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# Omega-3 fatty acid supplementation does not improve maximal aerobic power, anaerobic threshold and running performance in well-trained soccer players

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In a randomized, placebo-controlled study the effect of 10 weeks of supplementation with either 5.2 g of a concentrated fish oil triglyceride (Triomar™) enriched in omega-3 fatty acids (1.60 g/day EPA and 1.04 g/day DHA) or 5.2 g corn oil (serving as placebo) on maximal aerobic power, anaerobic threshold and running performance was assessed in 28 well-trained male soccer players (18–35 years). Supplements were given as 650-mg capsules. Capsule assignment was randomized to one omega-3 group ($n$=15), given eight Triomar™ capsules per day, and one placebo group ($n$=13), given eight capsules of corn oil per day. During the 10-week supplementation period the subjects maintained their usual diets and training regimes. Red blood cell (RBC) osmotic fragility, triglycerides and fatty acid composition in plasma were assessed before and after the supplementation period. The pre- and post-supplementation tests of maximal aerobic power, anaerobic power and running performance showed no significant difference between the two groups. Subjects in the omega-3 group had significantly reduced plasma triglycerides, rised EPA (175%) and DHA (40%) in the total lipid fraction of plasma after supplementation. RBC osmotic fragility did not change. In conclusion, the results do not support the hypothesis that endurance athletes can improve maximal aerobic performance by omega 3-fatty acid supplementation.

**T. Raastad[1], A. T. Høstmark[2], S. B. Strømme[1]**

[1]The Norwegian University of Sport and Physical Education, Laboratory of Physiology,
[2]Department of Preventive Medicine, University of Oslo, Oslo, Norway

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Truls Raastad, The Norwegian University of Sport and Physical Education, PB 4014 UH, 0806 Oslo, Norway
Tel.: +47 22 18 57 09; fax: +47 22 23 42 20
E-mail: Trulsr@Brage.Idrettshs.No

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It has been suggested by Leaf & Rauch (1) that omega-3 fatty acid supplementation may increase maximal aerobic power. This suggestion was based on data from a double blind randomized controlled trial in athletes receiving 2.1–4.2 g/day eicosapentaenoic acid (EPA) and 0.9–1.8 g/day docosahexaenoic acid (DHA) for 6 weeks. Pre- and post-supplementation determination of $\dot{V}O_{2max}$ showed that subjects in the low dose group (2.1 g/day EPA+0.9 g/day DHA, $n$=6) significantly improved $\dot{V}O_{2max}$ (from 55.8 to 61.2 ml · kg$^{-1}$ · min$^{-1}$, $P$<0.05).

In contrast, Brilla & Landerholm (2) and Warner et al. (3), measuring $\dot{V}O_{2max}$ before and after an omega-3 fatty acid supplementation period with or without training, did not find any effect of omega-3 fatty acid supplementation alone.

The classical criterion to assure that $\dot{V}O_{2max}$ is achieved is a levelling off of the oxygen uptake with increasing work load. This is usually supplemented with secondary criteria, such as a respiratory exchange ratio >1.05–1.15, and heart rate close to the subject' maximal heart rate ($HR_{max}$) (4). Neither Brilla & Landerholm (2) nor Warner et al. (3) used any of these criteria in their $\dot{V}O_{2max}$ tests. The method used by Leaf & Rauch (1) was based on predictions of $\dot{V}O_{2max}$ from the maximal percentage grade attained when running to exhaustion on a treadmill at constant speed (modified Bruce protocol). Such a test of $\dot{V}O_{2max}$ may be influenced by uncontrolled factors such as the subject's motivation and running technique.

Theoretically, omega-3 fatty acid supplementation may improve performance by increasing tissue oxygenation due to reduced blood viscosity (5–7), there-

Table 1. Physiological characteristics of the subjects, baseline values (mean±1 SD)

| | Placebo group ($n$=13) | Omega-3 group ($n$=15) |
|---|---|---|
| Age (years) | 23.6±3.3 | 23.5±2.7 |
| $V\dot{O}_{2max}$ (ml · kg$^{-1}$ · min$^{-1}$) | 62.8±4.1 | 63.6±4.1 |
| Anaerobic threshold (km · h$^{-1}$) | 10.7±0.6 | 11.0±0.9 |
| (% of $V\dot{O}_2$ max) | 80.2±6.4 | 81.2±3.4 |
| Weight (kg) | 78.9±7.5 | 78.9±8.1 |
| Triglycerides in plasma (mM) | 1.2±0.7 | 1.2±0.6 |
| Peak heart rate (beats · min$^{-1}$) | 194±5 | 189±8 |

by increasing maximal cardiac output and the peripheral blood flow.

The purpose of the present investigation was to re-investigate the hypothesis that omega-3 fatty acid supplementation increases maximal aerobic power in athletes, by using direct measurement of $V\dot{O}_{2max}$ and well-established test protocols.

## Material and methods

### Subjects

Fifty male soccer players were recruited from four teams in the national Norwegian soccer league (1.–3. division). Twenty-eight subjects completed the study. The reasons for dropping out were acute injuries during training and matches (e.g. sprained ankles, $n$=8), infections (e.g. upper respiratory track infections, $n$=5), exclusion from the soccer team ($n$=2), prior use of fish oil supplements ($n$=3) and lack of motivation ($n$=4). Soccer players were chosen since the players of each team represent a relatively homogeneous group concerning training patterns, duration and intensity. After being given full information about the practical implementation, risks and possible unpleasantness connected with the experimental protocols, the members of each team were randomly assigned to the omega-3 fatty acid group (hereafter called omega-3 group) or the placebo group.

Physiological characteristics of the subjects are presented in Table 1. To qualify for participation, the subjects had to have $V\dot{O}_{2max}$ equal to or higher than 55 ml · kg$^{-1}$ · min$^{-1}$, and did not smoke or use supplements containing omega-3 fatty acids. Initially, there were no significant differences in the physiological characteristics between the placebo group and the omega-3 group.

The omega-3 group received eight 650 mg capsules of Triomar™ per day, providing a daily dose of 3.04 g omega-3 fatty acids. The placebo group received eight identical 650 mg capsules with corn oil per day. The composition of the supplement capsules is described in Table 2. Initially, the study was intended to be double blind, but because 65% of the subjects recognized the taste of the oils, the study should be considered as single blind.

The exercise and blood tests were done 1–7 days prior to the supplementation period and the day the supplementation period ended. The supplementation period lasted on average for 10.6 weeks (range 10–12). A daily exercise diary was collected from each subject to determine training duration and intensity (total amount of training). Dietary intakes were measured prior to and at the conclusion of the supplementation period.

### Testing of $V\dot{O}_{2max}$, anaerobic threshold (AT) and running performance

Each subject went through two trials on the treadmill to determine $V\dot{O}_{2max}$, AT and running performance, i.e. before and after the supplementation period. The exercise testing followed the following protocol: (a) 10 min warming up, (b) determination of AT, (c) 5 min recovery and (d) measurement of $V\dot{O}_{2max}$, peak HR and running time until exhaustion. All runs were performed at least 3 h after the last meal.

*Determination of AT.* After warming up for 10 min at a work load of 50–55% $V\dot{O}_{2max}$, the subjects ran for 5 min on five different speeds (representing workloads ranging from 60–95% of $V\dot{O}_{2max}$), with a 1-min pause for fingertip sampling of capillary blood (50 µl) to be used for determination of blood lactate [La$^-$]. HR was registered for the last 2 min of every workload, and the average value noted. The $V\dot{O}_2$ was measured from the 3rd to the 4.30th min of every load. The speed of the treadmill was increased in steps of 1 km · h$^{-1}$, and all measurements were carried out at 3° inclination. Blood [La$^-$] was measured by a YSI Model 23L lactate analyser (USA). Non-hemolysed blood was used in all lactate measurements. AT was reached at an oxygen uptake that gave 1.5 mM higher blood [La$^-$] than those found immediately after the warming-up period (8).

Table 2. Composition of supplementation capsules

| | Corn oil | Triomar™ |
|---|---|---|
| Fatty acids (mg/capsule) | | |
| Palmitic acid (16:0) | 52 | – |
| Stearic acid (18:0) | 10 | – |
| Oleic acid (18:1, ω-9) | 150 | – |
| Linoleic acid (18:2, ω-6) | 294 | – |
| EPA (20:5, ω-3) | – | 200 |
| DHA (22:6, ω-3) | – | 130 |
| OTA (18:4, ω-3) | – | 18 |
| ETA (20:4, ω-3) | – | 7 |
| DPA (22:5, ω-3) | – | 23 |
| Total omega-3 fatty acids | – | 380 |
| Total weight (mg/capsule) | 650 | 650 |
| Total energy (kJ/capsule) | 29 | 29 |

*Measurement of* $Vo_{2max}$, *peak HR and running time until exhaustion.* All subjects rested for 5 min after having completed the AT determination. $Vo_{2max}$ was measured during running at 3° inclination according to a procedure described by Åstrand & Rodahl (9). The subjects started the test by running at the same speed, or 1 $km \cdot h^{-1}$ above the speed by which they had finished the AT determination. If the subject agreed, the speed was increased by 1 $km \cdot h^{-1}$ every min. The subjects were told that the test should lead to exhaustion within approximately 3–4 minutes. During the first test the subjects determined the increment of the speed, while at the second test the increment of the speed was exactly the same as during the first test. The classic criterion with levelling off of the oxygen uptake with increasing workload, together with a respiratory exhange ratio above 1.1, were applied to assure that $Vo_{2max}$ was achieved. Expired air was analysed using a Jaeger EOS-Sprint oxygen analyser (10). Running time until exhaustion was defined as the time elapsed from starting the $Vo_{2max}$ test untill the subject jumped off the treadmill. Peak HR was defined as the highest attained heart rate during this run. The heart rate was recorded using a Sport Tester with a memory (PE 3000 Polar Electro, Finland).

## Blood tests

Venous blood samples (5 ml) were taken at baseline and on the same day as the supplementation period ended. One milliliter was immediately used for studying osmotic fragility of RBC. The remaining 4 ml was centrifuged at 2500 r.p.m. for 10 min. Plasma was kept frozen at −80°C and used for determination of triglycerides and fatty acid composition of whole plasma lipids. For practical reasons we did not obtain fasting blood samples.

*Osmotic fragility of RBC.* For each subject 30 µl of blood was added to a series of tubes containing 4 ml of various NaCl solutions ranging from 0.90 to 0.30%. Complete hemolysis was obtained in $H_2O$. After a short incubation, hemolysis was measured as optical density at 450 nm, using a Vitatron filter photometer. Results are presented as the NaCl concentration causing 50% of complete hemolysis.

*Triglycerides and fatty acid composition.* Plasma triglycerides were determined enzymatically, using Boehringer kit reagents. Plasma fatty acid composition was measured following a procedure described by Berg et al. (11). Lipid extraction was carried out with methanol/chloroform. Gas chromatographic separation of fatty acids was performed on a Carlo Erba gas chromatograph (Milan, Italy).

## Dietary intake

A quantitative food-frequency questionnaire, described by Solvoll et al. (12), was administered to monitor changes in dietary intake. The self-administered questionnaire comprises approximately 180 food items with portion size and frequency alternatives. It is designed to cover the total diet and includes foods normally used in bread-based meals, foods and dishes for main meals, cakes and cookies, fruits, typical snack products, and beverages. Dietary supplements, such as cod liver oil, fish oil capsules and vitamin supplements, were included.

## Statistical analyses

Student's unpaired *t*-test was used to compare differences between the omega-3 group and the placebo group. Student's paired *t*-test was used to compare baseline values with post-supplementation values

Table 3. Results of the exercise tests (mean±1 SD)

| | Baseline | Post-suppl | Pre minus post | *P* |
|---|---|---|---|---|
| $Vo_{2max}$ ($ml \cdot kg^{-1} \cdot min^{-1}$) | | | | |
| Placebo | 62.8±4.1 | 61.2±2.9 | 1.6 | NS |
| Omega-3 | 63.6±4.1 | 62.4±3.7 | 1.2 | NS |
| Anaerobic threshold ($km \cdot h^{-1}$) | | | | |
| Placebo | 10.7±0.7 | 11.0±0.5 | −0.3 | <0.05 |
| Omega-3 | 11.0±0.9 | 11.3±0.8 | −0.3 | <0.05 |
| (% of $Vo_{2max}$) | | | | |
| Placebo | 80.2±6.4 | 80.8±5.6 | −0.6 | NS |
| Omega-3 | 81.2±3.4 | 81.3±3.2 | −0.1 | NS |
| Running time until exhaustion (min) | | | | |
| Placebo | 3.71±0.45 | 3.86±0.50 | −0.16 | NS |
| Omega-3 | 3.91±0.34 | 4.18±0.46 | −0.26 | NS |
| Peak HR ($beats \cdot min^{-1}$) | | | | |
| Placebo | 194±5 | 193±8 | 1 | NS |
| Omega-3 | 189±8 | 189±8 | 0 | NS |

$n$=13 (placebo) and $n$=15 (omega-3).

Table 4. Composition of fatty acids in the total lipid fraction of plasma before and after the supplementation period (mean±1 SD)

| Fatty acid | Baseline (mg · ml plasma$^{-1}$) | Post-suppl. (mg · ml plasma$^{-1}$) | P |
|---|---|---|---|
| Omega-3 group ($n$=10) | | | |
| Palmitic acid (16:0) | 0.53±0.13 | 0.49±0.07 | NS |
| Palmitoleic acid (16:1, ω-7) | 0.06±0.02 | 0.06±0.04 | NS |
| Stearic acid (18:0) | 0.22±0.06 | 0.21±0.02 | NS |
| Oleic acid (18:1, ω-9) | 0.61±0.17 | 0.49±0.10 | <0.05 |
| Linoleic acid (18:2, ω-6) | 0.88±0.12 | 0.81±0.11 | NS |
| Linolenic acid (18:3, ω-3) | 0.02±0.01 | 0.02±0.01 | NS |
| Arachidonic acid (20:4, ω-6) | 0.15±0.04 | 0.14±0.02 | NS |
| EPA (20:5, ω-3) | 0.04±0.06 | 0.11±0.08[a] | <0.01 |
| DHA (22:6, ω-3) | 0.05±0.01[b] | 0.07±0.02[a] | <0.01 |
| Placebo group ($n$=10) | | | |
| Palmitic acid (16:0) | 0.48±0.12 | 0.48±0.10 | NS |
| Palmitoleic acid (16:1,ω-7) | 0.05±0.02 | 0.05±0.02 | NS |
| Stearic acid (18:0) | 0.18±0.03 | 0.19±0.04 | NS |
| Oleic acid (18:1, ω-9) | 0.52±0.14 | 0.53±0.17 | NS |
| Linoleic acid (18:2, ω-6) | 0.81±0.15 | 0.81±0.11 | NS |
| Linolenic acid (18:3, ω-3) | 0.02±0.01 | 0.01±0.004 | <0.01 |
| Arachidonic acid (20:4, ω-6) | 0.14±0.04 | 0.14±0.03 | NS |
| EPA (20:5, ω-3) | 0.04±0.02 | 0.02±0.02[a] | NS |
| DHA (22:6, ω-3) | 0.06±0.03[b] | 0.05±0.02[a] | NS |

[a] Significant difference between groups after the supplementation period.
[b] Significant difference between groups before the supplementation period.

within each group. All results are given as mean±1 standard deviation (SD). The tests were considered significant when $P$-values were less than or equal to 0.05.

## Results

There were no significant group differences in any of the exercise variables measured in response to supplementation (Table 3). Both groups increased the running speed at AT significantly from the first to the second test.

The fatty acid supplementation did not affect the osmotic fragility of red blood cells. The NaCl solutions causing 50% hemolysis were 0.37±0.01% and 0.36±0.01% (pre), and 0.37±0.01% and 0.36±0.01% (post) for the placebo and the omega-3 group respectively. A significant drop in plasma triglycerides during supplementation was observed in the omega-3 group (1.1±0.6 mM pre, and 0.8±0.3 mM post, $P$<0.05), but not in the placebo group (1.2±0.7 mM pre, and 1.1±0.6 mM post).

A significant increase in total plasma lipid EPA, 20:5, ω-3, (175%) and DHA, 22:6, ω-3, (40%) was found in the omega-3 group (Table 4). A significant decrease in the contribution of linolenic acid,18:3, ω-3, (50%), was found in the placebo group.

### Dietary intake and physical training

Average intake of protein, fat and carbohydrates did not change during the supplementation period (Table

Table 5. Average daily intake of total energy, fatty acid, and % energy from the enery giving nutrients at the beginning (pre) and at the end (post) of the supplementation period (mean±1 SD). The contribution of energy and nutrients from the supplementation capsules is not calculated in these numbers

| Nutrient | Omega-3 group | | | Placebo group | | |
|---|---|---|---|---|---|---|
| | Pre | Post | P | Pre | Post | P |
| Energy (mJ) | 14.6±3.1 | 14.2±3.4 | NS | 14.0±2.6 | 13.1±3.4 | NS |
| Saturated fatty acids (g) | 49±22 | 46±15 | NS | 51±13 | 50±26 | NS |
| Monounsaturated fatty acids (g) | 39±13 | 41±14 | NS | 40±11 | 39±14 | NS |
| Polyunsaturated fatty acids (g) | 21±9 | 22±10 | NS | 20±8 | 19±6 | NS |
| % energy from protein | 15±2 | 15±2 | NS | 14±2 | 14±3 | NS |
| % energy from fat | 30±5 | 32±4 | NS | 32±5 | 34±6 | NS |
| % energy from carbohydrates | 55±3 | 53±4 | NS | 53±6 | 52±6 | NS |
| % energy from alcohol | 0.6±0.5 | 0.8±0.9 | NS | 0.7±0.6 | 0.8±0.7 | NS |

Table 6. Average daily intake of vitamins and minerals at the beginning (pre) and at the end (post) of the supplementation period (mean±1 SD).The contribution of nutrients from the supplementation capsules is not calculated in these numbers

| | Omega-3 group | | | Placebo group | | | |
|---|---|---|---|---|---|---|---|
| Nutrient | Pre | Post | *P* | Pre | Post | *P* | RDA (13) |
| Vitamin A (μg RE) | 2408±848 | 2433±1430 | NS | 1584±639 | 1682±995 | NS | 1000 |
| Vitamin D (μg) | 7.9±4.4 | 9.6±10.5 | NS | 7.3±5.1 | 7.5±5.5 | NS | 5 |
| Thiamin (mg) | 2.5±0.5 | 2.2±0.5 | NS | 2.2±0.4 | 1.8±0.4 | <0.05 | 1.5 |
| Riboflavin (mg) | 3.3±0.7 | 2.9±0.8 | NS | 2.9±0.5 | 2.5±0.8 | <0.05 | 1.7 |
| Niacin (mg) | 28±6 | 27±7 | NS | 25±7 | 21±6 | NS | 19 |
| Vitamin $B_{12}$ (μg) | 11±3 | 11.0±4.8 | NS | 9.8±3.6 | 9.5±4.2 | NS | 2 |
| Folate (μg) | 271±66 | 235±52 | <0.05 | 220±52 | 194±48 | <0.05 | 200 |
| Vitamin C (mg) | 150±73 | 106±59 | <0.05 | 113±49 | 92±45 | NS | 60 |
| Calcium (mg) | 1541±360 | 1347±457 | NS | 1487±434 | 1350±408 | NS | 800 |
| Iron (mg) | 18±5 | 17±4 | NS | 16±2 | 14±4 | <0.05 | 10 |
| Magnesium (mg) | 579±117 | 552±137 | NS | 529±142 | 451±122 | <0.05 | 350 |

5). In particular, there was no change in the intake of polyunsaturated fatty acids.

The intake of vitamin C, folate, iron, magnesium, thiamin, and riboflavin showed small but significant changes during the supplementation period (Table 6). There was a significant difference between the omega-3 group and the placebo group in the intake of vitamin A and folate at baseline and in the intake of tiamin, niacin and iron at the end of the supplementation period. The average intake of all nutrients were for both groups above the RDA (13) values (except folate for the placebo group, post-test).

The exercise diaries showed no significant differences between the two groups in training duration and intensity (total amount of training) (Table 7).

## Discussion

Contrary to the finding of Leaf & Rauch (1), that their low dose group had a significant increase in estimated $\dot{V}O_{2max}$, we did not observe any effect of omega-3 fatty acid supplementation on maximal aerobic power, anaerobic threshold or running performance.

The method used by Leaf & Rauch (1) for determination of $\dot{V}O_{2max}$ is based on constant treadmill speed with gradient increaments every 3 min until exhaustion. There is reason to believe that the result from such an all-out test may have been influenced by the subjects' motivation, and thereby the estimation of $\dot{V}O_{2max}$. This suggestion is supported by the fact that Leaf & Rauch observed an average increase in maximal heart rate of 8 beats · $min^{-1}$ from the pre- to the post-test in the low dose group, and that they surprisingly did not observe a significant increase in $\dot{V}O_{2max}$ in the high dose group (4.2 g EPA/day and 1.8 g DHA/day).

Theoretically, there are reasons to suggest that omega-3 fatty acid supplementation could affect aerobic performance. Several investigators have observed reduced blood viscosity after omega-3 fatty acid supplementation (5–7). Blood viscosity seems to be an important determinant of maximal cardiac output and peripheral blood flow, which are determinants of maximal aerobic capacity (14–17).

Some studies have shown that well-trained endurance athletes have reduced blood viscosity compared with more sedentary subjects (17–19). Ernst (20) observed a reduction in blood viscosity of athletes during a period of heavy endurance training. Thus, the positive effect of omega-3 fatty acid supplementation on blood viscosity seen in normal populations might perhaps be less marked in endurance-trained populations because of their lower baseline values.

Another possible explanation for the negative outcome of this study is that the effect of omega-3 fatty acid supplementation on maximal aerobic power was so small that it was impossible to detect with the number of subjects completing the study. With our material we had a 80% chance to find a significant supplementation effect if the difference in $\dot{V}O_{2max}$ had been about 2 ml · $kg^{-1}$ · $min^{-1}$ (21).

Table 7. Time spent on training at five different intensities during the 10-week supplementation period (mean±1 SD)

| | Intensity 1 60–75% of $HR_{MAX}$ | Intensity 2 75–85% of $HR_{MAX}$ | Intensity 3 85–90% of $HR_{MAX}$ | Intensity 4 90–95% of $HR_{MAX}$ | Intensity 5 >95% of $HR_{MAX}$ | Total training time |
|---|---|---|---|---|---|---|
| Placebo (*n*=13) | 1160±486 min | 1209±407 min | 654±266 min | 634±514 min | 61±52 min | 3934±763 min |
| Omega-3 (n=15) | 1188±898 min | 1160±443 min | 700±309 min | 723±455 min | 86±78 min | 4178±610 min |

The increase in running speed at AT observed in both groups was probably a result of better running technique. This is supported by the fact that the subjects consumed less oxygen at the same submaximal running speed (minus 1.3 $ml \cdot kg^{-1} \cdot min^{-1}$) during the second test compared with the baseline values.

Not surprisingly, we observed a reduction in triglycerides in the omega-3 group during the supplementation period. This is the most common effect after omega-3 fatty acid supplementation both in normal subjects (22–25) and in hyperlipidemic subjects (3, 26–28). This effect was observed in the present study in spite of the fact that we were unable to obtain fasting blood samples. The hypotriglyceridemic effect of omega-3 fatty acids appears to be caused primarily by an inhibition of very low density lipoprotein–triglyceride synthesis (29), probably with EPA (20:5, ω-3) as a major inhibitor of this synthesis (30).

The incorporation of EPA (20:5, ω-3) and DHA (22:6, ω-3) in the total lipid fraction of plasma suggests that the subjects took the capsules throughout the supplementation period. The fact that we did not observe any differences in training patterns, intensity and duration between the placebo group and the omega-3 group suggests that the results were not biased by variation in physical training.

We observed significant changes in the average intake of some nutrients during the investigation. But, because the average intake of the measured nutrients seemed to be close to what is considered as optimal at both time-points, and for both groups, it is reasonable to assume that the results were not affected by the diet.

In conclusion, 10 weeks of omega-3 fatty acid supplementation did not result in any detectable effects on maximal aerobic power, anaerobic threshold or running performance in well-trained soccer players.

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