Habitual Fish Consumption, Serum Lipids and Platelet Function

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Recently, in the Dutch town of Zutphen, the relation between the habitual fish consumption and the mortality from coronary heart disease (CHD) was investigated (1). Information about the fish consumption of 852 middle-aged men, who were free of coronary heart disease, was collected in 1960, 1965, 1970, and 1985 by the cross-check dietary history method (2). The average fish consumption was 20 g/day. Between 1960 and 1980 seventy-eight men died from CHD. Fish consumption related inversely to 20 year mortality from CHD after adjustment for other risk factors (p < 0.05). The risk ratio's of death from CHD were about 2.5 times lower among men who consumed more than 30 g of fish per day as compared to those who did not eat fish (FIG. A-14-1).

The relation between fish consumption and mortality from CHD may be explained by an effect of dietary fish on lipid metabolism and/or on changes in platelet functions, resulting from an altered fatty acid profile and prostanoid formation (3). We tested this hypothesis in a subgroup of the original study population.
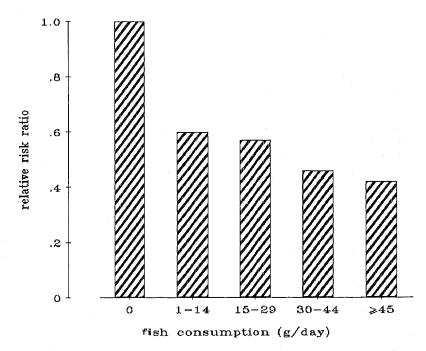
FIG. A-14-1. Relative risk ratios of death from CHD to fish consumption in 1960, among 852 middle-aged men, during the 20 year follow up period.

Methods

Sixty four healthy men were selected on the basis of their habitual fish consumption. During the period between 1960-1985 they always consumed more than 13 g of fish per day (mean 31.3 g; fish group) or less than 13 g of fish per day (mean 3.8 g; control group).

Early 1986 fish consumption was estimated again. On that occasion fasting blood samples were collected for the measurement of serum lipids and lipoproteins and for determination of the fatty acid composition of serum phospholipids. Moreover, bleeding times were measured (Simplate II method) and another blood sample was taken for the measurement of collagen-induced platelet aggregation (Impedance method) and adenosine triphosphate (ATP) release (luminiscence technique) in citrated whole blood. At the end of each measurement, plasma was collected and stored at −20 C. Finally, a venous blood sample (5 ml) was allowed to clot for one hour at 37 C. Serum was collected and stored

at -20 C. In these plasma and serum samples thromboxane B_2 (TxB_2) concentrations were measured by radio immuno assay (RIA).

Results and Discussion

Due to an inconsistent fish consumption during the last year, 24 volunteers had to be removed from the study. From the remaining 40 men the mean fish consumption over the last year was 2.3 $\pm$ 1.0 g/day for the control group (n = 15) and 31.5 $\pm$ 2.7 g/day for the fish group (n = 25) (p < 0.0001). This difference was also reflected by significant differences in the concentrations of timnodonic acid (20:5 n-3) and cervonic acid (22:6 n-3) in serum phospholipids.

For timnodonic and cervonic acid the relationships between the concentration in serum phospholipids and the dietary uptake was less pronounced than for linoleic acid. This indicates that the methods to assess the fatty acid intake are less reliable for (n-3)polyenes than for (n-6)polyenes.

Significant differences between the fish and the control group were observed for total triglyceride and intermediate density lipoprotein (IDL)-triglyceride (p < 0.05). No significant differences were found for the cholesterol and triglyceride contents of the other lipoproteins, nor for the apoproteins AI, AII, and B, and for Lechitin cholesterol acyltransferase and Cholesterol ester transfer protein.

Between both groups no differences were observed in platelet number and cutaneous bleeding time. Collagen induced platelet aggregation and ATP release in whole blood did not differ significantly either. TxB_2 concentrations were not significantly different between both groups neither in plasma of collagen-activated blood, nor in serum.

These data indicate that the inverse relationship between fish consumption and coronary heart disease mortality, as observed in the Dutch town of Zutphen is unlikely to be explained by an effect on function of blood platelets. The causal role of certain changes in lipoprotein metabolism needs further evaluation.

References

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