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Conference Abstract

The Effect of Low-Intensity Exercise Duration on Acute Skeletal Muscle Signalling Responses

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Abstract

Introduction: Elite endurance athletes typically demonstrate a polarised or pyramidal training intensity distribution, comprising of large volumes of low-intensity training (70-90%) and smaller volumes of moderate- and high-intensity training (10-30%) (Stöggl & Sperlich, 2015). Considerable research has investigated high-intensity training optimisation (Laursen & Buchheit, 2019), however, there is limited research identifying best practice for manipulating the duration and frequency of low- intensity training sessions.

Mitochondrial biogenesis is initiated by homeostatic perturbations within skeletal muscle which activate sensor proteins, including AMPK, CaMKII, and p38 MAPK. Subsequent upregulation of downstream signalling pathways results in an increase in the abundance and/or function of mitochondrial proteins and, over time, an improvement in physiological function (Egan & Zierath, 2014). Whilst it has been shown that low-intensity exercise upregulates signalling responses involved in mitochondrial biogenesis (Almquist et al., 2020), the role of low-intensity exercise duration is yet to be understood.

The aim of the present study was to determine the effect of low-intensity exercise duration (2 h vs. 5 h) on acute skeletal muscle signalling responses involved in mitochondrial biogenesis.

Methods: Eight trained males (age: 22.0 ± 3.7 yr; body mass: 70.7 ± 7.6 kg; VO2max, 65.5 ± 9.1 mL kg min^{-1}) volunteered for this study. Participants completed submaximal and maximal ramp tests to determine the first lactate threshold (LT; 0.4 mmol L^{-1} rise in blood lactate concentration above baseline) and VO2max. Participants subsequently completed three familiarisation sessions, involving cycling at 75% of LT power output for 1, 2, and 3 hours.

In a repeated-measures study design, participants completed 2 h and 5 h bouts of exercise at 75% of LT power output, separated by a minimum of 14 days. Throughout each trial, participants consumed 60 g hr^{-1} of carbohydrate in the form of maltodextrin drinks and maltodextrin-fructose gels.

Pulmonary gas exchange and heart rate were sampled, and venous blood (for analysis of lactate and glucose concentration) was obtained every 15 min for the first hour and every hour for the remaining duration of exercise. Muscle biopsies (vastus lateralis) were obtained pre-exercise, post-exercise, and 3 h post-exercise. Samples were analysed for glycogen content and Western blotting for phospho-AMPK^{Thr172}, phospho-CaMKII^{Thr286}, phospho-p38 MAPK$^{Thr180/182}$, as well as total content of AMPK, CaMKII, and p38 MAPK.

Muscle signalling responses were expressed as phospho/total relationships for each target and analysed using a linear mixed model. Physiological responses were analysed using a repeated measures ANOVA. Where there were significant main effects, Bonferroni post hoc tests were used to locate the differences. Significance was accepted when $P < 0.05$ and data are presented as mean ± SD.

Results:
Muscle glycogen Power output during the trials was 131 ± 17 W. Pulmonary VO_2 after 15 min was 2.25 ± 0.17 and 2.20 ± 0.16 L min^{-1} in the 2 h and 5 h trials, respectively. VO_2 increased from 15 min to end-5 h (Δ 0.30 ± 0.17 L min^{-1}; $P = 0.02$) and remained unchanged in the 2 h trial. Heart rate after 15 min was 121 ± 10 and 122 ± 18 beats min^{-1} in the 2 h and 5 h trials, respectively. Heart rate increased from 15 min to end-5 h (Δ 27 ± 17 beats min^{-1}; $P = 0.042$) and remained unchanged in the 2 h trial. Blood lactate and glucose remined unchanged during exercise. Carbohydrate oxidation decreased (2.30 ± 0.21 to 1.9 ± 0.55 g min^{-1}; $P < 0.001$) and fat oxidation increased (0.15 ± 0.09 to 0.48 ± 0.16 g min^{-1}; $P < 0.001$) from 15 min to end-5 h and remained unchanged in the 2 h trial.content at rest was 551 ± 123 and 635 ± 178 mmol kg dw^{-1} in the 2 h and 5 h trials, respectively. Post-exercise muscle glycogen decreased to a greater extent ($P = 0.049$) in the 5 h trial compared to the 2 h trial (Δ 474 ± 232 vs. Δ 228 ± 168 mmol kg dw^{-1}, respectively).
There was a main effect of time for AMPK ^{Thr172} phosphorylation ($P < 0.001$), which was greater from pre- to post-exercise ($P < 0.001$) and pre- to 3 h post-exercise ($P = 0.009$). Post-hoc analysis revealed an increase in AMPK^{Thr172} phosphorylation from pre- to post-exercise in the 5 h trial (6.62 ± 7.17-fold; $P = 0.025$) and no change in the 2 h trial (3.1 ± 3.0-fold; $P = 0.362$). CaMKII^{Thr286} and p38 MAPK$^{Thr180/182}$ phosphorylation remained unchanged ($P > 0.05$) post-exercise and 3 h post-exercise in both trials.

Conclusion: Extending the duration of low-intensity exercise from 2 h to 5 h increased post-exercise AMPK ^{Thr172} phosphorylation. This was accompanied by a greater reduction in muscle glycogen content following the 5 h trial compared to the 2 h trial. The duration-dependent AMPK signalling response supports recent data suggesting exercise duration is an important factor in the magnitude of the post-exercise AMPK phosphorylation response (Rothschild et al., 2022). Forthcoming analyses will determine the effect of low-intensity exercise duration on the subcellular translocation of key transcriptional regulators (e.g. PGC-1), down-stream gene expression, fibre type-specific signalling, and angiogenic signalling responses. These initial data provide an insight into the training-induced signalling responses involved in the regulation of mitochondrial biogenesis.

Keywords: Training, Physiology, Adaptation, Signalling

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