

Effects of a liquid egg product containing fish oil on selected cardiovascular disease risk factors: A randomized crossover trial

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Abstract

The objective of the present human study was to evaluate the potential for a liquid egg product containing fish oil (providing eicosapentaenoic acid (EPA) plus docosahexaenoic acid (DHA)) as a functional food to favourably modify circulating triglyceride levels and other risk factors for cardiovascular disease (CVD). A two-period randomized, controlled, crossover trial was conducted in 16 healthy males with moderately elevated triglyceride levels. The participants were randomly assigned to consume either a breakfast containing the liquid egg product providing 1.3 g/day of EPA/DHA combined or a control breakfast, each for 21 days.

The breakfast containing the liquid egg product significantly decreased the plasma triglyceride levels by 32% ($P < 0.05$), the triglyceride:HDL-cholesterol ratio by 37% ($P < 0.05$), and moderately reduced blood pressures whereas no such effects were observed with the control breakfast. No effects on total cholesterol and LDL-cholesterol were observed. The fatty acid composition of serum phospholipid showed an increase of 210% ($P < 0.001$) in EPA and 96% ($P < 0.001$) in DHA and shifting to a lower risk status for the EPA/DHA sum. Use of this liquid egg product as a functional food could serve as a dietary intervention for supporting CVD risk management.

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1. Introduction

Elevated serum lipid concentrations have been identified as major risk factors for cardiovascular disease (CVD) (NCEP Expert Panel, 2001). It has been confirmed that high total cholesterol and low-density lipoprotein-cholesterol (LDL-C) concentrations and low high-density lipoprotein-cholesterol (HDL-C) concentrations are associated with increased risk for CVD. However, the relation of serum or plasma triglyceride concentrations and CVD has been controversial (Gotto, 1998; Miller, 1998). Emerging evidence suggests that triglycerides may act as an independent risk factor for CVD (Cullen, 2000). Several studies, including the Framingham Study (Castelli, 1992), the Copenhagen Male Study (Jeppesen, Hein, Suadicani, & Gyntelberg,

1998), and the PROCAM Study (Assmann, Schulte, & von Eckardstein, 1996) found that elevated triglycerides were predictive of CVD. Some of the most convincing evidence is a recent meta-analysis of 17 population-based prospective studies of hypertriglyceridemia as a risk factor for CVD that suggested a 1.0 mmol/L increase in triglyceride is associated with a 76% and 32% increase in CVD risk for women and men, respectively (Austin, Hokanson, & Edwards, 1998). The ratio of triglyceride: HDL-C has also been shown to be a strong predictor of myocardial infarction (Gaziano, Hennekens, O'Donnell, Breslow, & Buring, 1997), as well as having a strong association with CVD incidence and involvement in the transition from atheroma to atherothrombosis (Sharrett et al., 1999).

It has long been recognized that the Greenland Inuit, with a high intake of seafood, exhibit significantly lower death rates from acute myocardial infarction when compared to Danes (Bang & Dyerberg, 1980). A recent meta-analysis of the cohort studies has indicated that

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increased fish consumption is associated with decreased mortality from CVD (He et al., 2004). Fish are enriched in the long chain polyunsaturated $n - 3$ (Omega-3) fatty acids, eicosapentaenoic acid (EPA, 20:5 $n - 3$) and docosahexaenoic acid (DHA, 22:6 $n - 3$) and exhibit their cardiovascular benefits via numerous mechanisms (Holub & Holub, 2004).

A strong inverse relationship between the levels of EPA plus DHA in blood plasma phospholipid and the risk of fatal ischemic heart disease has been reported (Lemaitre et al., 2003). Controlled intervention trails with encapsulated fish oil supplements containing EPA plus DHA have established their triglyceride-lowering effects (Harris, 1996) and their modifying influence on other CVD risk factors independent of blood lipid-lowering (Harper & Jacobson, 2005; Holub & Holub, 2004).

The American Heart Association Guidelines (Revision 2000) recommended 900 mg/day as the target intake of $n - 3$ fatty acids (EPA plus DHA), from fish or a fish oil supplement, in patients with coronary disease (Krauss, Eckel, & Howard, 2000). Current mean intakes of EPA and DHA combined in the US and Canada are approximately 130–150 mg/day (Denomme, Stark, & Holub, 2005; Kris-Etherton et al., 2000; Raper, Cronin, & Exler, 1992) or 14% of the recommendation. Multiple reasons for the wide nutrition gap, actual versus advised intakes, have been reported for both fish and fish oil capsules. The main reasons reported for the unpopularity of fish are unpleasant taste and smell, oiliness, bones, lack of knowledge of proper cooking techniques and the perception that fatty fish consumption is fattening (Jones & Le Cornu, 1994). Encapsulated fish oil supplements often produce side effects such as fishy tastes (or burps) and nausea (Mueller & Talbert, 1988) and many consumers have difficulty with the capsule size and number.

Recently, an EPA/DHA-enriched liquid egg product (Break Free Omega-3; Burnbrae Farms, Lyn, ON) has become available as a functional food in Canada offering a new optional source of $n - 3$ fatty acids previously available in substantial amounts only from fatty fish or fish oil capsules. The purpose of the present study was to evaluate its effects on selected cardiovascular risk factors, especially circulating triglyceride levels and the triglyceride:HDL-cholesterol ratio, as well as the fatty acid composition of blood serum phospholipid, in a two-period, randomized, controlled, crossover trial.

2. Methods

2.1. Subjects

Men between the ages of 30 and 65 were recruited from the Guelph community area in Canada. The individuals were screened and only men with fasting triglyceride levels of >88.5 mg/dL (>1.0 mmol/L) and those who had not consumed fish or fish oil capsules two weeks prior were considered eligible for the study. Individuals taking lipid-

altering medications were excluded. Individuals on blood pressure-altering medications were allowed to participate in the study so long as they had been on the treatment for more than 6 months. A total of 16 subjects took part in the study. One subject withdrew from the study because of unrelated causes. None of the subjects were diagnosed with CVD. The Human Subjects Committee of the University of Guelph approved this study and all subjects gave their written and informed consent.

2.2. Study design

For preliminary screening purposes, fasting blood lipid profiles were measured using an instant blood lipid analyzer and a finger prick test (Cholestech LDX System; Cholestech, Hayward, CA). Each subject was randomly assigned to either receive the liquid egg breakfast or the control breakfast first. The means of the screened blood lipid values were balanced during the assignment. The subjects were not allowed to consume fish or fish oil capsules while enrolled in the study.

A two-period, randomized, controlled, crossover study was conducted as shown in Fig. 1. The subjects were assigned to either eat the liquid egg breakfast or a control breakfast for an initial 21-day period and the alternate for the second 21-day period. The two periods were separated by a washout period of 10 weeks, during which the subjects consumed their usual diets. The liquid egg breakfast provided 0.63 g/day of EPA and 0.64 g/day of DHA, a total of 1.3 g/day of EPA plus DHA, whereas the control breakfast provided a total of only 0.013 g/day of EPA plus DHA.

The subjects were given their breakfast between 7 and 9 a.m., for 21 days, while under supervision. The control breakfast consisted of back bacon, frozen waffles, non-hydrogenated light margarine and syrup. The liquid egg breakfast consisted of the egg product scrambled, toasted white bread and non-hydrogenated light margarine. These breakfasts were balanced for protein, carbohydrate, and total fat content (Table 1). Optional beverages were constant for each individual throughout both periods.

Subjects were required to provide venous blood samples after fasting overnight (at least 12 h) on day 0 and day 22 of each period of the study. These samples were analyzed for plasma total cholesterol, LDL-C, HDL-C, triglyceride, and glucose. The fatty acid compositions of the serum phospholipid, including levels of EPA and DHA, were also measured to assess the physiological $n - 3$ fatty acid status.

Duplicate measures of sitting blood pressure and resting heart rate were determined with a computerized oscillometric blood pressure monitor computer program (Cardio-Vision Model MS-2000; International Medical Device Partners, Inc., Las Vegas, NV). Duplicate measures of height were taken on day 0 of period one, while weight was measured at each visit. Each subject completed a

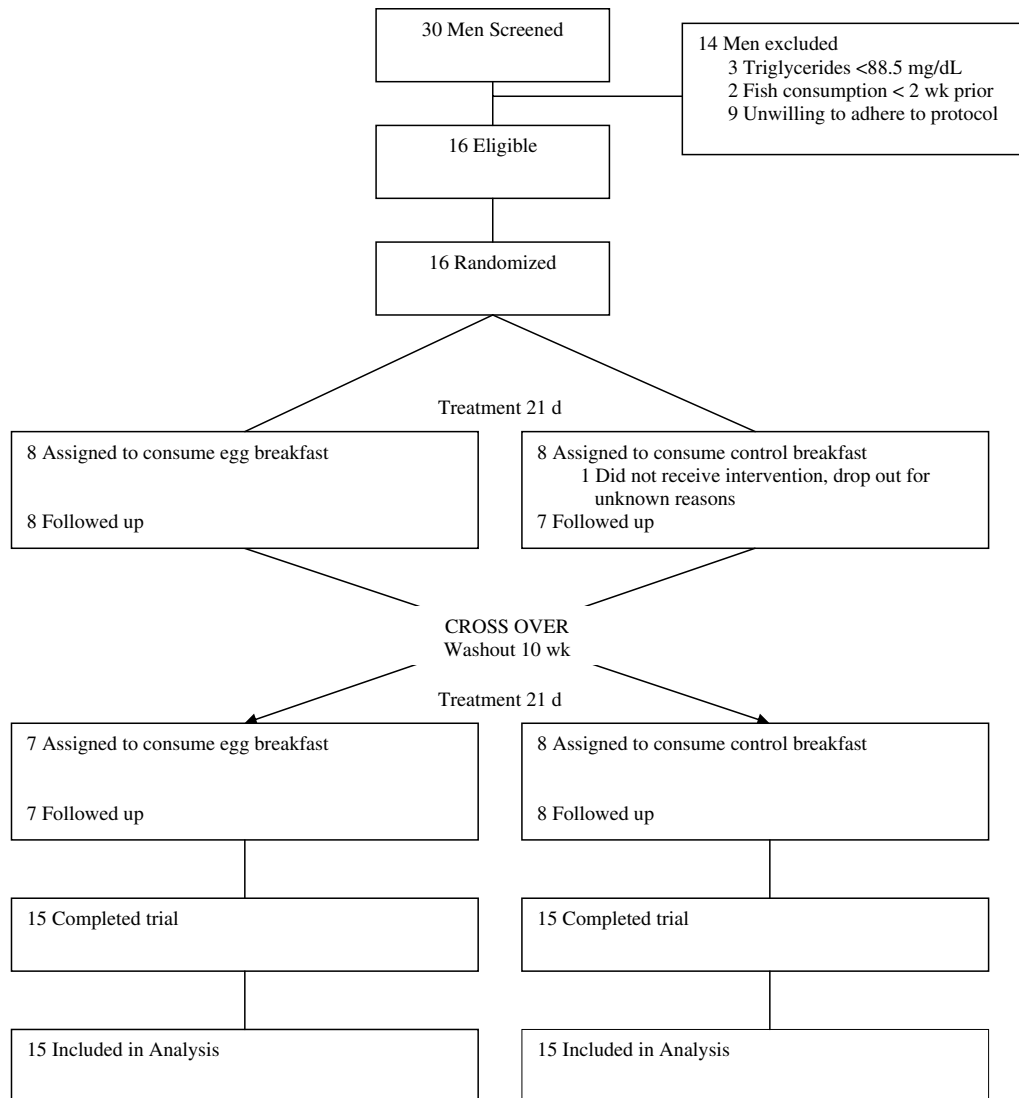


Fig. 1. Study design.

Table 1
Breakfast meals and daily dietary intakes (including the breakfast meals)^a

	Egg breakfast	Control breakfast
<i>Macronutrient content of the breakfast meals^b</i>		
Protein (g)	35.0 (0.8)	35.6 (1.3)
Carbohydrate (g)	57.7 (6.6)	50.4 (7.9)
Fat (g)	12.7 (3.3)	12.5 (4.7)
EPA plus DHA (g)	1.30	0.01
<i>Daily dietary intakes</i>		
Protein (g/day)	127.3 (28.8)	122.3 (19.1)
Carbohydrate (g/day)	350.4 (98.8)	367.8 (98.5)
Fat (g/day)	96.9 (23.4)	93.5 (25.6)
Saturated fat (g/day)	35.4 (12.1)	33.1 (10.4)
Monounsaturated fat (g/day)	32.6 (9.1)	34.4 (9.2)
Polyunsaturated fat (g/day)	17.8 (6.6)	14.1 (4.4)
Dietary fiber (g/day)	16.9 (8.7)	18.6 (8.4)
Cholesterol (mg/day)	237.6 (99.4)	244.4 (92.4)
Alcohol (% of total energy)	1.7 (2.7)	2.4 (4.0)
Total energy (kcal)	2801 (587)	2838 (603)

^a Values express as mean (SD).^b 250 ml of liquid egg product provided 240 kcal.

3-day dietary record (on 2 weekdays and 1 weekend day) during each period of the study. Dietary records were analyzed by using Food Processor, a nutrition analysis system (version 7.11; ESHA Research, Salem, OR).

2.3. Laboratory analysis

Blood was collected by venipuncture into evacuated tubes (Vacutainer; Becton Dickinson, Rutherford, NJ). After the samples sat for 1 h, they were centrifuged (1000g for 15 min at 30 °C). The recovered serum and plasma were divided into aliquots and they were stored at –80 °C until analyzed. Plasma triglyceride, total cholesterol, HDL-C, and glucose were quantified enzymatically (Synchron CX Systems; Beckman Coulter, Inc., Fullerton, CA). LDL-C concentrations were determined by using the Friedewald equation (Friedewald, Levy, & Fredrickson, 1972). The fatty acid compositions, including EPA and DHA, of serum phospholipid were determined by capillary

gas–liquid chromatography following isolation of the phospholipid by thin-layer chromatography and trans-methylation (Dewailly et al., 2001).

Four sample breakfasts from each breakfast treatment were analyzed to verify their macronutrient content. AOAC laboratory methods and calculations were utilized.

2.4. Statistical analysis

Statistical analyses were performed using SAS version 6.12 (SAS Institute, Cary, NC). Baseline comparisons between the liquid egg breakfast first group and the control breakfast first group were made with an independent *t* test. The macronutrient content of the breakfast meals was tested to confirm balancing using an independent *t* test. After checking for normality, the triglyceride concentrations were logarithmically transformed before the analyses because of skewness. Carryover effects were determined by two-tailed paired *t* tests before the data from each period was pooled. A paired *t* test was used to test baseline and follow-up values within the liquid egg breakfast and the control breakfast. For the comparison between the liquid egg breakfast and the control breakfast, an analysis of variance (ANOVA) was performed with the general linear model procedure, with subject (nested within group), group, treatment, and period in the model. If there was a significant treatment effect by *F* test, follow up comparisons of treatment means were done by the least-significant-difference test. The data are given as mean (SD) values with 95% confidence intervals for the mean differences.

3. Results

No differences were noted in the baseline characteristics of the two assigned groups (Table 2). There was no carry-over effect seen in the study, as the values at baseline in period one were not significantly different from the baseline values in period two.

Breakfast and dietary intake data revealed no statistically significant differences between the two breakfasts except for the presence of EPA and DHA in the liquid egg breakfast (1.30 g versus 0.01 g in the control) (Table 1).

Plasma lipid concentrations are shown in Table 3. Consumption of the liquid egg breakfast containing *n* – 3 fatty acids was associated with a significant decrease (by 32% overall) in triglyceride concentrations. All but one of the individuals exhibited a triglyceride-lowering effect. There was also a significant reduction (by 37% overall) in the triglyceride: HDL-C ratio. No significant changes were observed in either breakfasts for plasma cholesterol, LDL-C, or the cholesterol: HDL-C and LDL-C: HDL-C ratios. A modest decrease in systolic blood pressure and mean arterial pressure was observed in the liquid egg breakfast.

The fatty acid compositions of the serum phospholipid are shown in Table 4. EPA levels rose by 210% overall (0.63 to 1.95% by weight of the fatty acids) and DHA levels

Table 2
Characteristics of the subjects at baseline^a

Characteristic	All subjects (<i>n</i> = 15)	Egg breakfast first (<i>n</i> = 8)	Control breakfast first (<i>n</i> = 7)
Age (y)	47.8 (8.1)	49.1 (9.1)	46.3 (7.0)
Height (cm)	177.2 (7.2)	174.6 (6.9)	180.2 (6.7)
Weight (kg)	89.6 (17.6)	90.1 (14.9)	89.1 (21.5)
BMI (kg/m ²)	28.6 (5.7)	29.7 (6.3)	27.2 (5.1)
Systolic BP (mm Hg)	140.1 (21.0)	140.7 (13.6)	139.4 (28.4)
Diastolic BP (mm Hg)	91.8 (9.0)	94.3 (5.8)	89.0 (11.0)
MAP (mm Hg)	107.9 (11.2)	109.8 (7.7)	105.8 (14.6)
Heart rate (beats/min)	71.3 (10.9)	66.9 (9.2)	77.0 (11.1)
Pulse pressure (mm Hg)	47.7 (16.7)	46.5 (10.5)	49.1 (22.7)
Cholesterol (mg/dL)	210.4 (45.1)	210.8 (46.3)	211.4 (47.3)
LDL-C (mg/dL)	135.5 (30.1)	135.4 (27.3)	136.8 (35.5)
HDL-C (mg/dL)	42.8 (6.9)	41.0 (6.8)	45.3 (6.8)
Triglyceride (mg/dL)	165.0 (79.0)	172.3 (96.4)	147.5 (66.4)
Glucose (mg/dL)	92.3 (8.94)	93.8 (7.38)	92.3 (11.1)

BMI: body mass index; BP: blood pressure; MAP: mean arterial pressure; LDL-C: low-density lipoprotein cholesterol; HDL-C: high-density lipoprotein cholesterol. To convert mg/dL to mmol/L for cholesterol, LDL-C and HDL-C multiply by 0.0259, for triglycerides multiply by 0.0113, and for glucose multiply by 0.05555.

^a Values are expressed as mean (SD).

by 96% overall (2.47 to 4.83%), respectively, with consumption of the *n* – 3 fatty acid-containing liquid egg breakfast. Total *n* – 3 fatty acid levels approximately doubled, as did the *n* – 3:*n* – 6 ratio. In contrast, no significant fatty acid changes were found with the control breakfast.

The subjects reported no adverse effects while on either of the breakfasts. Further, no detection of a fishy taste in the liquid egg breakfast was reported.

4. Discussion

The predominant source of EPA and DHA in the US and Canadian diet is fish which accounts for 90% and 75%, respectively, of the total daily consumption of these *n* – 3 fatty acids (Raper et al., 1992). Currently, the US populations are only consuming approximately 130–150 mg/day of EPA and DHA combined (Denomme et al., 2005; Kris-Etherton et al., 2000; Raper et al., 1992), which is well below the recommended intake of 900 mg/day from fish or fish oil supplementation in patients with CHD based on recent recommendations released by the American Heart Association (Krauss et al., 2000). It has been estimated that the average yearly contribution of EPA and DHA from fish oil supplements is only 0.6–0.9 mg/person indicating that fish oil supplements are currently only a trivial source of *n* – 3 fatty acids (Kris-Etherton et al., 2000). Unpleasant fishy taste plus other factors are responsible for the very low consumption of fish and fish oil supplements (Jones & Le Cornu, 1994; Mueller & Talbert, 1988). The liquid egg product as used herein provided 1.3 g/day of EPA and DHA, as a breakfast food, in 240 kcal, which represented approximately 10% of the total daily caloric intake. The EPA/DHA content per 100 g in the

Table 3
Effects of breakfast meals on cardiovascular risk factors^a

Variable	Egg breakfast				Control breakfast			
	Baseline	Follow up	Mean difference (95% CI)	<i>P</i> value ^b	Baseline	Follow up	Mean difference (95% CI)	<i>P</i> value ^b
Weight (kg)	89.8 (18.0)	90.0 (18.0)	0.2 (−2.3 to 3.1)	0.77	89.5 (17.6)	89.4 (17.7)	−0.1 (−1.0 to 0.9)	0.91
BMI (kg/m ²)	28.6 (5.8)	28.7 (5.7)	0.0 (−0.4 to 0.5)	0.81	28.5 (5.6)	28.5 (5.7)	−0.02 (−0.2 to 0.1)	0.70
Systolic BP (mm Hg)	138 (15.6)	129 (15.3)	−9.0 (−14.4 to −3.6) ^c	0.003	132 (21.9)	131 (17.9)	−0.8 (−9.0 to 7.3)	0.83
Diastolic BP (mm Hg)	90.0 (8.7)	83.4 (8.0)	−6.5 (−10.5 to −2.6)	0.003	83.9 (14.5)	83.7 (8.4)	−0.2 (−6.8 to 6.4)	0.95
MAP (mm Hg)	106 (10.2)	98.5 (9.4)	−7.4 (−11.1 to −3.6) ^c	0.0009	100 (14.7)	99.6 (9.1)	−0.4 (−6.1 to 5.2)	0.88
Heart rate (beats/min)	74.6 (11.9)	72.8 (13.6)	−1.9 (65.2 to 80.3)	0.49	74.2 (11.1)	76.0 (8.9)	1.6 (−2.8 to 6.1)	0.44
Pulse pressure (mm Hg)	47.7 (11.0)	46.6 (12.4)	−1.1 (−5.5 to 3.3)	0.10	45.3 (16.5)	47.0 (14.3)	1.7 (−5.6 to 9.0)	0.62
Cholesterol (mg/dL)	209 (40.0)	205 (37.6)	−3.6 (−12.7 to 5.5)	0.009	208 (43.3)	198 (34.4)	−10.9 (−22.3 to 0.6)	0.06
LDL-C (mg/dL)	132 (25.3)	139 (27.4)	7.0 (−1.4 to 15.4)	0.09	136 (30.3)	130 (25.5)	−5.8 (−61.7 to 10.8)	0.04
HDL-C (mg/dL)	43.5 (8.1)	47.0 (10.0)	3.5 (1.0 to 5.9)	0.008	42.5 (6.1)	45.5 (7.0)	3.1 (0.2 to 5.9)	0.32
Triglyceride (mg/dL)	165 (79.0)	111 (62.2)	−53.1 (−77.7 to −28.5) ^c	0.0003	152 (83.5)	127 (48.2)	−25.4 (−61.7 to 10.8)	0.16
Cholesterol: HDL-C	4.88 (1.0)	4.49 (1.0)	−0.39 (−0.67 to −0.12)	0.008	4.94 (0.9)	4.37 (0.6)	−0.57 (−0.9 to −0.2)	0.005
LDL-C: HDL-C	3.10 (0.72)	3.10 (0.80)	−0.05 (−0.27 to 0.18)	0.56	3.22 (0.70)	2.86 (0.40)	−0.36 (−0.63 to −0.09)	0.004
Triglyceride: HDL-C	3.89 (2.0)	2.5 (1.5)	−1.42 (−1.9 to −0.89) ^c	0.0001	3.58 (1.8)	2.83 (1.1)	−0.76 (−1.6 to 0.1)	0.14
Glucose (mg/dL)	95.0 (17.6)	95.7 (14.6)	0.71 (−4.1 to 5.6)	0.76	90.0 (9.0)	95.0 (10.7)	5.0 (−0.9 to 10.9)	0.09

CI: confidence interval; BMI: body mass index; BP: blood pressure; MAP: mean arterial pressure; LDL-C: low-density lipoprotein cholesterol; HDL-C: high-density lipoprotein cholesterol. To convert mg/dL to mmol/L for cholesterol, LDL-C and HDL-C multiply by 0.0259, for triglycerides multiply by 0.0113, and for glucose multiply by 0.05555.

^a Values are expressed as mean (SD).

^b *P* values determined by a paired *t*-test (follow up vs. baseline).

^c *P* < 0.05 compared with control breakfast, using LSD test.

Table 4
Effects of breakfast meals on fatty acid compositions of serum phospholipid^a

Fatty acids	Egg breakfast				Control breakfast			
	Baseline	Follow up	Mean difference (95% CI)	<i>P</i> value ^b	Baseline	Follow up	Mean difference (95% CI)	<i>P</i> value ^b
<i>% by wt of total fatty acids</i>								
16:0	27.27 (1.4)	27.77 (1.0)	0.51 (−0.15 to 1.2) ^c	0.12	27.67 (1.5)	26.99 (1.2)	−0.68 (−1.4 to 0.03)	0.06
18:0	13.43 (1.0)	13.29 (0.8)	−0.14 (−0.69 to 0.41)	0.59	13.21 (0.7)	13.17 (0.6)	−0.05 (−0.44 to 0.35)	0.80
18:1	11.80 (1.4)	10.57 (1.1)	−1.23 (−1.9 to −0.53) ^d	0.002	11.79 (1.1)	12.12 (1.0)	0.33 (−0.25 to 0.91)	0.24
18:2 <i>n</i> − 6	20.80 (2.2)	18.20 (1.9)	−2.60 (−3.1 to −2.1) ^c	0.0001	20.72 (2.6)	20.79 (2.0)	0.07 (−0.87 to 1.0)	0.88
20:3 <i>n</i> − 6	3.30 (0.5)	2.70 (0.5)	−0.60 (−0.78 to −0.41) ^d	0.0001	3.31 (0.6)	3.32 (0.4)	0.01 (−0.25 to 0.27)	0.96
20:4 <i>n</i> − 6 (AA)	10.26 (2.0)	9.92 (1.5)	−0.35 (−0.87 to 0.18)	0.18	10.06 (1.8)	10.45 (1.8)	0.39 (0.01 to 0.76)	0.04
20:5 <i>n</i> − 3 (EPA)	0.63 (0.3)	1.95 (0.5)	1.32 (1.1 to 1.5) ^e	0.0001	0.69 (0.3)	0.64 (0.2)	−0.06 (−0.12 to 0.01)	0.09
22:5 <i>n</i> − 3	0.95 (0.2)	1.19 (0.2)	0.25 (0.16 to 0.33) ^e	0.0001	0.97 (0.2)	0.99 (0.2)	0.02 (−0.03 to 0.07)	0.46
22:6 <i>n</i> − 3 (DHA)	2.47 (0.5)	4.83 (0.6)	2.36 (2.0 to 2.7) ^e	0.0001	2.45 (0.6)	2.39 (0.5)	−0.06 (−0.23 to 0.10)	0.41
<i>n</i> − 6 (sum)	36.08 (1.6)	32.31 (1.6)	−3.76 (−4.3 to −3.2) ^e	0.0001	35.77 (2.0)	36.21 (1.3)	0.44 (−0.36 to 1.2)	0.76
<i>n</i> − 3 (sum)	4.47 (0.7)	8.49 (1.1)	4.03 (3.5 to 4.6) ^e	0.0001	4.47 (0.7)	4.44 (0.5)	−0.04 (−0.29 to 0.21)	0.26
<i>n</i> − 3: <i>n</i> − 6 ratio	0.12 (0.02)	0.26 (0.04)	0.14 (0.12 to 0.16) ^e	0.0001	0.13 (0.02)	0.12 (0.01)	−0.0007 (−0.009 to 0.007)	0.87
EPA:AA	0.06 (0.03)	0.20 (0.06)	0.14 (0.12 to 0.17) ^e	0.0001	0.07 (0.02)	0.06 (0.02)	−0.007 (−0.01 to −0.0002)	0.04
DHA:AA	0.25 (0.08)	0.50 (0.1)	0.25 (0.20 to 0.29) ^e	0.0001	0.25 (0.08)	0.24 (0.07)	−0.01 (−0.03 to 0.002)	0.08

CI: confidence interval; BMI: body mass index; BP: blood pressure; MAP: mean arterial pressure; LDL-C: low-density lipoprotein cholesterol; HDL-C: high-density lipoprotein cholesterol. To convert mg/dl to mmol/L for cholesterol, LDL-C and HDL-C multiply by 0.0259, for triglycerides multiply by 0.0113, and for glucose multiply by 0.05555.

^a Values are expressed as mean (SD).

^b Values determined by a paired *t*-test (follow up vs. baseline).

^c *P* < 0.05 compared with control breakfast, using LSD test.

^d *P* < 0.01 compared with control breakfast, using LSD test.

^e *P* < 0.001 compared with control breakfast, using LSD test.

liquid egg product, as consumed without reports of fishy taste, is significantly higher than that for many types of fish, including tuna, halibut, cod, others (Holub & Holub, 2004; Kris-Etherton et al., 2000). Egg products can serve as ideal functional foods for the dietary delivery of the cardioprotective $n - 3$ fatty acids (EPA/DHA) since the US population consumes relatively high levels of eggs (Song & Kerver, 2000), which are also rich sources of various other important nutrients, including essential amino acids, vitamins, etc.

As reviewed (Austin et al., 1998; Gotto, 1998), serum/plasma triglyceride levels are now becoming accepted as both an independent risk factor for CVD and a synergistic risk factor with other lipid risk factors. The NCEP clinical guidelines recognize triglyceride levels of 150–199 mg/dL as being borderline-high (NCEP Expert Panel, 2001). The daily consumption of the liquid egg breakfast produced a 32% reduction in fasting plasma triglyceride levels in our male subjects having group mean entry levels of 165 mg/dL. This reduction was without an accompanying change in total cholesterol or LDL-C levels. Other investigators using very similar intakes of EPA/DHA combined per day to those used herein have reported blood triglyceride-lowering of approximately 25% (Brown & Roberts, 1991; Demke, Peters, Linet, Metzler, & Klott, 1988). In general, subjects with moderately elevated triglyceride levels (as used herein) tend to show greater triglyceride reductions with fish oil than those with lower initial triglyceride levels. The marked suppression observed in the triglyceride:HDL-C ratio (by 37% overall) is of considerable interest since this ratio was shown to be strongly associated with the risk of myocardial infarction (Gaziano et al., 1997) and to be a possible marker for the progression of atherosclerosis (Sharrett et al., 1999). The moderate decrease in systolic blood pressure, observed herein on the liquid egg breakfast, has been observed in several fish oil trials as revealed by meta-analysis (Morris, Sacks, & Rosner, 1993). Based on the magnitude of the triglyceride reduction after consumption of the liquid egg breakfast, a calculated reduction in the risk of CVD of 22% is estimated (Austin et al., 1998) and a 27% reduction in the risk of myocardial infarction based on the PROCAM data (Assmann et al., 1996). It is of interest to note that one-third of the adult males in the US have triglyceride levels >150 mg/dL (Ford, Giles, & Dietz, 2002) who could potentially benefit from functional foods with demonstrated triglyceride-lowering capacity. Interestingly, a 24% reduction in the combined outcome of death from CHD, non-fatal myocardial infarction and stroke has been reported in men with CHD given gemfibrozil which produced a 31% reduction in serum triglyceride and a 6% rise in mean HDL-C levels.

The liquid egg breakfast produced a marked increase in the total $n - 3$ polyunsaturated fatty acids and DHA in the blood serum phospholipid, a recognized biomarker for their physiological status and intake (Andersen, Solvoll, & Drevon, 1996; Bjerve et al., 1993; Dewailly et al., 2001). The summed levels of EPA plus DHA in the serum

phospholipid (Table 4) following the liquid egg breakfast (6.8% of fatty acids) is in the range wherein these have shown an inverse relationship to plasma triglyceride levels in a population study (Dewailly et al., 2001). Further, the shifting of the summed EPA plus DHA levels from a mean entry value of 3.1 to 6.8% of total fatty acids (that is >4.6%) is consistent with a shift from a higher risk to low risk status for fatal ischemic heart disease based on Lemaitre et al. (2003).

5. Conclusion

In conclusion, a convenient liquid egg breakfast as a novel source of considerable levels of EPA plus DHA was very well tolerated as a functional food. This human study indicates the potential for a functional food (liquid egg) containing fish-derived EPA/DHA to favourably influence multiple risk factors for cardiovascular disease. Wide application of such a functional food in alternative or complementary cardioprotective management, of healthy individuals and those with moderate hypertriglyceridemia as part of the metabolic syndrome, may be particularly important in those who eat little or no fish for various reasons.

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