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Effect of a fish oil-containing beverage on changes in plasma lipid fatty acids in patients with malabsorption

Karen C. McCowen, MD, MRCPI^{1, #}, Pei-Ra Ling, MD¹, Mario Ollero, PhD, DVM¹, Nicholas Tawa, MD², and Bruce R. Bistrian, MD, PhD¹

¹Department of Medicine, Beth Israel Deaconess Medical Center, Boston, MA

²Department of Surgery, Beth Israel Deaconess Medical Center, Boston, MA

Abstract

Background—The aim of this pilot study was to assess tolerance of a beverage containing ω -3 fatty acids (fish oil) in patients with malabsorption receiving chronic parenteral nutrition (PN). We wanted to determine whether fish oil could be absorbed and incorporated into plasma fatty acids, and reduce markers of inflammation.

Methods—This was a small intervention study in home-dwelling PN-dependent patients with chronic malabsorption. Ten patients were provided a drink containing 1.5g of fish oil per day for 12 weeks. Baseline and post-supplement serum fatty acid profiles were compared.

Results—Five of 10 patients withdrew from the study due to gastrointestinal side effects, principally worsened diarrhea, associated with the supplement. Modest increases were found in 20:5 ω -3, 22:5 ω -3, and 22:6 ω -3 levels in both phospholipids and triglycerides in plasma (all $p < 0.05$). In phospholipids, a reduced arachidonic acid level was seen ($p = 0.02$). These changes were not sufficient to effect improvements in serum tumor necrosis factor- α (TNF α), soluble TNF receptor, c-reactive protein or Interleukin-6.

Conclusions—Some patients with severe malabsorption can absorb oral ω -3 fatty acid supplements and can incorporate these fatty acids into serum phospholipids and triglycerides. However, side effects are very common, and no anti-inflammatory effect was found, presumably related to the modest level of fatty acid change.

Keywords

fatty acids; omega-3; fish oils; parenteral nutrition; malabsorption syndromes; short bowel syndrome; arachidonic acid

INTRODUCTION

A poorly understood phenomenon is the near-ubiquitous finding in home parenteral nutrition (HPN) patients of elevated serum lipid arachidonic acid. This occurs in parallel with low levels of its precursor linoleic acid, [1–3]. Lipid emulsions currently in use in the United

[#]To whom communications and reprint requests should be addressed: Harvard Vanguard Medical Associates, 133 Brookline Avenue (Medical Specialties), Boston, MA 02215, Tel:- 617 421 2142, Fax- 617 421 2707, kmccowen@bidmc.harvard.edu.

States are derived from soybean oil. They contain approximately 50% linoleic acid and 9% α -linolenic acid, the 2 essential fatty acids (Table 1). Arachidonic acid is not supplied in IV fat emulsion; the noted elevation in HPN patients might possibly represent abnormal essential fatty acid metabolism.

In our HPN patients, we have documented elevations in sensitive markers of inflammation [soluble tumor necrosis factor (TNF) receptor and interleukin-6 (IL-6)] [4], although not to the levels seen in obvious inflammatory states. Others have shown that the proportion of fat energy in HPN predicts the future development of PN-associated liver dysfunction [5,6]. The pro-inflammatory potential of the excessive arachidonic acid found in serum lipid might contribute to the development of this complication through modulation of eicosanoid production [7]. It has been documented that lowering of serum lipid arachidonic acid can be associated with reductions in markers of inflammation [8], in parallel with a rise in eicosapentaenoic (EPA) and docosahexaenoic acids (DHA).

Fish oil effectively lowers arachidonic acid in cell membranes. Fish oil also exhibits anti-inflammatory potential, likely through modification of cytokine and eicosanoid production [9–12]. The principal fatty acids of importance in fish oil are EPA and DHA, which are more potent at lowering arachidonic acid than is linolenic acid [8]. In the US, parenteral formulations of EPA and DHA are not available. Enteral formulations are often poorly tolerated.

In the present study, we have attempted to enhance enteral absorption of EPA and DHA by using a palatable oral solution containing approximately 1.5 g EPA + DHA per 8 fluid oz (300 kcal). This product (Prosure, Abbott Nutrition, Columbus, OH, see Table 2 for exact constituents) was well tolerated in patients with severe anorexia related to cancer of the pancreas [13]. The main goal was to determine whether our patients with severe malabsorption could tolerate this preparation of fish oil. Our hypothesis was that we could demonstrate incorporation of EPA and DHA into serum lipids. An ancillary goal was to determine whether this would be associated with reduction of arachidonic acid and lowering of the pro-inflammatory markers previously shown to be elevated in similar patients cared for by our team [4].

METHODS

Ten patients with malabsorption receiving HPN agreed to participate in the study [Table 3]. All had been dependent on HPN for more than 10 years and infused PN at least 5 out of 7 nights per week. All patients ate their usual diet both before and during the research study. This usual diet was not controlled or assessed in any way during or before the study. The Beth Israel Deaconess Medical Center Institutional Review Board including the ethics committee approved the research, and informed consent was obtained from all participants. All patients underwent thorough history and physical examination prior to enrollment and were deemed clinically stable and afebrile, with stable body weight over the preceding 3 months. Other enrollment criteria included ingestion of a regular diet, normal white blood cell count, and serum albumin level >3.5 g/L. Baseline blood samples were obtained at least

3 hours after discontinuation of the PN. Serum was separated within 2 hours of the blood draw, and frozen at -80°C until batched analyses.

Patients were provided with cans of ω -3 fatty acid-containing beverage and asked to ingest one-half can per day for 3 days to assess tolerance. Over the course of the next 2 weeks, they were instructed to increase by one-half can per day every 4 days to a maximum of 2 cans per day as tolerated and to report any worsening of baseline diarrhea or other symptoms. Weekly telephone calls to the participants ensured their well-being and maintenance of their usual diet and usual HPN. After 12 weeks, the patients returned for follow-up blood sampling and the study was discontinued. Patients were asked to have their usual breakfasts, and blood tests were obtained without regard to prior food intake.

Lipids from serum were extracted by using 6 vol of chloroform-methanol (2:1, v/v), centrifuged at $800 \times g$ for 3 min, and the resulting lower phase aspirated. Heptadecanoic acid was added to all samples as internal standard in the form of triheptadecanoyl glycerol and diheptadecanoyl phosphatidylcholine [$30 \mu\text{g}$ of each, from chloroform:methanol (1:1, v/v) stock solutions] (Nu-Chek Prep, Elysian, MN) prior to extraction. Lipid extracts from the different sample preparations were fractionated into triglycerides and phospholipids by solid phase chromatography using an aminopropyl column, as described elsewhere [14]. The resulting fractions were evaporated to dryness under nitrogen. Fatty acids were transmethylated by alkaline methanolysis using the BF_3 reagent kit (Supelco, Bellefonte, PA). Dry fractions were resuspended in 0.5 mL of methanolic base, vortexed and incubated at 100°C for 3 min. Subsequently, boron trifluoride-methanol (0.5 mL) was added, samples vortexed and incubated at 100°C for 1 min, prior to addition of hexane (0.5 mL), repeat vortexing, incubation at 100°C for 1 min, and addition of 6.5 mL of saturated NaCl. Samples were finally centrifuged at $800 g$ for 2 min. The hexane upper layer was transferred to a new glass tube and an aliquot injected in a Hewlett Packard 5890A gas chromatograph. A Supelcowax column of 30 m length and 0.5 mm internal diameter was used. Initial temperature was 150°C and final temperature 260°C . Detector temperature was 300°C and the total running time 27 min. Fatty acid methyl ester peaks were identified by comparison of retention times of standard mixtures (Nu-Chek-Prep, Elysian, MN), and quantified in comparison with the internal standard (methylheptadecanoate) detector response. Results are expressed as nmol percent of total fatty acid content in the sample as well as total nmol.

Serum levels of TNF α , soluble TNF receptor-II, and IL-6 were assayed using ELISA kits (Biosource International Inc., Camarillo, CA), with a detection threshold of 0.5 pg/mL, 0.1 ng/mL, and 0.16 pg/mL, respectively. CRP was measured in serum using an ELISA kit (Alpha Diagnostics International, San Antonio, TX), with a lower assay limit of 0.35 ng/mL.

STATISTICAL ANALYSIS

Data are presented as mean $\pm$ standard deviation (SD) for the patients who completed the study only. Paired t tests were used to compare values before and after the treatment. (SigmaStat 2.0, SSPS Inc., Chicago IL). Significance is defined by the 95% CI.

RESULTS

Clinical diagnoses and pertinent nutrition information for all 10 patients are shown in Table 3. Five patients were intolerant of the beverage and did not complete the 12 weeks of study. These tended to be the patients with extreme short bowel, 2 with history of volvulus requiring extensive small bowel resection, and 2 with superior mesenteric thrombosis. All of these subjects suffered from an unacceptable increase in stool frequency during the study. The patients who completed the protocol tolerated the supplement largely without incident, although (4 of 5) experienced diarrhea while drinking 2 cans per day, and reported consumption of 1 can/d only (Table 3). In addition, patient #4, was admitted with septicemia related to indwelling catheter infection during the final week of the study. All patients maintained their usual oral diet and their usual HPN during the study.

Table 4 shows changes in plasma phospholipid fatty acids before and after 12 weeks of supplement in the 5 patients who completed the study. Significant increases in incorporation of EPA (20:5 ω -3) and DHA (22:6 ω -3) as well as the intermediate docosapentaenoic acid (22:5 ω -3) were found, with an approximate doubling in either total amount, or percent of total fatty acids for EPA and DHA. Percentage of arachidonic acid was reduced in serum phospholipids. Triglyceride changes (Table 5) were very similar. In neither lipid fraction was relative conversion of linoleic acid to arachidonic acid altered, although this conversion was estimated only by product/ precursor ratios.

Figure 1 shows the effect of 12 weeks of the fatty acid-containing beverage on circulating markers of inflammation in each individual patient. No significant changes were found. The patient in whom septicemia developed at the end of the study is noted for each marker.

DISCUSSION

This small pilot study suggests that some patients with malabsorption who are PN dependent may be able to absorb ω -3 fatty acids from palatable formulations to effect phospholipid and perhaps membrane incorporation. This was associated with a reduction in phospholipid arachidonic acid, to levels seen in healthy controls from other labs [1] and from surgical controls in our own laboratory [15]. Whether such manipulation can be translated into any relevant clinical end-points remains to be investigated in larger studies and possibly over a longer duration. Unfortunately, many PN-dependent patients will be unwilling or unable to take oral nutritional supplements in addition to their nightly PN. We did not document the percentage of fat malabsorption in our participants, but others have shown that >50% malabsorption exists in most of these patients [16]. In our patients, it became obvious that those patients predicted to have the shortest bowel based on historical diagnosis were more likely to demonstrate poor tolerance for the supplement. We have noticed in clinical practice that our patients find commercially available pure fish oil capsules unpalatable, and elected to use a fish oil-containing mixed supplement based on patients' initial reports of tolerance during a tasting at our clinic.

Despite the small number of tolerant participants in the present study, we were able to demonstrate incorporation of EPA and DHA into serum phospholipids and triglycerides.

However, this occurred only to a modest degree compared to substantially larger degrees of incorporation when similar amounts of EPA and DHA are provided to subjects with intact intestinal function [17]. Similarly arachidonic acid accounted for a smaller percentage of fatty acids in the phospholipid fraction only, possibly as a result of displacement by EPA and DHA, unlike clear reductions seen in normal subjects. Triglyceride data may have been affected more readily by recent oral intake since we were unable in a clinical practice setting to restrict the patients' meals. A further limitation of this pilot study is that we made no attempt to perform any metabolic balance experiments to determine changes in conversion of linoleic acid to arachidonic acid. It is possible however, that additional effects of the ω -3 fatty acids included reduced conversion of linoleic acid to arachidonic acid, a known effect of the ω -3 fatty acids.

We have previously published the findings that IL-6 and sTNF receptor- II in urine and serum and TNF α in urine are elevated in a larger cohort of HPN patients compared with controls [18]. At that time, arachidonic acid in serum phospholipids and triglycerides was substantially elevated, and relative conversion from linoleic acid was similarly increased. One might propose a cause and effect relationship, based on the large literature that suggests pro-inflammatory properties for arachidonic acid. We have also published our findings in a rat model of malabsorption, in which rats underwent massive small bowel resection followed by therapy with PN, either with or without lipids [19]. We demonstrated that the presence of even short-term malabsorption leads to abnormal sensing of infused fatty acids, such that serum phospholipid arachidonic acid was significantly higher than in sham-operated rats receiving identical nutrition support.

Generally, EPA and DHA are considered anti-inflammatory by virtue of the ability, particularly of EPA, to displace arachidonic acid from membranes, as well as their inhibitory effect on delta 6 and delta 5 desaturases (the enzymes principally responsible for conversion of linoleic acid to arachidonic acid) [20]. However, a short course of supplementation that lead to a near-doubling of EPA and DHA incorporation into phospholipids in the present study may have been inadequate to improve the inflammatory state, if our hypothesis is correct. The data from the patient who developed septicemia around the time that the 12 weeks were ending clouded the data. However, even omission of those results did not suggest a trend to benefit in the remaining 4 patients. Clearly, had the supplement been well-tolerated, a larger sample size of subjects or perhaps even more ingested supplement among the subjects might have been able to show some effect. Fish oil supplementation has been shown to reduce inflammation in many conditions where the degree of inflammation is considerably more severe than the mild and subclinical inflammatory state seen with HPN [20]. Thus it is likely that the limited efficacy in this pilot study relates to the modest level of EPA and DHA incorporation and arachidonic acid lowering that was achieved.

Future studies for oral ω -3 supplementation of HPN patients should focus on patients with malabsorption who have not had major bowel resection. The greatest barrier to this therapy is gastrointestinal intolerance, which was most notable in those participants with extreme short bowel. Fish oil was only a small component of the beverage used in this study. Even though well-tolerated in anorectic subjects, perhaps providing much smaller amounts of supplement as the active agent only would have been better tolerated. Use of fish oil-

structured lipids as the source of fish oil might also be more effective [21] and should be investigated. In fish oil-structured lipids, the EPA or DHA can be located specifically in the sn-2 position of the triglyceride, rather than randomly as is the case in the triglyceride in the fish oil-containing beverage that we used. After initial digestion in the intestine, the 2-monoglyceride has superior absorption to the fatty acids from sn-1 or -3 positions, thus providing a particular advantage in patients with malabsorption [22]. Parenteral forms of ω -3 fatty acids (not yet available in the US) are used in Europe as a component of PN. These would presumably eliminate the problems with intolerance, and should be investigated for their anti-inflammatory properties in HPN patients. Preliminary studies in gut-intact postoperative patients have demonstrated lower circulating markers of inflammation when parenteral ω -3-enriched PN was compared with standard soybean oil [23–25].

Conclusions: use of oral fish oil supplements in short gut patients at risk of abnormal fatty acid status should be considered. If well tolerated, incorporation of these fatty acids into plasma lipids and presumably cell membranes can be anticipated based on our results.

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Figure 1.

Effect of 12 weeks of oral ω -3 supplementation on serum markers of inflammation in 5 patients completing the study.

* = patient #4, who developed septicemia at the conclusion of the study, at the time of the final blood draw.

CRP, C-reactive protein; IL, interleukin; TNF, tumor necrosis factor; RII, receptor-II

Table 1

Composition of Usual Soybean Oil Lipid Emulsion used in Parenteral Nutrition

Fatty Acid	%
Linoleic acid	50
Oleic	20
Palmitic	10
α -Linolenic	9
Stearic	3.5

Table 2

Constituents of a fatty acid-containing beverage per 237 ml (8 fluid ounces) (Prosure, Abbott Nutrition, Columbus, OH)

Energy profile

- Energy- 300 kcal (1.266 kcal/mL)
- Carbohydrates- 61% of total energy
- Protein- 21% of total energy
- Fat- 18% of total energy

Key Ingredients

- Refined deodorized sardine oil providing 1.09 g eicosapentaenoic acid (EPA) and 0.46 g docosahexaenoic acid (DHA)
 - Fermentable fiber system containing gum arabic and fructooligosaccharides (FOS)
 - Enhanced antioxidants containing beta-carotene and vitamin E
-

Table 3
Clinical Characteristics of Patients with malabsorption participating in trial of fatty acid-containing beverage

Patient	Age, sex	Weight	PN nights	PN kcal/d	%lipid energy kcal/kg/d	diagnosis	Bowel problem	Beverage tolerance (kg)
1	67, F	58	7	1739	20%	30	Motility disorder	Malabsorption 1 – 2 cans/d
2	63, F	62	7	1648	4%	27	Crohn's disease	Malabsorption 1 can/d
3	33, M	63	6	1411	4%	22	volvulus	Short bowel Drop-out- diarrhea
4	42, M	76	7	1833	10%	24	Volvulus	Short bowel Drop-out- diarrhea, nausea
5	77, M	57	6	1591	15%	28	Radiation enteritis	Malabsorption 1 can /d
6	46, F	54	6	1324	12%	25	SMT*	Short bowel Drop-out- diarrhea
7	43, F	48	6	1419	3%	30	Crohn's disease	Malabsorption 2 can/d
8	48, F	55	7	1808	7%	33	SMT*	Short bowel Drop-out- diarrhea
9	49, M	57	7	1599	20%	23	Crohn's disease	Short bowel 1 can/d
10	60, F	50	7	1288	15%	10	radiation enteritis	Malabsorption Drop-out- diarrhea

* SMT = superior mesenteric thrombosis; PN, parenteral nutrition

Table 4Changes in Plasma Phospholipid Fatty Acid Profile with fatty acid-containing beverage, mean $\pm$ SD

Fatty Acid	Before nmol/mL	After nmol/mL	Before mol %	After mol %
16:0	163.4 $\pm$ 25	170.3 $\pm$ 18.6	33.3 $\pm$ 3.7	33.6 $\pm$ 3.6
16:1 (ω -7)	6.8 $\pm$ 3.4	6.8 $\pm$ 1.9	1.4 $\pm$ 0.7	1.4 $\pm$ 0.5
18:0	56.5 $\pm$ 15.5	59.1 $\pm$ 14.1	11.3 $\pm$ 1.6	11.4 $\pm$ 1.4
18:1 (ω -9)	70 $\pm$ 17.7	71.7 $\pm$ 17.8	14 $\pm$ 1	14 $\pm$ 2.4
18:2 (ω -6)	94 $\pm$ 34	88 $\pm$ 29	18.7 $\pm$ 5	17.2 $\pm$ 5
18:3 (ω -3)	1.2 $\pm$ 0.8	1.3 $\pm$ 0.7	0.24 $\pm$ 0.1	0.26 $\pm$ 0.15
20:3 (ω -9)	0.23 $\pm$ 0.09	0.19 $\pm$ 0.08	0.05 $\pm$ 0.01	0.04 $\pm$ 0.01
20:3 (ω -6)	16 $\pm$ 5.4	13.6 $\pm$ 1.9	3.2 $\pm$ 0.07	2.7 $\pm$ 0.5
20:4 (ω -6) ³	61 $\pm$ 21	55.8 $\pm$ 23.7	12.1 $\pm$ 3.5	10.9 $\pm$ 4.2
20:5 (ω -3) ¹	4.1 $\pm$ 1.6	10.9 $\pm$ 6.5	0.8 $\pm$ 0.3	2.1 $\pm$ 1.2
22:4 (ω -6)	3 $\pm$ 1.2	2 $\pm$ 0.6	0.6 $\pm$ 0.2	0.39 $\pm$ 0.08
22:5 (ω -6)	2.6 $\pm$ 1.2	1.9 $\pm$ 0.6	0.5 $\pm$ 0.2	0.37 $\pm$ 0.1
22:5 (ω -3) ²	5 $\pm$ 1.3	7.5 $\pm$ 1.6	1 $\pm$ 0.2	1.5 $\pm$ 0.3
22:6 (ω -3) ¹	13.5 $\pm$ 7.4	21.2 $\pm$ 7.4	2.8 $\pm$ 1.7	4.1 $\pm$ 1.1
AA/ LA	0.76 $\pm$ 0.5	0.71 $\pm$ 0.47		

Beverage: Prosure, Abbott Nutrition, Columbus, OH

¹ p<0.05,² p<0.01, before Prosure Vs after Prosure³ p=0.02, before Prosure Vs after Prosure, nmol % only (for total nmol p=0.2, NS)

Table 5Changes in Plasma Triglyceride Fatty Acid Profile with fatty acid-containing beverage, mean $\pm$ SD

Fatty Acid	Before nmol/mL	After nmol/mL	Before mol %	After mol %
16:0	96.4 $\pm$ 24	131.3 $\pm$ 25	21.3 $\pm$ 4.4	22.9 $\pm$ 5.4
16:1 (ω -7)	38.6 $\pm$ 21	45.8 $\pm$ 16	8.6 $\pm$ 4.5	7.9 $\pm$ 2.7
18:0	6.1 $\pm$ 1.2	8.5 $\pm$ 1.4	1.4 $\pm$ 0.2	1.5 $\pm$ 0.4
18:1 (ω -9)	131 $\pm$ 31	171 $\pm$ 28	28.3 $\pm$ 1.2	29.3 $\pm$ 1.4
18:2 (ω -6)	147 $\pm$ 71	166 $\pm$ 50	31 $\pm$ 10.9	28.7 $\pm$ 8.2
18:3 (ω -3)	1.9 $\pm$ 0.5	2.5 $\pm$ 1	0.4 $\pm$ 0.1	0.45 $\pm$ 0.2
20:3 (ω -9)	0.03 $\pm$ 0.03	0.03 $\pm$ 0.03	0.01 $\pm$ 0.005	0.01 $\pm$ 0.005
20:3 (ω -6)	2.6 $\pm$ 0.6	2.9 $\pm$ 0.6	0.6 $\pm$ 0.08	0.5 $\pm$ 0.07
20:4 (ω -6)	29 $\pm$ 11	34 $\pm$ 18	6.4 $\pm$ 1.9	5.7 $\pm$ 2.4
20:5 (ω -3) ¹	3.1 $\pm$ 1.4	9.4 $\pm$ 5.5	0.7 $\pm$ 0.2	1.6 $\pm$ 0.9
22:4 (ω -6)	0.8 $\pm$ 0.3	0.8 $\pm$ 0.3	0.18 $\pm$ 0.08	0.13 $\pm$ 0.05
22:5 (ω -6)	0.5 $\pm$ 0.2	0.6 $\pm$ 0.2	0.13 $\pm$ 0.05	0.1 $\pm$ 0.03
22:5 (ω -3) ²	0.9 $\pm$ 0.2	2 $\pm$ 0.6	0.2 $\pm$ 0.05	0.34 $\pm$ 0.1
22:6 (ω -3) ²	2.3 $\pm$ 1.7	5 $\pm$ 2.6	0.5 $\pm$ 0.3	0.82 $\pm$ 0.35
AA/ LA	0.24 $\pm$ 0.17	0.22 $\pm$ 0.1		

¹ p<0.05,² p<0.01, before Prosure Vs after Prosure

Beverage: Prosure, Abbott Nutrition, Columbus, OH