Acute incremental exercise, sprint exercise, as well as chronic intermittent hypoxia each decrease muscle Na^+K^+ATPase activity

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The Na^+K^+ATPase enzyme is vital in maintaining skeletal muscle excitability. Athletes commonly use hypoxic exposure to improve athletic performance, favouring the live high, train low approach (LHTL). Paradoxically, muscle Na^+K^+ATPase content is reduced by chronic hypoxia (Fraser *et al.*, 2002), which would be expected to reduce muscle performance. Muscle maximal Na^+K^+ATPase activity is also decreased with fatiguing single-leg kicking exercise (Fraser *et al.*, 2002), although the effects of acute intense cycle exercise are unknown. We therefore investigated the effects of acute incremental and sprint exercise and of LHTL on muscle Na^+K^+ATPase activity and exercise performance.

Two studies were performed, where control subjects slept and trained in Canberra (altitude ~600m), whilst the LHTL groups slept in a hypoxic room (study 1, 3000m for 23-nights; study 2, 2650 m for 20-n), and trained at 600m. In study 1, 13 endurance athletes were assigned to either a control (CON, n=6) or LHTL group (n=7). In study 2, 21 endurance athletes were assigned to a control (CON2, n=7), 20 consecutive night LHTL ($LHTL_c$, n=7), or an intermittent 20 night LHTL ($LHTL_i$, 4 x [5-n LHTL then 2-n CON]) group. The lower simulated altitude and intermittent exposure in study 2 were used to reflect common athletic practice. A vastus lateralis muscle biopsy was taken at rest and immediately after incremental exercise prior to (Pre) & after (Post) 23-n of LHTL (study 1); and at rest and immediately after ~1-min sprint exercise prior to (Pre) and after (Post) 20-n LHTL (study 2.) The timecourse of adaptation was investigated in study 2 via an additional rest and post sprint exercise muscle biopsy taken after 5-n of LHTL. Muscle was analysed for maximal *in vitro* Na^+K^+ATPase (K^+ stimulated, 3-*O*-MFPase) activity. Arterialised venous plasma [K^+] was analysed during and following incremental (study 1) and sprint exercise (study 2).

Muscle 3-*O*-MFPase activity was depressed to a similar extent after both incremental (-12.4±0.8%, study 1, exercise effect, $P<0.05$) and sprint exercise (-12.3±0.5%, study 2, exercise effect, $P<0.05$). In study 1, the change in resting 3-*O*-MFPase activity (Pre Post) was greater in LHTL (-2.9±1.1%, $P<0.05$) than CON (0.4±0.5%, NS). In study 2, resting muscle 3-*O*-MFPase was also reduced from Pre to Day 5 in both $LHTL_c$ and $LHTL_i$ groups (-2.1±0.4% and -2.3±0.2% respectively, $P<0.05$), but was unchanged in CON (0.3±0.9%, NS). Resting 3-*O*-MFPase activity (Day 5 Post) was unchanged in $LHTL_c$ and CON (-0.8±0.7% and 0.5±0.6 respectively, NS), but was reversed and increased in $LHTL_i$ (3.5±1.2%). The Pre - Post change in resting 3-*O*-MFPase activity was lowered in $LHTL_c$ (-2.9±0.7%, $P<0.05$), remained unchanged in CON (0.8±1.0%), but tended to increase in $LHTL_i$ (1.1 ± 1.2%). Plasma [K^+] rose with exercise and then declined post-exercise ($P<0.05$) in each study, but was unchanged by LHTL, $LHTL_c$ and $LHTL_i$ (data not shown).

In conclusion, markedly different exercise regimes each acutely depressed skeletal muscle maximal Na^+K^+ATPase activity, with no residual effect evident after 5d recovery. This effect was reproducible, suggesting an obligatory response to heavy exercise. LHTL at 3000m for 23-n, and at 2650m for 20-n each induced only a small reduction in resting Na^+K^+ATPase activity, which was reversed with inclusion of normoxic nightly exposure. In contrast to continuous hypoxic exposure, LHTL caused only a small depression in Na^+K^+ATPase activity. This was insufficient to adversely affect muscle performance or plasma K^+ regulation, but may be energetically advantageous and might explain why exercise performance is not impaired with LHTL.

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