Original Communication

Fish Oil Supplementation, Resting Blood Flow and Markers of Cellular Metabolism During Incremental Exercise

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Abstract: Dietary supplementation of fish oils (n-3 PUFA) have been observed to affect insulin action and hence metabolism, affecting the ability to carry out work. Here we examine the effects of fish oil supplementation in conjunction with a glucose load during exertion, on markers of substrate utilization. A pre-test, post-test design was performed on ten healthy young males to assess the effects of 4 weeks fish oil supplementation on muscle metabolism during incremental exertion. Breath-by-breath analysis for respiratory exchange ratio (RER) along with blood lactate and blood glucose were determined at baseline, during exercise following an acute glucose bolus (10 % solution at 4 mL/kg/bw), and again following supplementation of 4.2 g.day-1 (2.2 g EPA, 1.4 g DHA). To examine the effect of fish oil on blood flow, Doppler ultrasound was used to assess femoral blood flow at rest. Following consumption of fish oils, exercising blood glucose and RER were seen to change significantly (4.66 ± 0.44 vs. 4.58 ± 0.31 mmol.L-1 and 0.97 ± 0.03 vs. 0.99 ± 0.04; $p < 0.05$). Resting femoral arterial blood flow was seen to increase significantly ($p < 0.05$) pre- to post- test; $0.26 \pm 0.02 – 0.30 \pm 0.03$ L.min-1. Specific population groups such as those undertaking high-intensity exercise, and clinical groups such as intermittent claudicants, may benefit from the effects of fish oil supplementation.

Key words: omega 3, blood flow, exercise, carbohydrate, RER

Introduction

Eicosapentaenoic acid (EPA) is an n-3 polyunsaturated fatty acid (n-3 PUFA), widely known as omega-3, and is obtained naturally from oily fish [18]. The beneficial effects of supplementation of n-3 PUFAs have been previously documented, where studies have identified health benefits as a result of elevating the proportion of n-3 PUFAs in the diet [8, 11, 15, 22]. In addition, previous work has reported that a period of fish oil supplementation significantly ($p < 0.01$) increased the blood plasma levels of EPA compared to baseline [19]. There are also potential benefits that relate to exercise performance, including increased blood flow and vascular conductance, whereby nutrient delivery to the contractile tissue may be optimized [32, 35], and changes in substrate uptake and utilization, whereby glucose transport into the cell after fish oil supplementation was increased twofold

and GLUT 1 receptors were up regulated to a similar extent [1, 13, 28].

At high intensities of exercise (over 70 % of VO_{2max}), an increasingly higher proportion of glucose is oxidized via anaerobic glycolysis [20]. During incremental exercise, the energy system changes from predominantly aerobic (low intensity), where respiratory exchange ratio (RER) is approximately 0.7, to anaerobic (high intensity) where RER increases to 1 and beyond [6]. At approximately 60 % of VO_{2max}, a metabolic switch from free-fatty acid (FFA) metabolism to glucose metabolism occurs [6], and at 80 % VO_{2max} the vast majority of energy (75 %) is from muscular glycogen and to a lesser extent blood glucose [20]. A by-product of anaerobic glycolysis is lactic acid [17] and with increasing reliance on this energy system, blood lactate rises exponentially as exercise intensity increases [7].

Thus for high-intensity bouts of exercise, i. e. resistance training, where intermittent periods of high-intensity muscle contractions are interspersed with rest periods, muscle and blood glucose are the primary sources of fuel [27]. In order to allow the muscle to develop repeated maximal force outputs, it must have glucose readily available. A strategy to enable this would be to ingest glucose periodically to optimize glucose utilization. However, it has been shown that exogenous glucose intake during high-intensity exercise, which relies on simple insulin action to drive the glucose into the muscle cell, may not necessarily lead to increased utilization and hence increased work rate [13].

Previous studies have related n-3 PUFA (which contain both docosohexaenoic acid; DHA, and eicosapentaenoic acid; EPA), intake to improved insulin sensitivity [5, 24, 26, 33]. It has been demonstrated that supplementation with EPA can increase the uptake of plasma glucose into rat myocytes [25, 33, 34] and cultured human myocytes [1]. Supplementation of fish oil in humans has been shown to improve glucose uptake at rest when a glucose load is applied [13]. To date there are no studies examining this effect during incremental exercise and therefore the aims of the current study were to determine the effects of fish oil supplementation (n-3 PUFA) on glucose disappearance and substrate utilization during incremental exercise.

Materials and Methods

Subjects

Ten recreationally active males (mean age, height and weight = 23.8 years ± 0.9,177.2 cm ± 1.8, 76 ± 2.6 kg) participated in the study. All subjects were healthy nonsmokers and did not suffer from any diet-related illness. Before participating in the study, all subjects provided informed written consent, and approval was received from the institute. All testing conformed to guidelines of the Declaration of Helsinki.

Subjects were asked to maintain their habitual type and frequency of exercise, diet (with the exception of preventing the consumption of oily fish, nuts, and seeds), and caffeine consumption throughout the study. With regard to fish oil supplementation, subjects were reminded each day to take their supplements to ensure compliance.

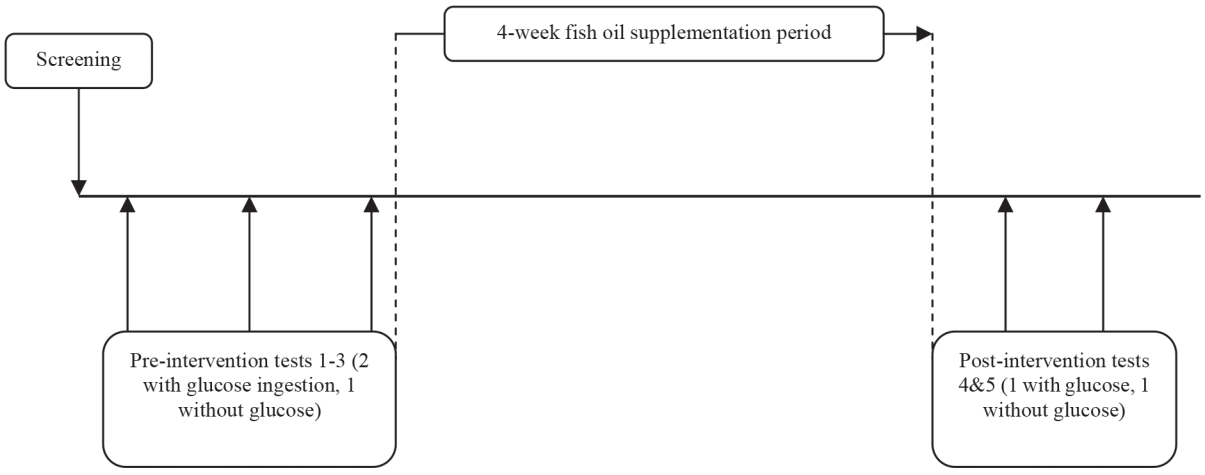

Figure 1: Overview of experimental timeline showing testing order and supplementation period.

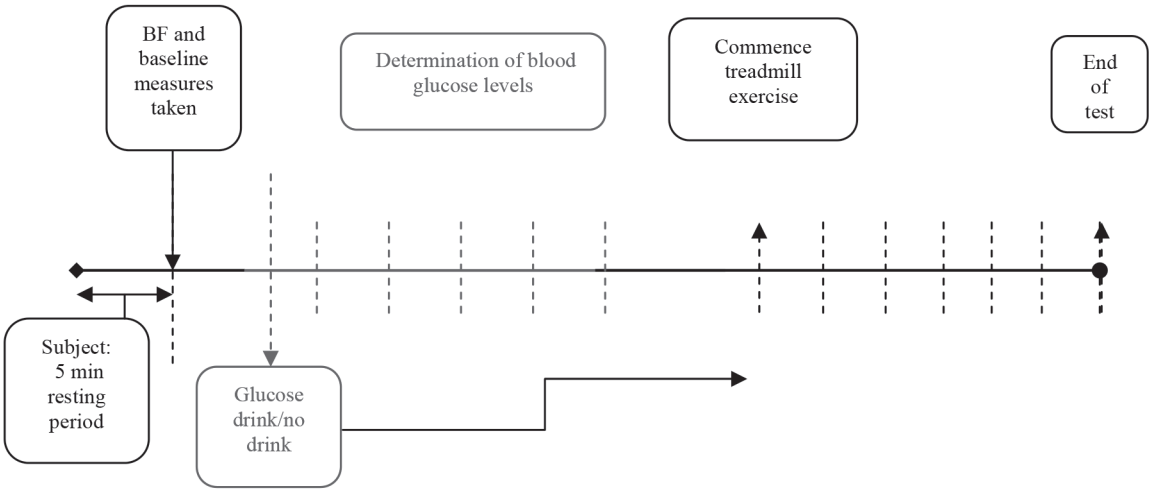

Figure 2: Details of experimental protocol order.

Experimental Design

The study is a within-subjects, pre- to post-test intervention design, consisting of a total of five test days (repeated sessions for reliability). Three of these were pre-intervention and two post-intervention, separated by a 4-week, 4.2 g per day fish oil supplementation period (2.2 g EPA, 1.4 g DHA gelatin capsules – Natures Best, Kent, UK). The experimental timeline displayed below (Figure 1).

Protocols

Baseline measures

Prior to arrival for testing, the subjects were in a fasted state (12 hours). On arrival, subjects completed a PARQ (physical activity readiness questionnaire) and had resting glucose and lactate measurements taken from the fingertips (see Figure 2). Following a five-minute quiet resting period (seated), blood flow (BF) measurements were taken from the femoral artery (superficial) at the lower third of the medial thigh in the sagittal plane (40 mm 7.5 MHz linear array probe) using PW Doppler ultrasound (MyLab 70, Esaote Italy), after initial identification of the vessel using color Doppler. The angle of insonation to the vessel wall was set to 60 degrees throughout. Briefly, standard measures of blood flow velocity were determined via standard algorithms in the software, utilizing measured vessel diameter from the formula $Q = \pi D2/4V_M\text{-}60$, where Q is blood flow, D is vessel diameter, and V is blood velocity averaged over a cardiac cycle.

Subjects then began either treadmill exercise immediately (non-glucose condition), or consumed a 10 % glucose solution at a volume of 4 mL/kg of body weight. Where glucose was administered prior to start of exercise, blood glucose was determined at five-minute intervals until such time as a glycemic plateau (gradient of < 0.05 between two subsequent time points) was reached, at which point exercise commenced.

Exercise measures

Testing consisted of incremental treadmill exercise (Woodway Ergo ELG55, Germany), starting at 3.5 mph for 4 minutes, followed by five stages of 4 minutes each (4.5, 5.5, 6.5, 7.5, 8.5 mph). During the period of exercise, expired gas was continuously analyzed breath by breath (Cortex Biophysik, Metalyzer 3B, Leipzig-Germany) in order to determine the respiratory exchange ratio (RER) (averaged over the last 60 seconds of each stage). All gas measurements were carried out after calibration of the device at each session. Lactate (Analox, P-GM Micro-stat, London) and blood glucose (Accu-check, Compact Plus, Ireland) measurements were taken at the end of each exercise stage, via capillary finger prick sampling after sterilization of the finger.

Statistical methods

Reliability of variables (blood glucose and lactate) was determined using intraclass correlation coefficients (ICC). To enable relative change in measured vari-

ables pre- to post-intervention, all post values variables were normalized to the pre-intervention starting levels (difference between start values subtracted from all subsequent values) to enable relative changes to be detected. Two-way ANOVA (factors – exercise level, condition (glucose/no glucose, pre/post) was carried out to determine the main effects of exercise level or drink (pre to post). Bonferonni *post hoc* tests were used to identify any pairwise significance across the exercise time points on all variables (glucose, lactate, and RER). Alpha level was set to 0.05. All data are presented as mean ± standard error of the mean (SEM).

Results

Resting

Pre-test repeated blood glucose and blood lactate resting values gave ICCs of 0.7 and 0.72, respectively.

Neither blood glucose (4.92 ± 0.11 vs. 4.97 ± 0.12 mmol.L^{-1}) nor blood lactate (1.30 ± 0.11 vs. 1.10 ± 0.16 mmol.L^{-1}) resting values were significantly different ($p > 0.05$) pre- to post-fish oil supplementation. However, resting blood flow was seen to significantly increase following supplementation (0.26 ± 0.02 – 0.30 ± 0.03 L min^{-1}, $p = 0.04$).

After ingestion of the pre-exercise carbohydrate solution, blood glucose profile was seen to initially rise and then plateau after a period of 25 minutes (see Figure 3). No significant differences were seen in the glucose profile pre to post ($p > 0.05$).

During exercise

In the no-glucose condition, pre/post values for blood glucose and lactate showed no significant differences ($p > 0.05$). Subsequently, only conditions following glucose ingestion were entered into the two-way ANOVA for analysis.

With exercise, and after glucose ingestion, blood glucose profiles both pre- and post-fish oil supplementation were seen to follow a similar pattern. From the onset of exercise, blood glucose values decreased over time (see Figure 4). Mean pre- to post-test values of blood glucose were significantly different, with the post values being lower than the pre values over the exercise period (4.58 ± 0.31 vs. 4.66 ± 0.44 mmol.L^{-1}, $p < 0.05$).

Blood lactate increased incrementally with each stage of exercise. Mean pre to post values of blood lactate were not significantly different (4.33 ± 0.82 vs. 4.37 ± 0.93 mmol.L^{-1}, $p > 0.05$).

During the incremental exercise bout, respiratory exchange ratio (RER) increased over each stage, indicating a greater reliance on glycogen as a substrate

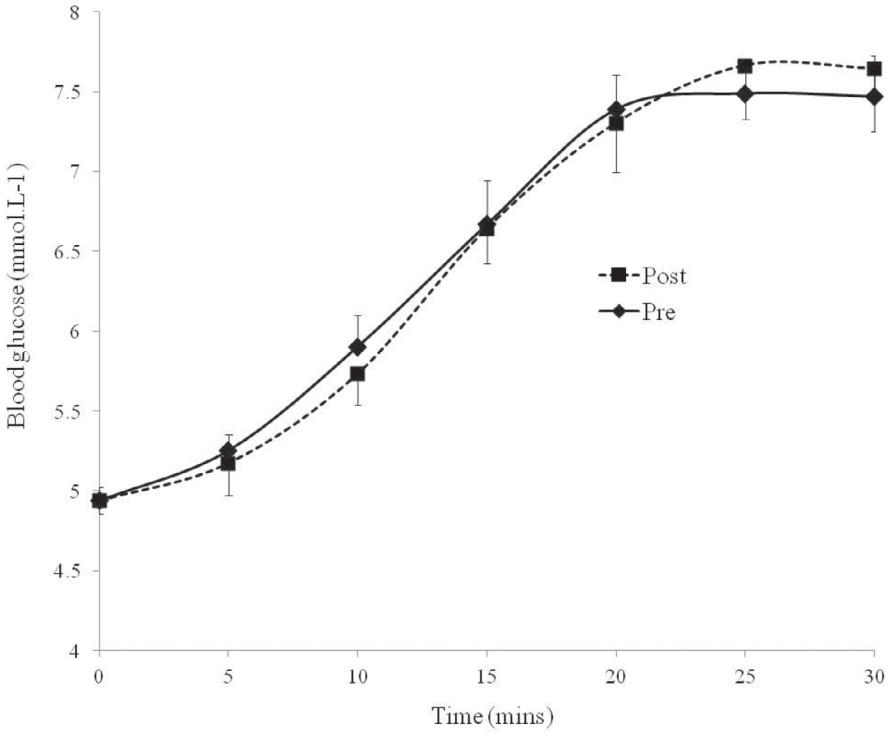

Figure 3: Mean (± SEM) blood glucose profile at rest following glucose bolus pre (◆) and post (■) fish oil intervention (time zero = glucose ingestion). Measures were taken at five-minute intervals until such time as a glycemic plateau (gradient of < 0.05 between two subsequent time points) was reached, at which point exercise commenced.

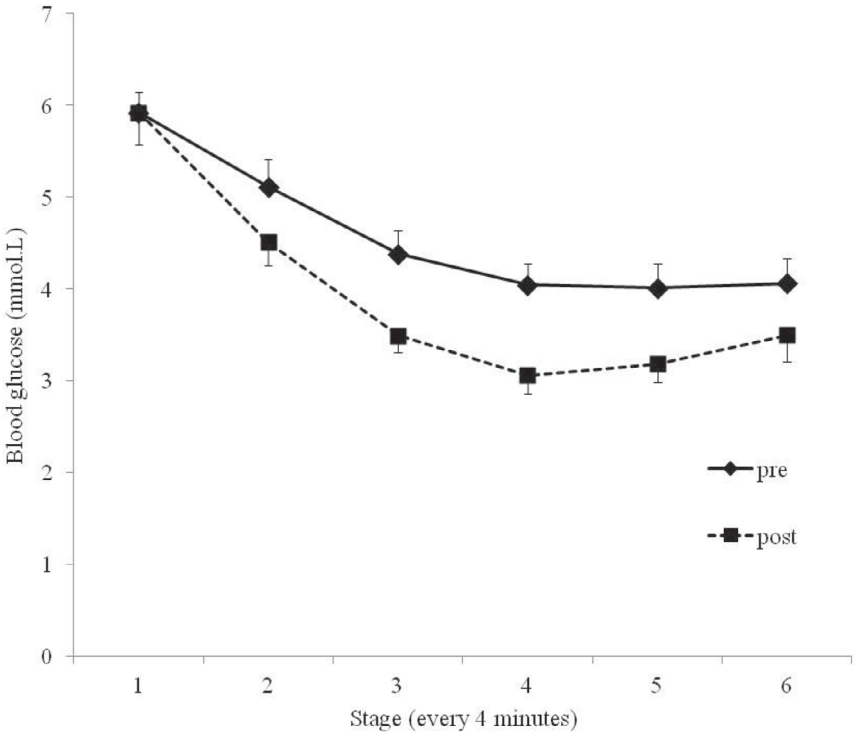

Figure 4: Mean (± SEM) blood glucose profile during incremental exercise pre (◆) and post (■) fish oils. Time points 3, 4 and 5 significantly differ ($p < 0.01$). Measures were taken at the end of each 4-minute stage. Pre to post values significantly different * ($p < 0.05$).

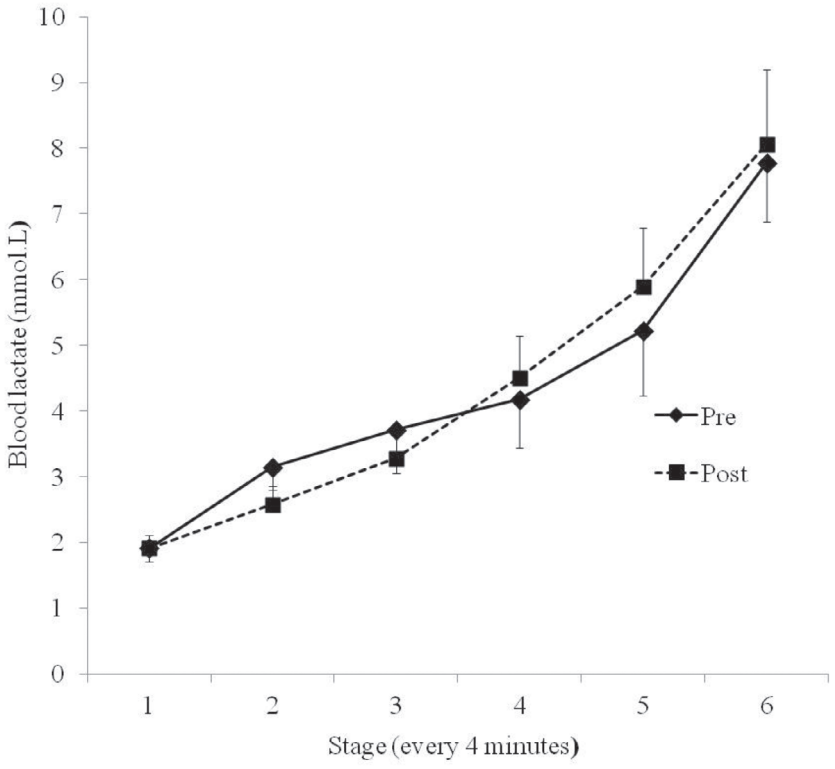

Figure 5: Mean (± SEM) blood lactate response to incremental exercise pre (◆) and post (■) fish oil supplementation. All measures were taken at the end of each 4-minute stage. Note crossover of blood lactate concentration during stage three of the exercise test.

for fuel over time. The mean post-RER value was seen to be significantly greater than the pre-RER value (0.99 ± 0.04 vs. 0.97 ± 0.03, p=0.04). This represents a 7 % increase in carbohydrate (CHO) utilization after fish oil supplementation during exercise (determined using a line fit of CHO to RER values via standard

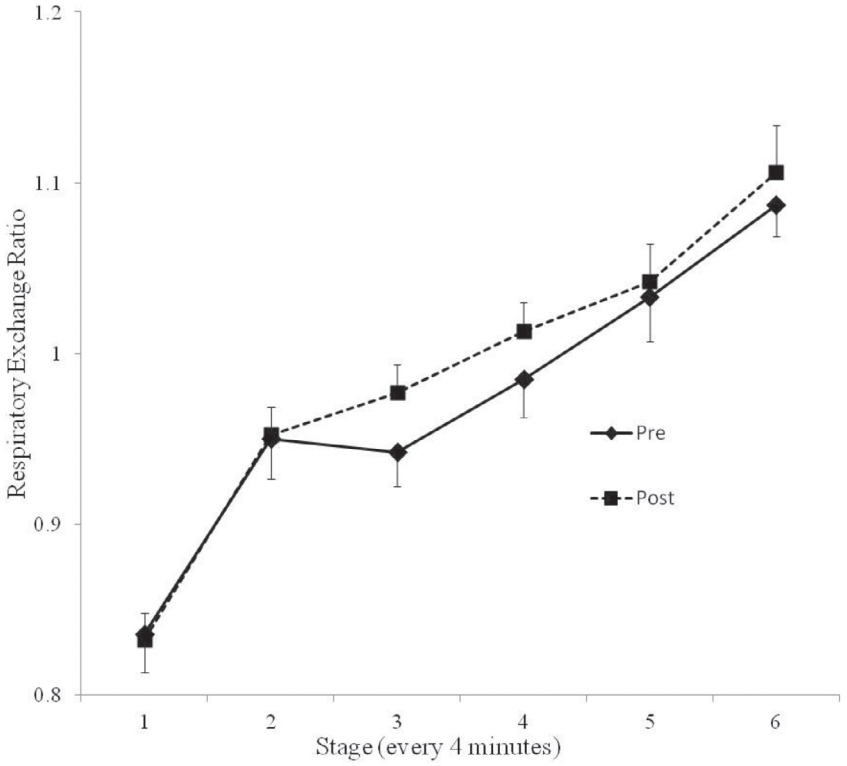

Figure 6: Mean (± SEM) values for RER averaged over the last 60 seconds of each stage of exercise showing incremental changes with increases in exercise intensity pre (◆) and post (■) supplementation. Note greater levels of RER from stage 2 onwards.

non-protein RER -[9]. No differences pre to post with time were observed (see Figure 5).

Discussion

The aim of the current study was to investigate the effect of fish oil supplementation on the markers of cellular metabolism during incremental exercise. The main findings show that following fish oil supplementation, with an acute glucose load, there was a significantly lower level of circulating blood glucose as shown by lower blood glucose levels at each stage of the treadmill exercise, which could be representative of disappearance into the skeletal muscle or liver. In the absence of fish oil supplementation, previous work has indicated that carbohydrate loading can in fact increase glucose utilization [4], however it is not known to date whether fish oil supplementation can further enhance this action. The work here suggests that acute carbohydrate loading, in the presence of fish oil supplementation, can in fact augment the effects of carbohydrate feeding alone.

This indicates that fish oil supplementation may increase insulin sensitivity, which supports previous research where EPA has been found to increase the uptake of plasma glucose into rat myocytes [25, 33, 34], cultured human myocytes [1], and healthy adults [13]. It is well known that glucose transport in human muscle cells is principally mediated by the action of insulin, which stimulates expression and then translocation of the GLUT4 glucose transporter to the cell membrane [2]. Increasing omega-3 PUFAs in the plasma membrane and changing the composition of fatty acids interacting with insulin receptors enhances insulin binding, and subsequently, glucose uptake in muscle sarcolemma [3, 21]. Previous research has shown a strong correlation between fish oil supplementation and increased residency of the primary glucose transporters GLUT 1 [1] and GLUT 4 [24, 34].

To assess the relative proportion of fuel usage during cellular metabolism, respiratory exchange ratio (RER), an acknowledged indirect determinant can be used [10]. Whilst it is acknowledged that RER is an indirect estimate of cellular metabolism, and that blood lactate levels affect RER, this measure can be differentially altered by the balance of lactate clearance versus lactate appearance. Despite this, it is still widely accepted that RER is representative of substrate utilization. Thus, all things being equal, where blood lactate increases as a consequence of increased anaerobic glycolysis, one would expect the RER to increase. In this study, higher RER values were seen from time point two (see Figure 6) during exercise post-fish oil supplementation, and although

not significantly greater, this shows a trend towards greater reliance on glucose for fuel. It may be argued that this higher observed RER could have been due to reduction in lactate clearance rates, although previous work has reported that lactate clearance rates in fact increase following fish oil supplementation [23]. Furthermore, our observations are further supported by the fact that no differences were seen pre to post in any of the measured variables without the glucose bolus, indicating that any changes seen in these variables must be as a consequence of the combined action of fish oils with the acute glucose bolus. In the current study, it must be noted that a placebo group was not used, as the intervention group acts as its own control. However, it may be pertinent for future studies to include a separate placebo group to confirm the robustness of the present findings.

In addition to the observed changes in fuel utilization, it was also noted that resting blood flow increased after fish oil supplementation (0.04 $L.min^{-1}$). These findings support previous studies which have shown increased blood flow during exercise following fish oil supplementation [29, 35]. With the ability of fish oil seen to increase blood flow, it could be postulated that this mechanism may enable a faster recovery and greater subsequent training volumes within any given time period, via increased supply of nutrients and oxygen in any given time period. Furthermore, this increased supply of blood and associated oxygen to the exercising tissue may enable greater work to be completed for a similar lactate production; further work would be required to examine this possibility. Also where exercise is acute and of a high intensity, the enhanced ability to supply glucose and also utilize glucose should enable greater amounts of work to be carried out. In addition to the benefit of this ability to athletic populations, it also has positive implications for some clinical groups where blood flow supply to muscle tissue may be compromised, i. e. claudicants.

Resistance training, or repeated bouts of high-intensity effort such as sprinting, which relies to a greater extent on glucose for fuel, may benefit from the outlined protocol in this study. The effects shown may allow for larger volumes of acute work at higher intensities during a given exercise session, potentially enhancing the training effect. Longer term, the ability to work at higher intensities may in fact allow for more effective increases in muscle hypertrophy. For example, fish oil supplementation alone has previously been reported to increase protein synthesis in young and older adults by acting on the anabolic pathways in skeletal muscle [30, 31]. This may be of particular benefit for example in certain population groups (i. e. elderly), where insulin resistance is prevalent [12,16]. For high-intensity bouts of exercise, this study has not only found potential benefits of fish oil supplementation in combination with an acute glucose load, but also that blood flow increased at rest, which may lead to an enhanced recovery process between subsequent training sessions by way of supplying essential nutrients to the depleted muscles, removing associated waste products, and aiding in the repair process. Future work needs to address the suggestion that fish oil supplementation may allow for increased volumes of high-intensity work and thus enhance the potential "training effect" in a variety of population groups. In addition, examination of chronic muscle tissue changes with training and associated performance markers will help validate this proposition.

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