced changes in phosphoinositide fatty acid composition underlie enhanced mammogenic responses to exogenous hormones and enhanced tumour incidence rates observed in animals fed high fat diets.


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Fasting and postprandial triacylglycerol responses to a standard test meal in subjects taking dietary supplements of n-3 fatty acids

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Dietary supplementation with fish-oils and consumption of fish-rich diets have been shown to reduce fasting triacylglycerol concentrations in normal subjects and patients with hypertriglyceridaemia [1–3]. The hypotriglyceridaemic properties of fish-oils have been attributed to the effects of the n-3 fatty acids, eicosapentaenoic acid and docosahexaenoic acid, found in high concentrations in fish and fish-oils. Mechanisms proposed to explain the lipid lowering effects of n-3 fatty acids include reduced rates of hepatic very-low-density lipoprotein (VLDL) output [4], and increased rates of endogenous and exogenous triacylglycerol clearance [5]. Hypertriglyceridaemia is commonly seen in patients with NIDDM (non-insulin-dependent diabetes mellitus) and has been attributed to increased hepatic VLDL synthesis secondary to raised circulating concentrations of both insulin and free fatty acids [6]. It might, therefore, be expected that fish-oils would have particularly beneficial effects in this group of patients. Schectman et al. [7] found lower fasting plasma triglyceride concentrations in NIDDM patients following a 4 week period of dietary fish-oil supplementation, whereas Kasim et al. [8], in a similar group of patients, found no effect of fish-oil supplementation on fasting serum triglyceride levels. Furthermore, both

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