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## Fish oil reduces heart rate and oxygen consumption during exercise

### Abstract

Dietary omega-3 polyunsaturated fatty acids (PUFAs) are readily incorporated into heart and skeletal muscle membranes where, in the heart, animal studies show they reduce O<sub>2</sub> consumption. To test the hypothesis that omega-3 PUFAs alter O<sub>2</sub> efficiency in humans, the effects of fish oil (FO) supplementation on O<sub>2</sub> consumption during exercise were evaluated. Sixteen well-trained men (cyclists), randomly assigned to receive 8 x 1 g capsules per day of olive oil (control) or FO for 8 weeks in a double-blind, parallel design, completed the study (control: n = 7, age 27.1 +/- 2.7 years; FO: n = 9, age 23.2 +/- 1.2 years). Subjects used an electronically braked cycle ergometer to complete peak O<sub>2</sub> consumption tests (VO<sub>2peak</sub>) and sustained submaximal exercise tests at 55% of peak workload (from the VO<sub>2peak</sub> test) before and after supplementation. Whole-body O<sub>2</sub> consumption and indirect measurements of myocardial O<sub>2</sub> consumption [heart rate and rate pressure product (RPP)] were assessed. FO supplementation increased omega-3 PUFA content of erythrocyte cell membranes. There were no differences in VO<sub>2peak</sub> (mL kg<sup>-1</sup> min<sup>-1</sup>) (control: pre 66.8 +/- 2.4, post 67.2 +/- 2.3; FO: pre 68.3 +/- 1.4, post 67.2 +/- 1.2) or peak workload after supplementation. The FO supplementation lowered heart rate (including peak heart rate) during incremental workloads to exhaustion (P < 0.05). In addition, the FO supplementation lowered steady-state submaximal exercise heart rate, whole-body O<sub>2</sub> consumption, and RPP (P < 0.01). Time to voluntary fatigue was not altered by FO supplementation. This study indicates that FOs may act within the healthy heart and skeletal muscle to reduce both whole-body and myocardial O<sub>2</sub> demand during exercise, without a decrement in performance.

### Keywords

Exercise, fish oil, heart rate, oxygen consumption, rate pressure product

### Disciplines

Arts and Humanities | Life Sciences | Medicine and Health Sciences | Social and Behavioral Sciences

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## **Fish oil reduces heart rate and oxygen consumption during exercise**

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**Running Title:** Fish oil, heart rate and oxygen in exercise

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## ABSTRACT

Dietary omega-3 polyunsaturated fatty acids (PUFA) are readily incorporated into heart and skeletal muscle membranes where, in the heart, animal studies show they reduce O<sub>2</sub> consumption. To test the hypothesis that omega-3 PUFA alter O<sub>2</sub> efficiency in humans, the effects of fish oil supplementation on O<sub>2</sub> consumption during exercise were evaluated. Sixteen well-trained men (cyclists), randomly assigned to receive 8x1g capsules per day of olive oil (control) or fish oil (FO) for 8 weeks in a double blind parallel design, completed the study (control n=7, age 27.1±2.7y; FO n=9, age 23.2±1.2y). Subjects used an electronically braked cycle ergometer to complete peak O<sub>2</sub> consumption tests (VO<sub>2peak</sub>) and sustained submaximal exercise tests at 55% of peak workload (from the VO<sub>2peak</sub> test), before and after supplementation. Whole body O<sub>2</sub> consumption and indirect measurements of myocardial O<sub>2</sub> consumption (heart rate and rate pressure product) were assessed. FO supplementation increased omega-3 PUFA content of erythrocyte cell membranes. There were no differences in VO<sub>2peak</sub> (ml/kg/min) (control: *pre*; 66.8±2.4 *post*; 67.2±2.3 : FO: *pre*; 68.3±1.4 *post*; 67.2±1.2) or peak workload following supplementation. The FO supplementation lowered heart rate (including peak heart rate) during incremental workloads to exhaustion (p<0.05). In addition, the FO supplementation lowered steady state submaximal exercise heart rate, whole body O<sub>2</sub> consumption and rate pressure product (p<0.01). Time to voluntary fatigue was not altered by fish oil supplementation. This study indicates that fish oils may act within the healthy heart and skeletal muscle to reduce both whole body and myocardial O<sub>2</sub> demand during exercise, without a decrement in performance.

**Key words:** fish oil, heart rate, rate pressure product, oxygen consumption, exercise.

## INTRODUCTION

Dietary fish oil supplementation alters myocardial membrane fatty acid composition<sup>1,2</sup> and modifies heart function in animals, providing direct antiarrhythmic effects<sup>2-4</sup> and improved pumping performance<sup>5,6</sup>. In addition, fish oil reduces myocardial O<sub>2</sub> consumption without any decrement in

cardiac work, indicating an increase in cardiac energy efficiency <sup>6</sup>. In humans the direct effects of dietary fish oil in the myocardium prevent fatal arrhythmia <sup>7,8</sup>, and reduce resting heart rate <sup>9,10</sup>. A small but consistent reduction in heart rate has recently been distilled from many clinical trials through meta-analysis <sup>11</sup> and indirectly supports the notion that myocardial oxygen consumption is decreased when fish is present in the diet. In view of the influential role of long chain omega-3 PUFA, found primarily in marine oils, in modifying cardiac function in disease states and preventing or treating cardiovascular disease, it is likely that they may play an important role in the healthy heart, including modifying myocardial O<sub>2</sub> demand during aerobic exercise.

Erythrocyte membrane content of the omega-3 polyunsaturated fatty acids (PUFA) eicosapentaenoic acid (EPA) + docosahexaenoic acid (DHA) is a known marker as a risk factor for cardiovascular disease being reflective of myocardial membrane composition <sup>12,13</sup>. Dietary fats are also reflected in skeletal muscle membrane fatty acid composition <sup>1,14,15</sup>. Compared to many other organs, both heart and skeletal muscle membranes incorporate high levels of DHA with fish oil feeding <sup>1,15</sup>. Increased DHA levels in skeletal muscle membrane modifies muscle function, increasing insulin sensitivity <sup>16</sup> and possibly substrate preference <sup>17</sup>. To our knowledge, there has been no published work that investigates if changes in membrane fatty acid composition are associated with altered skeletal muscle O<sub>2</sub> consumption as previously seen in the heart <sup>6</sup>.

It was the premise of this study that dietary fish oil may have direct effects on human heart function in well trained males to enhance cardiac energy efficiency during exercise and in addition, possibly enhance efficiency of oxygen use in exercising skeletal muscle.

## **SUBJECTS AND METHODS**

Twenty, healthy well-trained male cyclists were recruited and sixteen completed the dietary trial. Two subjects were eliminated from the study when their training patterns changed during the study and two subjects withdrew due to injury unrelated to the study. Cyclists were the subjects of choice due

to their extended periods of steady state training and the specificity of the cycle ergometer to this population. Furthermore, the highly trained characteristics of this group ensured central cardiovascular limitation rather than peripheral muscular fatigue during peak O<sub>2</sub> consumption testing. The study was approved by the University of Wollongong, Human Research Ethics Committee. All subjects completed an informed consent.

### **Study Design**

Subjects were randomly allocated, in a double blind manner to two groups and supplemented with 8 × 1g capsules per day of either olive oil (control) or omega-3 PUFA-rich tuna fish oil (FO) (Numega, Clover Corporation, Sydney, Australia) for 8 weeks. Some anthropometric characteristics of those completing the trial were: Control age: 27.1±2.7y; height: 177.3±1.3cm; weight: 75.2±2.9kg; skinfolds (sum of 7): 45.1±1.4mm or FO age: 23.2±1.2y; height: 176.5±1.6cm; weight: 69.3±1.9kg; skinfolds: 51.8±2.3mm. The relative composition of fatty acids in the capsules was quantified using gas chromatography (Table 1). For the FO group this provided 3.2g of omega-3 PUFA per day (0.8g of eicosapentaenoic acid (EPA) and 2.4g of docosahexaenoic acid (DHA)). Compliance was verified through capsule count and red blood cell fatty acid analysis.

### **[Table 1. near here]**

To limit influences of training variation, dietary supplementation and testing took place during a non-competitive period of the Australian cycle calendar. All subjects were asked to provide records of their training, specifically their resting morning heart rate and the duration, in minutes, spent at particular training heart rates (as indicated by their personal heart rate monitors). A mean weekly training impulse representing intensity and duration of training, was calculated according to the following formula <sup>18</sup>.

$$\text{Training Impulse} = \text{Duration (min)} \times (\text{HR}_{\text{ex}} - \text{HR}_{\text{rest}}) / (\text{HR}_{\text{max}} - \text{HR}_{\text{rest}})$$

Where: Duration = Time spent in steady state exercise;  $HR_{ex}$  = Exercise heart rate during training;  
 $HR_{rest}$  = Resting heart rate;  $HR_{max}$  = Maximum heart rate recorded in the laboratory.

Peak  $O_2$  consumption tests were conducted prior to and after 8 weeks of supplementation, each followed by a sustained submaximal cycling test to exhaustion one week later. Subjects refrained from high intensity cycling for 48h prior to testing. They were instructed to arrive at the laboratory on each testing day in a euhydrated (20mL fluid/kg body weight) and fed state (2g carbohydrates/kg body weight). Environmental room conditions were maintained at 18-20<sup>0</sup> C and 45-55% relative humidity.

An electronically braked cycle ergometer (Lode Ergo, Netherlands) was used for exercise testing. Heart rate was recorded using a heart rate monitor (Polar, NY, USA). Respiratory data was collected using the Sensor Medics Gas Analysis System (2900c) (Sensor Medics Corporation, CA, USA) over a 20s rolling average. Ratings of perceived exertion were made using the 15-point Borg scale <sup>19</sup>. Systolic blood pressure was recorded using the automatic oscillometric Dinamap<sup>TM</sup> Vital Signs Monitor (Johnson and Johnson Pty Ltd, North Ryde, Sydney). To reduce operator error, all measurements of blood pressure during exercise were taken on the subject's left arm, by the same person. Subjects were asked to relax the arm in a vertical plane next to their body.

### ***Peak oxygen consumption test***

Subjects commenced with a 10min warm up period at a workload of 150 watts. The workload was incremented by 2 watts every 3s until a workload was reached at which the subject could not maintain a peddling cadence greater than or equal to 40rpm. This point was defined as voluntary exhaustion. Peak  $O_2$  consumption, peak workload, submaximal heart rates and peak heart rate, and submaximal ratings of whole body perceived exertion were assessed. Blood pressure was not recorded during this test due to the demanding nature of the exercise and the likely interference with performance. The peak  $O_2$  consumption test was repeated after dietary supplementation.

### ***Submaximal cycling test***

Workload for the submaximal exercise test was set at 55% of the peak workload derived from the pre-supplementation peak O<sub>2</sub> consumption test. This same absolute workload was used following supplementation, allowing a direct comparison to be made for pre to post-supplementation measurements of whole body O<sub>2</sub> consumption, heart rate and systolic blood pressure. Subjects were instructed to continue to cycle until voluntary exhaustion. Pilot testing in our laboratory indicated that this level of steady state work could be maintained for at least 60min, thus ensuring that all tests represented prolonged steady state exercise. Subjects consumed water *ad lib*, throughout the tests and cycled to voluntary exhaustion.

All data were collected for the first 60min of submaximal exercise and during the final minutes before voluntary exhaustion. Heart rate was collected continuously (averaged over 5s). Systolic blood pressure, respiratory data and ratings of perceived exertion were taken every 10min and the rate pressure product (RPP) (product of the systolic blood pressure and heart rate) calculated.

### ***Fatty acid analysis***

Omega-3 PUFA level in red blood cell membranes was used as a marker of general tissue omega-3 PUFA uptake following dietary supplementation<sup>12,13,20</sup>. Prior to and following supplementation, blood samples were collected from the median cubital vein into EDTA and stored on ice until separation. Cells were sedimented by centrifugation at 4<sup>0</sup>C, plasma was removed and frozen, buffy coat was removed and the red blood cells stored at -80<sup>0</sup>C.

For analysis, red blood cells were thawed on ice, lysed in 10mL of Tris-buffer (10mM, pH=7.4), centrifuged at 4<sup>0</sup>C to separate red cell membranes. Membranes fatty acids were directly transesterified to produce methyl esters<sup>21</sup>. The relative proportions of fatty acids incorporated into the red blood cell membranes were analysed by flame ionization gas chromatography (Shimadzu

GC-17A, 30m x 0.25mm internal diameter capillary column). Fatty acids were identified by comparison with known standards and reported as relative percent by weight.

### ***Statistical analysis***

Values are presented as mean  $\pm$  SEM. A two-way analysis of variance (ANOVA) with repeated measures was used to detect significance between groups over time pre and post supplementation and within groups as an effect of supplementation. Differences between individual means, within and between dietary groups was further analysed by post-hoc Bonferroni analysis. Statistical significance was accepted at  $p < 0.05$  level. Statistical analysis was performed using Statistix for Windows (Analytical Software, Tallahassee, Florida, USA).

## **RESULTS**

### ***Subjects***

There was no significant difference in fish consumption between the groups prior to dietary supplementation (Control:  $3.3 \pm 0.3$ ; FO:  $2.8 \pm 0.3$  fish meals per month). The mean training impulse (TI) per week was consistent within groups during the 8 weeks supplementation (data not shown) and there was no significant difference between dietary groups (Control:  $512 \pm 26$ ; FO:  $462 \pm 99$  TI/week).

### ***Red blood cell membrane fatty acids***

There were no significant differences in red blood cell fatty acid composition prior to supplementation. There were no changes in fatty acid composition after control supplementation. Following FO supplementation there were significant increases in membrane fatty acid levels of DHA (22:6n-3), the sum of PUFA and the sum of omega-3 PUFA ( $p < 0.05$ ) (Table 2). The FO supplementation produced no change in the polyunsaturated/saturated (P:S) ratio (Control pre: 1.05; post: 1.06; FO pre: 1.02; post 1.07) but significantly decreased the (n-6)/(n-3) ratio (Control pre: 1.48; post: 1.44; FO pre: 1.6; post 1.17) ( $p < 0.05$ ).

**[Table 2. near here]**

### ***Peak Oxygen Consumption Test***

There were no differences in peak O<sub>2</sub> consumption or peak workload between groups prior to or after supplementation or within groups after supplementation (Table 3). Heart rate increased with increasing workload during the test. There was no significant difference in peak heart rate between control and FO groups prior to supplementation (Table 3). The heart rate response during increasing workload was not different after control supplementation, however after the FO supplementation the heart rate was significantly lower during increasing workload ( $p < 0.001$ ) and a significant decrease in peak heart rate was seen within the FO group following supplementation ( $p < 0.05$ ) (Figure 1).

Supplementation had no effect on ratings of perceived exertion (legs, chest, whole body), reported at four sub-maximal workloads (data not shown).

**[Table 3. near here] [Figure 1. near here]**

### ***Submaximal cycling test***

There was no significant difference during the first 60min of the test in mean whole-body submaximal O<sub>2</sub> consumption between groups prior to supplementation (Control Pre:  $49.2 \pm 2.0$ ; FO Pre:  $51.1 \pm 1.1$  ml/kg/min). Whole body O<sub>2</sub> consumption in the control group was not significantly different after supplementation. Whole body O<sub>2</sub> consumption during exercise was reduced in the FO group and the change was significantly different to the control group ( $p < 0.01$ ) (Figure 2). There was no significant difference in mean respiratory exchange ratio between groups prior to supplementation or within groups after supplementation (Control Pre:  $0.91 \pm 0.02$ ; Post:  $0.91 \pm 0.03$ ; FO Pre:  $0.92 \pm 0.02$ ; Post:  $0.92 \pm 0.03$ ).

**[Figure 2. near here]**

The FO group had significantly lower heart rate throughout sustained submaximal exercise ( $p < 0.01$ ) after supplementation compared with before (Figure 3). This reduction was such that the mean heart

rate of the FO group over the entire exercise period was significantly lower than the control group after supplementation ( $p < 0.05$ ) (Table 4). Systolic blood pressure showed no significant change with supplementation (data not shown).

**[Figure 3. near here] [Table 4. near here]**

The rate pressure product (heart rate x systolic blood pressure) during the first 60min of sustained submaximal exercise and the mean of the entire submaximal exercise trial were significantly lower after FO supplementation (Figure 4) and the mean change over time was significantly different to the control group ( $p < 0.01$ ) (Figure 4). Table 4 shows absolute rate pressure products. The FO group approached significantly lower absolute values compared to the controls after supplementation ( $p = 0.08$ ). There were no differences between groups in time to fatigue before or after supplementation and no significant differences after supplementation within groups (Table 4)

**[Figure 4. near here]**

Ratings of perceived exertion (legs, chest, whole body) were significantly higher in the fish oil group prior to supplementation but were not significantly different afterwards. Both groups reported changes in RPE, however, within the FO group there were significantly larger RPE changes reported overtime for chest and whole body ( $p < 0.01$ ) and this approached significance for legs ( $p = 0.07$ ) (data not shown).

## **DISCUSSION**

After dietary fish oil supplementation, already well-trained athletes exhibited lower heart rates throughout exercise across a wide range of workloads, with no change in peak oxygen consumption or peak workload. The heart rate, rate pressure product and whole body  $O_2$  consumption were also reduced throughout 60min of sustained submaximal exercise after FO supplementation. Thus, the present study indicates that efficiency of  $O_2$  use by the heart and skeletal muscle is increased after fish oil supplementation in humans as it is in rat hearts<sup>6,22</sup>.

Dietary supplementation with fish oil increased the incorporation of omega-3 PUFA into red blood cells to a level exceeding that observed with long term habitual intake of 0.5g per day <sup>23</sup>. Such habitual intake and accompanying red cell omega-3 PUFA levels are associated with reduced relative risk of primary (arrhythmic) cardiac arrest. The antiarrhythmic effects of fish oil have been linked to direct effects in the heart <sup>2,4,24,25</sup> dependent on myocardial membrane incorporation of omega-3 fatty acids, especially DHA. Myocardial membrane omega-3 PUFA composition is strongly associated with red cell membrane composition in both humans and animals <sup>12,13,15</sup>. Myocardial cells incorporate higher levels of omega-3 fatty acids than do red blood cells <sup>15,24</sup> or serum phospholipids <sup>26</sup>. Thus, the increase in red blood cell incorporation in cyclists not only confirmed compliance to the supplementation regimen but may be indicative of even greater incorporation into skeletal muscle and myocardial membranes. Human skeletal muscle incorporates increased omega-3 PUFA with fish oil consumption <sup>27</sup> and several studies have shown that physical training itself is associated with increased human skeletal muscle omega-3 PUFA content independent of diet <sup>28,29</sup>. The DHA content correlates with the muscle content of type I, slow oxidative fibres <sup>29</sup> which are more efficient in their use of oxygen, fatigue resistant and display higher insulin sensitivity <sup>28,29</sup>. The hypothesis that fish oil supplementation could lead to altered cardiac or skeletal muscle energy efficiency was dependent upon changes occurring in their membrane omega-3 PUFA composition. The observed changes in red blood cell membrane composition suggest that such changes were achieved. Importantly, in all of these young well-trained subjects, whether supplemented with fish oil or not, the erythrocyte omega-3 index (sum of EPA + DHA) was above the low levels proposed to represent heightened risk of cardiac disease and above the minimum levels associated with cardio-protection <sup>12,13</sup>.

Previously a study reported that peak O<sub>2</sub> consumption was increased following fish oil feeding <sup>30</sup>. The results of the current investigation do not concur with this finding. In fact, no change in peak O<sub>2</sub> consumption is consistent with two later studies also showing effect of fish oil supplementation <sup>31,32</sup>. Importantly, the present investigation used cyclists during a controlled stage of their training season,

with peak O<sub>2</sub> consumption indicative of well-trained individuals<sup>33</sup>. This reduced the possibility of changes in peak O<sub>2</sub> consumption occurring as an artifact of training, which could not be ruled out in the earliest study<sup>30</sup>. Furthermore, all subjects in the present investigation were familiar with and highly competent in using the cycle ergometer, as the testing modality, unlike the soccer players in an earlier investigation<sup>32</sup>.

Fish oil supplementation reduces resting heart rate in man<sup>11</sup> not unlike the low resting<sup>34</sup> and exercising heart rates<sup>35</sup> associated with regular physical training. While higher parasympathetic activity is in part responsible for the lower resting heart rates of trained athletes<sup>34</sup>, intrinsic beat rate may also be reduced, which may be attributable to altered myocardial cell metabolism leading to more efficient energy generation or utilization<sup>35</sup>. Studies of the isolated spontaneously beating heart show that fish oil can indeed reduce intrinsic heart rate<sup>2</sup> and increase cardiac energy efficiency when the heart is paced at a constant rate<sup>6</sup>. Skeletal muscle cellular metabolism is also altered with exercise<sup>36</sup>. In man, myocardial O<sub>2</sub> consumption correlates strongly with heart rate but most strongly with the product of heart rate and aortic systolic pressure (also termed the index of cardiac effort)<sup>37</sup>. In fish oil supplemented subjects, the lower heart rate and rate pressure product observed throughout both peak and submaximal exercise in this study suggest that myocardial O<sub>2</sub> consumption was reduced.

An additional, often small but consistent effect of fish oil is to reduce resting heart rate, in man<sup>38</sup> and in rats<sup>5,39</sup>. Regular fish consumption or erythrocyte DHA levels indicative of fish intake were associated with slowed heart rate at rest in a large epidemiological study of untrained adult males<sup>9</sup> and in both stand alone studies<sup>10,40</sup> and distilled meta-analysis of randomised controlled dietary supplementation trials<sup>11</sup>. In addition, heart rate is reduced in association with omega-3 PUFA supplementation not only in rat isolated spontaneously beating hearts<sup>2</sup>, but also in cardiac transplant patients in the absence of vagal tone, which excludes a neurogenic mechanism<sup>41</sup>. Furthermore, reduction in post-exercise recovery heart rate in this patient group is also improved

following fish oil supplementation <sup>42</sup>. The reduction in heart rate during exercise has been found in trained horses across a range of treadmill speeds <sup>43</sup> in a manner very similar to the present study using trained cyclists. A slowed heart rate and associated prolonged cardiac diastole would provide more time per minute for coronary artery perfusion and myocardial oxygenation. Moreover, with heart rate proposed as a risk factor for cardiovascular mortality <sup>44</sup>, its association with risk of death or hospitalisation in heart failure patients <sup>45</sup> provides further links to omega-3 PUFA. For in addition to reducing heart rate <sup>11</sup>, fish oil supplementation reduces mortality in heart failure patients <sup>46</sup> and fish consumption is associated with lower primary incidence of heart failure <sup>47</sup>. The slowed heart rate and improved oxygen efficiency together with the reduced exercise-induced muscle damage and inflammation associated with omega-3 PUFA <sup>48</sup> may also be important modulators of heart fatigue and even damage that is suggested to occur during extended exercise <sup>49</sup>.

The reduced whole body O<sub>2</sub> consumption seen after fish oil supplementation may reflect reduced (more efficient) use of O<sub>2</sub> by active skeletal muscle (the largest consumer of oxygen during exercise). Skeletal muscle shares the capacity of cardiac tissue to avidly incorporate omega-3 PUFA, particularly DHA, well above circulating levels <sup>1,15</sup>. In animals and man, unsaturation of skeletal muscle membrane increases insulin sensitivity <sup>16,28,29</sup> and results in an increased ability of skeletal muscle cells to take-up glucose, which might be expected to maintain contractile performance. Long chain omega-3 PUFA can also shift substrate preference to glucose in skeletal muscle <sup>17</sup> and since energy derived from the breakdown of 1 mole of glucose requires less O<sub>2</sub> than the equivalent amount derived from fat <sup>50</sup>, this may explain the reduced whole body O<sub>2</sub> consumption. However, the unchanged respiratory exchange ratio is contradictory. Alternatively, fish oil has been shown to improve calcium handling in the heart and reduce O<sub>2</sub> consumption attributed to excessive sarcoplasmic reticulum calcium cycling <sup>6</sup>. The sarcoplasmic reticulum membrane also avidly incorporates DHA when fish oil is present in the diet <sup>51</sup>. Human skeletal muscle has been calculated to use up to 37% of energy in the Ca<sup>++</sup> cycling of activation and relaxation <sup>52</sup>. Improved calcium

handling by skeletal muscle sarcoplasmic reticulum with fish oil supplementation could be a factor contributing to reduced O<sub>2</sub> demand of contracting skeletal muscle as in the heart <sup>25</sup>.

Despite requiring the subjects to cycle submaximally until voluntary fatigue, this study did not attempt to link fish oil supplementation to altered exercise performance. Time to fatigue as used in the present study carries a high degree of within-subject variability and therefore is considered a weak performance indicator <sup>53</sup> and a more suitable indicator such as the cycling time trial would be better indicator of fatigue during endurance performance <sup>54,55</sup>. However, it is unlikely that the fish oil induced improved oxygen efficiency could lead to measurable improvement in endurance performance because this type of exercise is not entirely oxygen limited <sup>56</sup> and elite performance is multi-factorial in nature <sup>57</sup>.

## **Conclusion**

This study showed that supplementing healthy, well-trained male subjects with fish oil over 8 weeks, significantly lowered heart rate, rate pressure product and whole body O<sub>2</sub> consumption during exercise. Importantly, the peak levels of whole body O<sub>2</sub> consumption and workload were not influenced despite the reduced peak heart rate. These results confirmed that fish oil supplementation reduces myocardial O<sub>2</sub> demand during exercise. Furthermore, there is evidence that skeletal muscle O<sub>2</sub> consumption is also reduced following fish oil supplementation.

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**Legends to figures:**

**Figure 1:** Change in heart rate response for control ( $\Delta$ ) and FO ( $\circ$ ) groups during incrementing workloads to peak performance following 8 weeks dietary supplementation. \*  $p < 0.01$ . 2-way ANOVA. Mean  $\pm$  SEM.

**Figure 2:** Change in whole body  $O_2$  consumption within control ( $\Delta$ ) and FO ( $\circ$ ) groups during submaximal cycling test following 8 weeks dietary supplementation. Measurements are shown over the first hour of exercise (\* $p < 0.01$  2-way ANOVA), and as a mean value representing the submaximal cycling test (\*\*  $p < 0.05$  1-way ANOVA). Mean Control ( $\blacktriangle$ ) and FO ( $\bullet$ ). Mean  $\pm$  SEM.

**Figure 3:** Change in heart rate response for control ( $\Delta$ ) and FO ( $\circ$ ) groups during submaximal cycling test following 8 weeks dietary supplementation. Measurements are shown over the first hour of exercise (\* $p < 0.01$  2-way ANOVA), and as a mean value representing the submaximal cycling test (\*\*  $p < 0.05$  1-way ANOVA). Mean Control ( $\blacktriangle$ ) and FO ( $\bullet$ ). Mean  $\pm$  SEM.

**Figure 4:** Change in rate pressure product response for control ( $\Delta$ ) and FO ( $\circ$ ) groups during submaximal cycling test following 8 weeks dietary supplementation. Measurements are shown over the first hour of exercise (\* $p < 0.05$  2-way ANOVA), and as a mean value representing the submaximal cycling test (\*\*  $p < 0.05$  1-way ANOVA). Mean Control ( $\blacktriangle$ ) and FO ( $\bullet$ ). Mean  $\pm$  SEM.