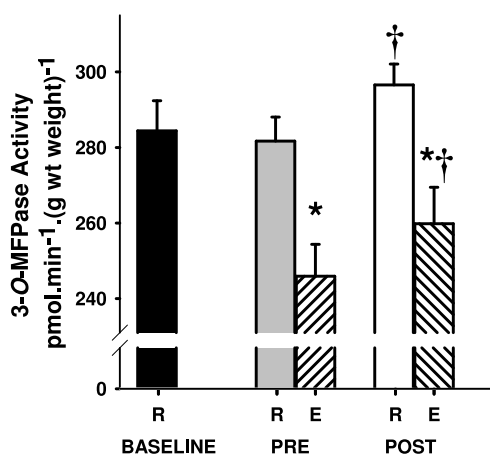


Opposing effects of acute and chronic high intensity exercise on Na⁺K⁺ATPase activity in skeletal muscle

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The Na⁺K⁺ATPase enzyme is critical in maintaining muscle trans-sarcolemmal [Na⁺] and [K⁺] gradients and membrane excitability. However, repeated maximal muscle contractions reduce maximal Na⁺K⁺ATPase activity (Fraser *et al.*, 2002). High-intensity interval training is commonly used by endurance athletes to improve endurance performance, but the effects of acute high-intensity interval exercise on muscle Na⁺K⁺ATPase activity are not known. Furthermore, although sprint training increases muscle Na⁺K⁺ATPase content (McKenna *et al.*, 1993), the effects of high-intensity interval training on muscle Na⁺K⁺ATPase activity are unknown. We therefore examined the possible contradictory effects of acute and chronic high-intensity interval exercise on the muscle Na⁺K⁺ATPase activity in seven male endurance-trained athletes.

A vastus lateralis muscle biopsy was taken at rest, 3-wks prior to (Base), at rest and immediately after exercise prior to (Pre) and after (Post) 3-wks of training. Muscle samples were analysed for maximal *in vitro* Na⁺K⁺ATPase (K⁺ stimulated 3-*O*-MFPase) activity. Performance was characterised by incremental $\dot{V}O_2$ and peak power output (PPO); and during a simulated 40km time-trial, by mean power output (MPO). A t-test was used for resting 3-*O*-MFPase activity to determine reproducibility (Base – Pre) and to compare change scores (Base-Pre, & Pre-Post) to identify a change with training. A two-way ANOVA with repeated measures was applied to test for main effects of exercise (Rest, Ex) and training (Pre, Post).



Resting muscle 3-*O*-MFPase activity did not differ between Base and Pre. Acute high-intensity interval exercise depressed muscle 3-*O*-MFPase activity by 12.7±1.2% (mean±SEM, Exercise main effect, $P<0.05$, *, see figure). In contrast, training increased 3-*O*-MFPase activity by 4.9±0.7% (training main effect, $p<0.05$, †, see figure). Resting 3-*O*-MFPase activity was increased after training by 5.4±1.0% (Pre – Post change score, $P<0.05$). Neither $\dot{V}O_2$ (Base 64.4±1.6; Pre 64.3±1.5; Post 65.8±1.9), PPO (Base 368±12; Pre 374±13; Post 379±14), or MPO in the 40km time-trial (Base 279±12; Pre 303±15; Post 303±13) differed significantly after training.

In conclusion, Na⁺K⁺ATPase measures were reproducible in resting muscle. Acute and chronic high-intensity interval exercise had converse effects on Na⁺K⁺ATPase activity. The small rise in Na⁺K⁺ATPase activity with training was insufficient to improve exercise performance.

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This study was partially funded by an Australian Research Council SPIRT grant (C00002552).