ORIGINAL ARTICLE

Effects of weight loss and seafood consumption on inflammation parameters in young, overweight and obese European men and women during 8 weeks of energy restriction

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Background/Objectives: *In vitro* studies have shown that long-chain n-3 polyunsaturated fatty acids (LC n-3 PUFAs) can affect inflammation; however, results from intervention studies in overweight or obese individuals are contradicting. The aim of this study was to investigate the effects of weight loss and seafood consumption on inflammation parameters during energy restriction.
Subjects/Methods: In this 8-week intervention trial, 324 subjects (aged 20–40 years, body mass index 27.5–32.5 kg/m^2 from Iceland, Spain and Ireland) were randomized to one of four energy-restricted diets (−30% relative to estimated requirements): salmon (3 × 150 g/week, 2.1 g LC n-3 PUFA per day); cod (3 × 150 g/week, 0.3 g LC n-3 PUFA per day); fish oil capsules (1.3 g LC n-3 PUFA per day); and control (sunflower oil capsules, no seafood). Body weight, high-sensitivity C-reactive protein (CRP), interleukin-6 (IL-6), glutathione reductase and prostaglandin F2 alpha (PGEF2α) were measured at baseline and end point.
Results: Subjects experienced weight loss (-5.2 ± 3.2 kg, $P < 0.001$). Taken together for all subjects, there were significant decreases in all inflammation parameters. On a group level, salmon consumption was most effective, three of the four inflammation parameters decreased in the salmon group (high-sensitivity CRP = −32.0%; IL-6 = −18.4%; PGEF2α = −18.5%; all $P < 0.05$). Cod consumption decreased high-sensitivity CRP and IL-6 (−21.5 and −10.8%, respectively, both $P < 0.05$). Changes in the other two groups were not significant, which can be partly explained by the large s.d.
Conclusions: The mean concentrations of inflammation parameters decreased during a period of weight loss and dietary intervention. In our study, salmon consumption was most effective, three of the four measured inflammation parameters decreased significantly in the salmon group.
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Keywords: seafood; fish; LC n-3 PUFA; weight loss; inflammation; young adults

Introduction

Obesity-related disorders have been characterized as a state of low-grade systemic inflammation (Forsythe *et al.*, 2008).Weight loss is capable of reversing the unfavorable inflammatory profile evident in the obese state (Forsythe *et al.*, 2008). However, weight loss can be difficult to maintain, and therefore additional nutritional strategies are required (Wing and Phelan, 2005).

Studies in cell cultures and animals have shown that supplementation with long-chain n-3 polyunsaturated fatty acids (LC n-3 PUFAs) can decrease the production of inflammatory markers (Arntzen *et al.*, 1998; Bhattacharya *et al.*, 2007; Strandberg *et al.*, 2009). In addition, epidemiological research has indicated that intake of LC n-3 PUFAs is inversely associated with plasma levels of inflammatory markers (Pischon *et al.*, 2003).

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Several intervention studies have investigated whether LC n-3 PUFA can affect systemic inflammation (Blok et al., 1997; Jellema et al., 2004; Sundrarjun et al., 2004; Fujioka et al., 2006; Plat et al., 2007; Calder, 2008; Damsgaard et al., 2008). Whereas studies in patients with immune disease such as rheumatoid arthritis have shown potential of LC n-3 PUFA to reduce inflammation parameters in blood and to improve clinical outcomes (Sundrarjun et al., 2004; Calder, 2008), evidence in overweight or obese, but otherwise healthy subjects is not conclusive (Blok et al., 1997; Jellema et al., 2004; Fujioka et al., 2006; Plat et al., 2007; Damsgaard et al., 2008). Differences in inflammation parameters measured, dosing regimes, background diets, accompanying weight loss and sample sizes might explain some of the disagreements observed between the studies, as well as cytokine gene polymorphisms (Markovic et al., 2004).

It is noticeable that most intervention studies exclusively investigated men and relatively few included women as well (Blok et al., 1997; Jellema et al., 2004; Plat et al., 2007; Damsgaard et al., 2008). A recent study from Japan including healthy, mostly middle-aged subjects (59 men and 82 women) did not find changes in inflammatory markers after daily 0.60 g eicosapentaenoic acid (EPA) and 0.26 g docosahexaenoic acid (DHA) supplementation for 12 weeks (Fujioka et al., 2006). The KANWU study involving Kuopio (Finland), Aarhus (Denmark), Naples (Italy), Wollongong (Australia) and Uppsala (Sweden), a multicenter study in which 162 men and women were supplemented with a much higher dose of 3.6 g n-3 PUFA per day or placebo for 3 months, also found no significant effects on plasma PGEF2α concentration (Nälsén et al., 2006).

To investigate the associations between seafood intake and inflammatory parameters in young, overweight and obese men and women from three European countries, the current analysis was conducted. This article is a secondary analysis of the data obtained from participants of the SEAFOODplus YOUNG intervention study (Thorsdottir et al., 2007), which measured the effects of seafood consumption on weight loss. The hypothesis of this analysis was that the consumption of fatty seafood, such as, salmon and fish liver oil, decreases inflammation parameters additionally to weight loss, which participants experienced in a course of an 8-week energy-reduced diet.

Subjects and methods

Participants

A total of 324 overweight individuals (138 men and 186 women) were included in the SEAFOODplus YOUNG study (http://www.seafoodplus.org) through advertisements, 140 from Iceland, 120 from Spain and 64 from Ireland. All subjects were screened for inclusion and exclusion criteria. The inclusion criteria were body mass index ranging from 27.5 to 32.5 kg/m^2, age 20–40 years and waist circumference ⩾94 and ⩾80 cm for men and women, respectively. Exclusion criteria were weight change (±3 kg) due to a

weight-loss diet within 3 months before the start of the study, use of supplements containing n-3 fatty acids, calcium or vitamin D during the last 3 months, allergy to fish, drug treatment of diabetes mellitus, hypertension or hyperlipidemia and pregnancy or lactation. Of the subjects, 85.8% ($n=278$) completed the intervention. The study was approved by the National Bioethical Committee in Iceland (04–031), the Ethical Committee of the University of Navarra in Spain (24/2004) and the Clinical Research Ethics Committee of the Cork University Hospital in Ireland. The study followed the Helsinki guidelines, and all participating subjects gave their written consent.

Study design

This study was a randomized, controlled dietary intervention trial, which was conducted at the Landspitali University Hospital in Reykjavik, Iceland, the University College of Cork, Ireland and at the University of Navarra in Pamplona, Spain, between April 2004 and November 2005. The intervention lasted for 8 consecutive weeks, during which the subjects were instructed to follow an energy-restricted diet, 30% of the estimated energy expenditure given by Harris–Benedict equations (Cankayali et al., 2004; Salvino et al., 2004) and physical activity level (Nordic Council of Ministers, 2004) (~600 (range, 473–718) kcal/day). All participants were randomly assigned to one of the following four groups, which varied by dietary protein source and amount of LC n-3 PUFAs:

Control, no seafood (6 sunflower oil capsules per day);
Lean fish (150 g cod, 3 times per week);
Fish oil (6 capsules per day); or
Fatty fish (150 g salmon, 3 times per week).

Groups receiving sunflower or fish oil capsules were single blinded. All diets were designed to supply identical macronutrient composition: total fat (~30% of total energy), carbohydrate (~50% of total energy), protein (~20% of total energy) and dietary fiber (approximately 20–25 g). Cod diet provided LC n-3 PUFA, which resulted in a daily consumption of ~0.3 g/day; salmon diet yielded ~2.1 g LC n-3 PUFA per day; and fish oil capsules provided ~1.3 g LC n-3 PUFA per day. Other sources of n-3 fatty acids were congruent between diet groups. The salmon diet and fish oil supplement were originally formulated to provide a similar amount of n-3 fatty acids. However, farmed salmon used in this study contained twice the fat expected (US Food Composition Database, 2005), leading to a different n-3 content in the salmon diet and in the fish oil supplement.

Each subject received a detailed meal plan to follow, as well as recipe booklets and instructions to minimize the difference between diets in sources of fat (other than LC n-3 PUFA), fruit and vegetable consumption and meal frequency. The participants' physical activity level remained unchanged during the intervention. A validated food frequency questionnaire (Birgisdottir et al., 2008; Thorsdottir et al., 2008) evaluated the consumption of fish and fish oil over the

previous 4 weeks and was completed by the participants at end point. It was asked whether the participants consumed the fish and capsules provided to them and did not consume any additional fish or fish oil capsules during the study period. Consumption of fish and capsules was in good agreement with the study protocol (Birgisdottir et al., 2008; Thorsdottir et al., 2008).

Anthropometric measurements

All anthropometric measurements (such as waist circumference, body weight, body height) were carried out at baseline and at the end point of the study using standard procedures as outlined in a research protocol approved and used by all countries participating in the study. Body weight was measured with the participants dressed in light underwear on a calibrated scale (Seca 708, Seca, Hamburg, Germany). The subjects' height was measured using a calibrated stadiometer (model no. 206; Seca). For the measurement of waist circumference, the subject stood erect with the abdomen relaxed, arms at the sides, feet together and with their weight equally divided over both legs. The lowest rib margin was first located. The iliac crest was then palpated in the midaxillary line. An elastic tape was then applied horizontally midway between the lowest rib margin and the iliac crest, and tied firmly so that it stayed in position around the abdomen about the level of the umbilicus.

Biochemical measurements

Subjects were told to avoid strenuous exercise and alcohol consumption the day before the blood samples were drawn at baseline and end point. Subjects were fasting when blood samples were collected. Prostaglandin F2 alpha (PGEF2α) levels were determined using commercially available ELISA (enzyme-linked immunosorbent assay) kits (R&D Systems Inc., Minneapolis, MN, USA) after serum elution in C18 Sep-Pack columns (Isolute SPE column, Argonaut Technologies, Mid Glamorgan, UK). Glutathione reductase (GSHR) activity was measured using the test kit from Randox (Randox Laboratories Ltd., Antrim, UK). Interleukin-6 (IL-6) concentration in serum was analyzed by ELISA (R&D Systems Europe Ltd., Abingdon, UK). High-sensitivity C-reactive protein (CRP) was measured with ELISA.

Statistical analysis

Data were entered into the SPSS statistical package 11.0 (SPSS, Chicago, IL, USA). Data are described as mean ± s.d. The Wilcoxon test was used to calculate whether there were significant changes in the variables between baseline and end point. The test was used to calculated differences in categorical variables between groups. Linear models were used to calculate the effects of weight loss and diet groups on end point inflammation parameters; the models additionally included the variables country, sex and inflammation

parameters at baseline. The variables were log transformed for these analyses. Results are shown as parameter estimates, in which three diet groups are compared each with the control group. The Bs in the parameter estimates were back transformed and are shown as $1-B$, thus producing percentage differences between groups with respect to end point variables. Variances were checked using Levene's test of homogeneity, and residuals of the statistical models were checked for normality using the Kolmogorov–Smirnov test. $P \leqslant 0.05$ was regarded as statistically significant.

Results

The subjects' baseline and end point values can be seen in Table 1. There was significant weight loss in all four groups. There were significant decreases in all four investigated inflammation parameters during the 8-week weight loss program when data were viewed for all subjects together. Mean changes in all four inflammation parameters were significant in male subjects; however, only changes in IL-6 and CRP were significant in women. A more detailed description of gender differences was given in the linear models (see below).

On a group level, changes were significant for the salmon and the cod group. The s.d. for most of the inflammation parameters were huge. PGEF2α decreased in the salmon group. Significant reductions in IL-6 and CRP were found in the cod and salmon groups.

Between 43.9 and 73.9% of participants experienced reductions in inflammation parameters during the 8 weeks, depending on the inflammation parameter. Correspondingly, between 56.1 and 26.4% of participants experienced an increase in inflammation parameters. At average, 52.9, 61.1, 64.2 and 58.4 % of participants experienced reductions in GSHR, IL-6, CRP and PGEF2α, respectively. Differences between diet groups were not significant. However, there was a significant difference between genders with regard to reduction in GSHR (male = 61.0%, female = 46.8%, $P = 0.020$).

According to the linear models (Table 2), neither diet groups nor weight loss were significant predictors for end point GSHR. The male sex was a significant predictor for end point IL-6 with 14% lower IL-6 concentrations than women. The linear models for the other three inflammation parameters indicated only minor (0.7–3%) nonsignificant differences between genders. Weight loss was the only significant predictor for end point high-sensitivity CRP. Surprisingly, the cod group was a predictor for a higher end point PGEF2α with a 37.2% difference compared with the control group.

Discussion

In this intervention trial, we investigated the effects of seafood consumption and weight loss on inflammation

Table 1 Baseline and end point data of the participants

	Control		Cod		Salmon		Fish oil	
	Male (n = 32)	Female (n = 48)	Male (n = 35)	Female (n = 45)	Male (n = 42)	Female (n = 42)	Male (n = 29)	Female (n = 51)
	Mean ± s.d.	Mean ± s.d.	Mean ± s.d.	Mean ± s.d.	Mean ± s.d.	Mean ± s.d.	Mean ± s.d.	Mean ± s.d.
Age (years)	32.6 ± 4.9	31.7 ± 5.6	32.5 ± 5.1	30.4 ± 5.9	31.6 ± 5.6	30.9 ± 5.0	31.0 ± 5.9	30.9 ± 5.0
BMI (basel.)	30.1 ± 1.5	29.9 ± 1.5	30.2 ± 1.2	30.2 ± 1.6	30.5 ± 1.3	30.3 ± 1.5	29.5 ± 1.2	30.1 ± 1.7
Weight (basel.) (kg)	96.7 ± 7.2	81.7 ± 6.7	96.3 ± 7.4	84.2 ± 7.3	97.5 ± 10.6	83.3 ± 6.9	93.0 ± 7.8	80.5 ± 7.0
Weight loss (kg)	5.3* ± 3.0	3.9* ± 2.6	6.5* ± 2.8	4.4* ± 2.3	7.0* ± 3.5	3.9* ± 2.3	6.7* ± 3.6	4.4* ± 2.6
GSHR (basel.) (Units/l)**	57.4 ± 5.7	55.0 ± 11.3	59.7 ± 7.4	55.2 ± 6.3	57.0 ± 8.7	54.3 ± 6.3	60.4 ± 8.6	53.6 ± 8.1
GSHR (endp.) (Units/l)	57.3 ± 5.2	54.4 ± 7.2	56.7 ± 8.7	55.6 ± 6.7	54.4 ± 9.5	55.2 ± 6.8	58.3 ± 5.7	52.4 ± 7.4
PGEF2α (basel.) (pg/ml)**	217 ± 203	178 ± 257	223 ± 173	234 ± 266	188 ± 182	202 ± 199	270 ± 451	228 ± 321
PGEF2α (endp.) (pg/ml)	108 ± 87	131 ± 106	251 ± 224	204 ± 204	170 ± 197	148 ± 184	189 ± 141	139 ± 199
IL-6 (basel.) (pg/ml)**	1.32 ± 1.02	1.70 ± 1.59	1.59 ± 1.38	1.73 ± 1.25	1.45 ± 1.12	1.62 ± 1.07	1.94 ± 1.41	2.16 ± 3.81
IL-6 (endp.) (pg/ml)	1.17 ± 0.78	1.68 ± 1.30	1.28 ± 0.87	1.65 ± 2.34	1.14 ± 0.74	1.35 ± 0.69	1.75 ± 1.25	2.09 ± 3.52
CRP (basel.) (mg/l)**	1.77 ± 2.95	3.54 ± 4.39	2.15 ± 2.23	2.81 ± 2.73	2.41 ± 3.02	2.73 ± 2.99	3.66 ± 5.12	3.26 ± 4.60
CRP (endp.) (mg/l)	1.45 ± 1.22	2.44 ± 3.05	1.32 ± 1.40	2.49 ± 4.71	1.54 ± 1.68	1.98 ± 1.96	2.17 ± 2.36	2.70 ± 2.90

Abbreviations: basel., baseline; BMI, body mass index; CRP, C-reactive protein; endp., end point; GSHR, glutathione reductase; IL-6, interleukin-6; PGEF2α, prostaglandin F2α.
*Significant before–after differences according to Wilcoxon's signed-ranks test ($P < 0.05$).
**Significant before–after differences according to Wilcoxon's signed-ranks test ($P < 0.05$) when the data were viewed for all subjects together.

Table 2 Linear models for the prediction of end point inflammation parameters

Parameter	B	95% CI		P-value	% Diff.
Dependent variable: end point GSHR (Units/l)					
Cod[a]	−0.004	−0.019	0.011	0.597	−0.9
Salmon[a]	−0.005	−0.020	0.009	0.472	−1.2
Fish oil[a]	−0.008	−0.023	0.007	0.296	−1.8
Male[b]	−0.005	−0.016	0.007	0.437	−1.0
Weight loss (kg)[c]	0.000	−0.002	0.002	0.949	0.0
Dependent variable: end point IL-6 (pg/ml)					
Cod[a]	−0.039	−0.113	0.035	0.297	−8.6
Salmon[a]	−0.050	−0.123	0.023	0.175	−11.0
Fish oil[a]	0.006	−0.069	0.081	0.871	1.4
Male[b]	−0.068	−0.124	−0.012	**0.018**	−14.4
Weight loss (kg)[c]	0.007	−0.002	0.017	0.131	1.7
Dependent variable: end point hsCRP (mg/l)					
Cod[a]	−0.055	−0.178	0.069	0.385	−11.8
Salmon[a]	−0.018	−0.141	0.105	0.772	−4.1
Fish oil[a]	0.037	−0.087	0.162	0.557	8.9
Male[b]	0.013	−0.082	0.108	0.791	3.0
Weight loss (kg)[c]	−0.025	−0.041	−0.009	**0.003**	−5.6
Dependent variable: end point PGEF2α (pg/ml)					
Cod[a]	0.151	0.029	0.273	**0.015**	41.7
Salmon[a]	−0.037	−0.157	0.083	0.548	−8.1
Fish oil[a]	−0.004	−0.126	0.119	0.954	−0.8
Male[b]	0.003	−0.089	0.095	0.951	0.7
Weight loss (kg)[c]	0.000	−0.016	0.015	0.956	−0.1

Abbreviations: CI, confidence interval; Diff., difference; GSHR, glutathione reductase; hsCRP, high-sensitivity C-reactive protein; IL-6, interleukin-6; PGEF2α, prostaglandin F2α.
The statistical models additionally contains inflammation parameter at baseline and country.
[a]Estimated differences in end point inflammation parameters relative to the control group.
[b]Estimated differences in end point inflammation parameters relative to female.
[c]Estimated effects of weight loss on inflammation parameters.
The bold values emphasize the significant differences.

parameters in overweight and obese European young adults. The most important finding is that the mean concentrations of inflammation parameters decrease during a period of weight loss and seafood consumption. In our study, salmon consumption was most effective, and three of the four measured inflammation parameters decreased significantly in the salmon group.

In vitro studies have shown that LC n-3 PUFAs can affect inflammation processes both directly (for example, by replacing arachidonic acid as an eicosanoid substrate and inhibiting arachidonic acid metabolism) and indirectly (for example, by altering the expression of inflammatory genes through effects on transcription factor activation) (Calder, 2006; Margioris, 2009). However, results from human intervention studies are less convincing and contradicting (Blok et al., 1997; Jellema et al., 2004; Fujioka et al., 2006; Plat et al., 2007; Damsgaard et al., 2008). It has been speculated that potential beneficial effects are difficult to document because of either the lack of sensitive inflammation markers or that the beneficial effect of n-3 PUFAs occurs within tissues, and because of this, it is difficult to detect using markers of inflammation in the systemic circulation in which the changes of their concentration is diluted in the large volume of extracellular fluid (Calder, 2006).

Although the mean concentrations of inflammation parameters decreased during our intervention, individual responses to the 8-week program were quite variable. Between 43.9 and 73.9% (depending on the inflammation parameter measured) of participants experienced reductions in inflammation parameters during the 8 weeks. Obviously, other factors can override the effects of weight loss or seafood.

It has been reported that the risk of ischemic heart disease increases by 60% in individuals who have CRP levels above

3 mg/l, as compared with those who have CRP levels below 1 mg/l (Zacho et al., 2008). In our study population, 27.2% had high-sensitivity CRP levels above 3 mg/l; significant reductions during the 8 weeks were observed in the cod and in the salmon group. According to the linear model, weight loss was a significant predictor with a mean weight loss of 5.16 kg resulting in a 28.4% reduction in high-sensitivity CRP (keeping all other predictor variables unchanged).

The IL-6 values in our participants were similar to those reported elsewhere (Cava et al., 2000; Elvevoll et al., 2008). Reductions after 8 weeks were significant in the cod and salmon groups, but not in the fish oil or control group. Reductions of similar size have been previously found in a Norwegian study providing 1.1 g EPA + DHA per day (Cava et al., 2000). A smaller study in 30 patients with type II diabetes mellitus providing a lower daily dose of 0.84 g EPA + DHA for 12 weeks did not detect significant changes in IL-6 levels (De Luis et al., 2009). Interestingly, baseline data categorized by gender and the statistical model indicated lower IL-6 values for male in comparison with female participants. The linear models for the other three inflammation parameters indicated only minor (0.7–3%) nonsignificant differences between genders.

Reduced production of PGEF2α from cell cultures supplemented with n-3 PUFAs has been reported (Arntzen et al., 1998). However, the KANWU study, a recent multicenter dietary intervention trial, with a controlled fat quality in the background diet and random assignment to supplementation with fish oil (3.6 g n-3 PUFA per day) or placebo, did not detect changes in PGEF2α concentrations (Nälsén et al., 2006). In our study, significant reductions were observed only in the salmon group. Mean reductions in the fish oil and control groups were of similar size; however, the s.d. in these groups were much larger and thus changes did not reach statistical significance. According to the linear model, the cod group had end point PGEF2α concentrations (corrected for baseline) than did the other three groups (37.4–59.2% difference), which was also indicated by the descriptive data. The high LC n-3 PUFA content in the salmon and fish oil groups can explain the observed difference. However, the control group ingested even less LC n-3 PUFA than did the cod group. A possible explanation for the high PGEF2α concentration in the cod group is that at end point, the highest content of arachidonic acid in erythrocyte phospholipids was measured in the cod group, and the lowest content was measured in the salmon group (data not shown). Arachidonic acid is the substrate in cyclooxygenase-mediated PGEF2α formation. As inflammatory cells typically contain a high proportion of n-6 PUFA arachidonic acid and low proportions of other 20-carbon PUFAs, arachidonic acid is usually the major substrate for eicosanoid synthesis. Increased consumption of EPA and DHA results in increased proportions of those fatty acids in inflammatory cell phospholipids. The incorporation of EPA and DHA into human inflammatory cells occurs in a dose–response manner and is partly at the expense of arachidonic

acid (Calder, 2006). It has been shown that supplementation of the diet of healthy young men with 1.5 g arachidonic acid per day for 7 weeks resulted in a marked increase in the production of several prostaglandins and leukotriens (PGE2, LTB4) by endotoxin-stimulated mononuclear cells (Kelley et al., 1998).

In a model of human fibroblast cell culture, n-3 LC PUFA supplementations results in an elevated catalytic activity of GSHR, a response which is also confirmed by the induction of expression of mRNA for GSHR (Arab et al., 2006). In our study, changes in GSHR were negligible. According to the linear model, neither diet groups nor weight loss were significant predictors of end point GSHR activity.

Against our expectations, fish oil consumption did not significantly decrease any of the four inflammation parameters measured. Several reasons might be responsible; therefore: (1) the daily LC n-3 PUFA dose provided by fish oil capsules was only half of the amount provided by salmon; and (2) the variance between subjects in the fish oil group was large resulting in huge s.d. (see Table 1), which made it difficult to detect differences as significant. Interestingly, cod consumption resulted in a significant decrease in two inflammation parameters, although providing only 0.27 g LC n-3 PUFA per day. Possibly, other compounds in cod, for example, sulfur containing amino acids (Elvevoll et al., 2008), can affect these outcomes.

A limitation of each dietary intervention trial is the uncertainty of whether the subjects' dietary intake during the study period was as reported or prescribed. As there was intense support of the study participants by our staff, including frequent phone contact and personal visits, this risk was minimized.

The interpretation of our results is limited by the fact that despite the considerable sample size of the SEAFOODplus YOUNG study, it is likely that the study was underpowered to detect more significant differences in inflammation parameters. The observed s.d. of inflammation parameters were large. The sample size was based on weight loss, the primary end point of the SEAFOODplus YOUNG study (Thorsdottir et al., 2007). On the basis of the aimed recruitment of 320 subjects at baseline, it was estimated that a 70–80% participation allowed a detection of ∼1 kg difference in weight loss between the four diet groups, assuming a s.d. of 3 kg, a significance P-value of 0.05 and a statistical power of 0.8. In addition, factors other than of dietary origin, factors which are beyond control, can affect inflammation parameters investigated in this study (Borish, 2003).

Conclusions

The mean concentrations of inflammation parameters decreased during a period of weight loss and dietary intervention. The individual responses to the 8-week program were variable, and obviously other factors can

override the effects of weight loss and seafood. In our study, salmon consumption was most effective, with three of the four measured inflammation parameters decreased significantly in the salmon group. These findings can be explained by the high dose of LC n-3 PUFA provided by the salmon diet in our study.

Conflict of interest

The authors declare no conflict of interest.

Acknowledgements

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