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Original article

The effects of herring-roe lyophilized powder on lipid metabolism

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ABSTRACT

Herring-roe, which contains large amounts of docosahexaenoic acid and eicosapentaenoic acid, has anti-dyslipidemia effects. Here, we evaluated the effects of herring-roe on lipid metabolism in 33 adult subjects in a randomized, double-blind, placebo-controlled study. We divided the subjects into a test group that ingested herring-roe lyophilized powder (herring-roe powder) and a placebo group that ingested non-herring-roe powder, with each member of each group ingesting 15 g daily for 8 weeks. Hematological tests and body composition measurements were performed before and after 4, 6, and 8 weeks of the study period. Although no significant differences in low density lipoprotein were observed, high density lipoprotein was found to be increased in subjects who ingested herring-roe powder. In addition, the level of free fatty acid was significantly improved in the herring-roe powder group. These results suggest that ingestion of herring-roe could influence lipid metabolism.

1. Introduction

Over the past few decades, the prevalence of dyslipidemia has markedly increased worldwide, particularly in wealthy industrialized countries such as Japan.[1] Most large-scale and long-term cohort studies over the past 5–20 years have indicated that a diet rich in animal fat was associated with higher all-cause mortality.[2] High-fat westernized diets have been implicated in the increasing prevalence of dyslipidemia, a risk factor of atherosclerosis.[3] Thus, it is important to investigate the utility of functional foods and the bioactive components of food to improve and prevent dyslipidemia.

Fish oil contains rich polyunsaturated fatty acids (PUFAs), particularly eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA).[4] EPA and DHA have a number of reported health benefits in humans, such as decreasing blood triglyceride concentrations in hypertriglycemic patients and providing protective effects against cardiovascular diseases.[5] This has led to recommendations from health agencies worldwide to increase dietary intake of these fatty acids.[6] The mechanism of DHA and EPA for improvement of lipid metabolism is the downregulation of the mature form of Sterol Regulatory Element-Binding Proteins (SREBP)-1 by decreasing SREBP-1c mRNA expression, with corresponding decreases of mRNAs of cholesterologenic and lipogenic enzymes.[7] In addition, DHA and EPA facilitate β-oxidation of fatty acid.[8] The Ministry of Health, Labour and Welfare recommended that adult intakes more than 1 g of DHA and EPA per day; however, dietary intake of DHA and EPA was not sufficient in Japanese people, possibly because of decreased fish intake in Japan. Thus, it is important to research functional foods to supplement DHA and EPA intake.

Herring-roe lipids contain a large amount of EPA and DHA. In addition, DHA and EPA are the major molecular constituents in the phosphatidylcholine of herring-roe.[9] Therefore, DHA and EPA contained in herring-roe is stable and difficult to oxidize. Moreover, herring-roe contains little cholesterol compared with that of other fish roe (260 mg cholesterol per 100 g herring-roe).

These findings suggest that herring-roe, which is rich in DHA and EPA and phosphatidylcholine-contained DHA and EPA, improves lipid metabolism. However, the clinical studies assessing herring-roe are very few. Here, we evaluated whether the ingestion of the herring-roe powder can improve dyslipidemia in an adult population.

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2. Methods

2.1. Test meal preparation and ingestion method

The composition of herring-roe powder used in this study is given in Table 1. Herring-roe was fished in Sitka, Alaska, USA. The test meal was prepared by freeze-drying in a conventional method. The production and packing was performed at Ihara Suisan Co., Ltd. (FM96883/ISO9001 certification).

The subjects were instructed to ingest 15 g per day of herring-roe lyophilized powder (herring-roe powder) (containing DHA 540 mg and EPA 300 mg) or a placebo powder (11 g of unpolished rice powder containing 4 g of soybean curd used to imitate the texture and appearance of herring-roe powder) in two parts per day.

2.2. Study subjects

Thirty-three volunteers (6 males and 27 females, age 40–67 years) were enrolled in this study. None had a recent history of gastrointestinal disorders, pregnancy, significant disease, surgery, severe allergic reaction to food, or current use of any medication, including anti-hyperlipidemia medication. The subjects' age, body weight, height, body mass index (BMI), and body fat percentage are listed in Table 2.

The clinical intervention was conducted as a double-blind, placebo-controlled trial. At randomization, the 33 eligible subjects were blindly assigned to one of two groups: the test group who ingested herring-roe powder and the placebo group who ingested the placebo powder. The time schedule of this clinical study is shown in Fig. 1.

We performed hematological examinations and body composition (body weight, BMI, and body fat rate) measurements at the baseline (0 week) and post-intervention (4, 6, and 8 weeks) for the two groups. The hematological examinations were consigned to Sapporo Clinical Laboratory Inc. (Sapporo, Japan). Leptin and adiponectin was measured by the Human Leptin Quantikine ELISA Kit and the Human Total Adiponectin/Acrp30 Quantikine ELISA Kit (R&D Systems, Minneapolis, MN, USA). The subjects' body composition and blood pressure were measured with an In-Body device (Biospace Co., Tokyo) and an Omron HEM-780 automatic blood pressure monitor (Omron Corp., Kyoto, Japan).

All subjects provided written informed consent prior to undergoing any study-related tests, and the protocol was approved by the Ethics Committee of Hokkaido Information University (a certificate number; 2014-01). The study protocol conformed to the Helsinki Declaration and registered at the UMIN Clinical Trial Registration System (a certificate number; UMIN000017072).

2.3. Statistical analysis

The average and standard deviation of age and other parameters were calculated for each group. The changes in the values of various parameters were analyzed by Student's *t*-test. Statistical analyses were performed with the program IBM SPSS Statistic 19 (IBM, Armonk, NY). *P* values less than 0.05 were considered significant.

Table 1
Composition of herring-roe powder compared with placebo powder per 15 g.

Component	Herring-roe	Placebo
Calories (kcal)	70.2	62.6
Water (g)	0.11	0.26
Proteins (g)	12.3	1.73
Lipids (g)	2.30	0.93
Carbohydrates (g)	0.09	11.8
Ash (g)	0.23	0.29
Sodium (mg)	29.6	2.06
Docosahexaenoic acid (mg)	540	–
Eicosapentaenoic acid (mg)	300	–

Table 2
Characteristics of the subjects in the placebo and herring-roe powder intake groups.

Characteristic	Herring-roe	Placebo	*P*-value
Number of subjects	n = 17	n = 16	–
Number of males (male %)	4 (76.47%)	2 (87.50%)	0.656
Age (years)	51.71 ± 9.25	53.19 ± 9.31	0.650
Height (cm)	158.43 ± 7.93	159.8 ± 5.00	0.560
Body weight (kg)	52.39 ± 9.74	58.93 ± 10.34	0.071
BMI (kg/m^2)	20.73 ± 2.08	23.04 ± 3.69	0.033
Body fat percentage (%)	25.21 ± 5.42	30.18 ± 7.57	0.037

Values shown are mean ± standard deviation (SD). Statistical analysis was performed by analysis of variance (ANOVA) for age, height, body weight, and BMI, and by chi-square test for gender.

3. Results

3.1. Effects of herring-roe powder on lipid metabolism

There were no significant differences in age, body weight, height, between the control and herring-roe groups (Table 2). Although BMI and body fat was significantly low in herring-roe group compared to placebo group, it did not influence the result of our clinical study. First, to determine the effect of herring-roe powder on lipid metabolism, we measured total cholesterol (T-Cho), high density lipoprotein cholesterol (HDL-Cho), LDL cholesterol (LDL-Cho), arteriosclerosis index, triglyceride (TG), nonesterified fatty acid (NEFA) (Fig. 3). T-Cho was significantly decreased by ingestion of placebo powder at the 8th week (placebo: −9.44 ± 18.65 mg/dl, herring-roe: 4.65 ± 11.96 mg/dl, as the change in the level of T-Cho from baseline to 8 weeks, $P = 0.01$) (Fig. 3a). However the herring-roe powder intake group improved HDL-Cho compared with the placebo powder intake group at 8 weeks (placebo: −1.38 ± 5.94 mg/dl, herring-roe: 4.41 ± 4.96 mg/dl, as the change in the level of HDL-Cho from baseline to 8 weeks, $P = 0.01$) (Fig. 3c). In addition, NEFA was decreased at 8 weeks by the ingestion of herring-roe powder (placebo: 0.01 ± 0.14 mEq/l, herring-roe: −0.12 ± 0.12 mEq/l, as the change in the level of NEFA from baseline to 8 weeks, $P = 0.01$) (Fig. 3f). There was no significant difference between the two groups of other lipid metabolism parameters.

3.2. Effects of herring-roe powder on adiponectin and leptin

We also examined the effect of herring-roe powder on adiponectin and leptin. Although no significant between-group differences were observed in the level of serum adiponectin (Fig. 2a), leptin was slightly increased by herring-roe powder intake (Fig. 2b) (placebo: −0.44 ± 1.76 ng/ml, herring-roe: 0.69 ± 1.62 ng/ml, as the change in the level of leptin from baseline to 8 weeks, $P = 0.06$).

3.3. Levels of biomarkers of blood metabolism, liver and renal function, glucose metabolism and body composition after the ingestion of herring-roe powder

We examined the levels of several biomarkers of blood metabolism, liver and renal function, and body composition. As shown in Table 3, minimal changes were observed in the parameters of glucose metabolism [fasting plasma glucose (FPG) and hemoglobin A1c (HbA1c)], liver function [alkaline phosphatase (ALP), aspartate aminotransferase (AST), alanine aminotransferase (ALT), lactate dehydrogenase (LDH), and gamma glutamyl transpeptidase (γ-