# Research Abstracts: krill oil

#### Evaluation of the effects of Neptune Krill Oil on the clinical course of hyperlipidemia

OBJECTIVE: To assess the effects of krill oil on blood lipids, specifically total cholesterol, triglycerides, low-density lipoprotein (LDL), and high-density lipoprotein (HDL). METHODS: A multi-center, threemonth, prospective, randomized study followed by a three-month, controlled follow-up of patients treated with 1 g and 1.5 g krill oil daily. Patients with hyperlipidemia able to maintain a healthy diet and with blood cholesterol levels between 194 and 348 mg per dL were eligible for enrollment in the trial. A sample size of 120 patients (30 patients per group) was randomly assigned to one of four groups. Group A received krill oil at a body mass index (BMI)-dependent daily dosage of 2-3 g daily. Patients in Group B were given 1-1.5 g krill oil daily, and Group C was given fish oil containing 180 mg eicosapentaenoic acid (EPA) and 120 mg docosahexaenoic acid (DHA) per gram of oil at a dose of 3 g daily. Group D was given a placebo containing microcrystalline cellulose. The krill oil used in this study was Neptune Krill Oil, provided by Neptune Technologies and Bioresources, Laval, Quebec, Canada. OUTCOME MEASURES: Primary parameters tested (baseline and 90-day visit) were total blood cholesterol, triglycerides, LDL, HDL, and glucose. RESULTS: Krill oil 1-3 g per day (BMI-dependent) was found to be effective for the reduction of glucose, total cholesterol, triglycerides, LDL, and HDL, compared to both fish oil and placebo. CONCLUSIONS: The results of the present study demonstrate within high levels of confidence that krill oil is effective for the management of hyperlipidemia by significantly reducing total cholesterol, LDL, and triglycerides, and increasing HDL levels. At lower and equal doses, krill oil was significantly more effective than fish oil for the reduction of glucose, triglycerides, and LDL levels. Bunea R, El Farrah K, Deutsch L. Altern Med Rev. 2004 Dec;9(4):420-8.

## Evaluation of the effects of Neptune Krill Oil on the management of premenstrual syndrome and dysmenorrhea

PRIMARY OBJECTIVE: To evaluate the effectiveness of Neptune Krill Oil (NKO) for the management of premenstrual syndrome and dysmenorrhea. SECONDARY OBJECTIVE: To compare the effectiveness of NKO for the management of premenstrual syndrome and dysmenorrhea with that of omega-3 fish oil. METHODS/ DESIGN: Double-blind, randomized clinical trial. SETTING: Outpatient clinic. PARTICIPANTS: Seventy patients of reproductive age diagnosed with premenstrual syndrome according to the Diagnostic and Statistical Manual of Mental Disorders, Third Edition, Revised (DSM-III-R). INTERVENTIONS: Treatment period of three months with either NKO or omega-3 fish oil. OUTCOME MEASURES: Self-Assessment Questionnaire based on the American College of Obstetricians and Gynecologists (ACOG) diagnostic criteria for premenstrual syndrome and dysmenorrhea and number of analgesics used for dysmenorrhea. RESULTS: In 70 patients with complete data, a statistically significant improvement was demonstrated among baseline, interim, and final evaluations in the self assessment questionnaire (P less than 0.001) within the NKO group as well as between-group comparison to fish oil, after three cycles or 45 and 90 days of treatment. Data analysis showed a significant reduction of the number of analgesics used for dysmenorrhea within the NKO group (comparing baseline vs. 45- vs. 90-day visit). The between-groups analysis illustrated that women taking NKO consumed significantly fewer analgesics during the 10-day treatment period than women receiving omega-3 fish oil (P less than 0.03). CONCLUSION: Neptune Krill Oil can significantly reduce dysmenorrhea and the emotional symptoms of premenstrual syndrome and is shown to be significantly more effective for the complete management of premenstrual symptoms compared to omega-3 fish oil. Sampalis F, Bunea R, Pelland MF, Kowalski O, Duguet N, Dupuis S. Altern Med Rev. 2003 May;8(2):171-179.

#### Fatty acids of astaxanthin esters in krill determined by mild mass spectrometry

Krill is a major source of astaxanthin, which has strong antioxidant activity. Fractions with astaxanthin monoesters and diesters of Antarctic krill Euphausia superba were isolated. Astaxanthin esters were separated by C18-HPLC depending on the number of carbons and double bonds of esterified fatty acid(s). Small amounts of other lipids remained in the samples, but relative molecular masses of carotenoid esters could be measured by field desorption mass spectrometry without fragmentation and interference from contaminant lipids. The fatty acids were determined by calculation of difference between astaxanthin and astaxanthin esters. Only five kinds of fatty acids, dodecanoate, tetradecanoate, hexadecanoate, hexadecenoate and octadecenoate, were detected. Fast atom bombardment mass spectrometry and secondary ion mass spectrometry showed similar spectra. The fatty acids in carotenoid esters by a combination of HPLC elution profile and mild mass spectrometry is found to be a useful tool. Takaichi S, Matsui K, Nakamura M, Muramatsu M, Hanada S. *Comp Biochem Physiol B Biochem Mol Biol.* 2003 Oct;136(2):317-22.

### Effects of n-3 and n-6 fatty acids on the activities and expression of hepatic antioxidant enzymes in autoimmune-prone NZBxNZW F1 mice

Menhaden fish oil (FO) containing n-3 fatty acids dramatically extends the life span and delays the onset and progression of autoimmune disease in (NZBxNZW)F1 (B/W) female mice as compared to those fed corn oil (CO) rich in n-6 lipids. As an inefficient antioxidant defense system has been linked to autoimmune diseases, the present study was undertaken to determine whether the protective action of n-3 lipids is mediated through their antioxidant defense system. Weanling B/W mice were fed a nutritionally adequate, semipurified diet containing CO or krill oil (KO) or FO at 10% level (w/w) ad libitum until the mice were 6.5 months old. All diets contained the same level of vitamin E (21.5 mg/100 g diet). We compared the effects of feeding n-6 and n-3 lipids on survival, kidney disease, hepatic microsomal lipid composition, peroxidation, and on the activity and mRNA expression of the antioxidant enzymes catalase, glutathione peroxidase (GSH-Px) and superoxide dismutase (SOD) in 6.5-month-old B/W mice. The results showed that when compared to livers from CO-fed mice, livers from KO- and FO-fed mice showed: (i) significantly higher (P < 0.001) activities and expression of CAT, GSH-Px and SOD; (ii) significantly lower (P < 0.001) arachidonic acid (20:4n-6) and linoleic acid (18:2n-6) and higher (P < 0.001) eicosapentaenoic acid (20:5n-3) and docosahexaenoic acid (22:6n-3) levels in hepatic microsomes; and (iii) significantly lower (P < 0.001) estimated peroxidation indices and thiobarbituric acid reactive substances generation. Venkatraman JT, Chandrasekar B, Kim JD, Fernandes G. Lipids. 1994 Aug;29(8):561-8.

#### Inhibition of low-density lipoprotein oxidation by astaxanthin

Marine animals produce astaxanthin which is a carotenoid and antioxidant. In this study we determined the in vitro and ex vivo effects of astaxanthin on LDL oxidation. The oxidation of LDL was measured in a 1 ml reaction system consisting of increasing concentrations of astaxanthin (12.5, 25.0, 50.0 microg/ml), 400 microM V-70 (2, 2'-azobis(4-methoxy-2, 4-dimethylvaleronitrile)), and LDL (70 microg/ml protein). Astaxanthin dose, dependently significantly prolonged the oxidation lag time (31.5, 45.4, 65.0 min) compared with the control (19.9 min). For the ex vivo study 24 volunteers (mean age 28.2 [SD 7.8] years) consumed astaxanthin at doses of 1.8, 3.6,14.4 and 21.6 mg per day for 14 days. No other changes were made in the diet. Fasting venous blood samples were taken at days 0, +14. LDL lag time was longer (5.0, 26.2, 42.3 and 30.7% respectively) compared with day 0 after consuming astaxanthin at doses of 1.8, 3.6,14.4 and 21.6 mg for 14 days. Our results provide evidence that consumption of marine animals producing astaxanthin inhibits LDL oxidation and possibly therefore contributes to the prevention of atherosclerosis. Iwamoto T, Hosoda K, Hirano R, et al. *J Atheroscler Thromb.* 2000;7(4):216-22.

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