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. Schechtman 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

**Abbreviations used:** VLDL, very-low-density lipoprotein; NIDDM, non-insulin-dependent diabetes mellitus; LDL, HDL, low- and high-density lipoprotein, respectively.

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studies reported potentially detrimental effects of fish-oil supplements in this group of patients, since fasting low-density lipoprotein (LDL) cholesterol, apo-B and glucose concentrations were raised after the period of fish-oil supplementation. Effects of dietary supplementation with fish-oils on postprandial triacylglycerol concentrations have not been extensively studied in normal subjects and there are no reports in the literature of postprandial triacylglycerol concentrations in diabetic subjects taking fish-oil supplements.

In the present study, fasting total and high-density lipoprotein (HDL) cholesterol concentrations were determined in normal subjects (n = 7), and in subjects with NIDDM (n = 8), before and 6 weeks after supplementation with fish-oil capsules (9 capsules/day providing 2.7 g of n-3 fatty acids). Fasting and postprandial triacylglycerol concentrations were determined after a standard test meal, before and 6 weeks after the period of fish-oil supplementation. Diabetic subjects being treated for their condition by oral hypoglycaemic agents and diet, or by diet alone, were included in the study. None was suffering gross hyperlipidaemia, liver disease or other medical condition and none was taking drugs likely to influence the outcome of the experiment. Normal subjects were recruited from healthy volunteers none of whom were taking medication or were using oral contraceptives during the period of study.

At each visit, subjects attended after an overnight fast and consumed a standard mixed test meal (1000 kcal, 36 g of protein, 53 g of fat and 100 g of carbohydrate). Subjects consumed the meal within 15 min; blood samples were taken at time zero and at 15 minute intervals for the first hour, and half-hourly intervals for the following 2.5 h. Determinations of total and HDL cholesterol and triacylglycerol concentrations were made on serum samples stored at -20°C, and analyses were carried out within 6 weeks of collection.

The results shown in Table 1 demonstrate that fish-oil supplementation significantly lowered fasting and postprandial triacylglycerol concentrations in normal subjects, but not in diabetic subjects, in whom there was a non-significant increase in postprandial triacylglycerol concentrations at the second (post fish-oil), test meal. Total cholesterol concentrations were significantly elevated in the diabetic, but not in the normal subjects. Recent kinetic studies of VLDL and LDL metabolism in miniature pigs have shown that n-3 fatty acid feeding increases the conversion of VLDL to LDL [9], and this may explain higher total cholesterol concentrations in diabetic subjects, in whom other factors regulating LDL cholesterol concentrations may be abnormal. Failure to observe triacylglycerol lowering effects of n-3 fatty acids in subjects with NIDDM suggests that the mechanism involved in n-3 fatty acid-induced hypotriglyceridaemia may be defective in this condition.


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