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Does six-weeks of high-intensity cycle training with induced changes in acid-base balance lead to mitochondrial adaptations?

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Abstract

Background: Endurance training leads to an improved ability of muscle to utilize oxygen. This is related to an increased density and function of mitochondria. The biogenesis and adaptation of mitochondria is a complex process mediated by various signalling pathways and seems to be highly sensitive to the type of exercise and the local environment in the muscle. Changes in the muscle environment in terms of altered metabolism and substrate accumulation are affected by changes in acid/base balance in response to exercise. Recent studies (Bishop et al., 2010:Am J Physiol Endocrinol Metab, 299:E225-233; Edge et al., 2006: J Appl Physiol, 101:918-925) have shown that changes in acid/base balance may affect the regulation of mitochondrial adaptation to acute exercise; however, how these changes are affected by training and relate to performance adaptations in humans is unclear. Similarly, the effect of acid/base balance on mechanisms underlying mitochondrial biogenesis is unclear.

Purpose: The objectives of this study were to examine the relationship between acid/base balance, mitochondrial biogenesis and adaptation.

Methods: Nineteen recreationally active men undertook six-weeks (3 x wk) high-intensity interval training programme, known to produce increases in mitochondrial biogenesis. Training sessions involved repeated 2-min efforts at an intensity ranging from 85-110% VO_2peak separated by 1 min recoveries. Participants were matched for maximum oxygen consumption and randomly assigned to one of two different training groups. One group ingested sodium bicarbonate (alkaline, 0.4 $g \cdot kg^{-1}$ body weight) and the other ingested a placebo prior to each training session. Performance test results, blood samples and muscle biopsies were collected before and after the training period and assessed for changes in aerobic fitness, blood metabolites and markers of mitochondrial function and biogenesis. Changes in gene expression associated with mitochondrial biogenesis were also examined.

Results: Post training, there were significant ($P < 0.05$) improvements in time to fatigue (mean ± SD) for placebo (54.9 ± 26.6%) and alkaline (74.7 ± 40.0%) groups, significant increases in maximum power output (8.8 ± 5.% vs 10.6 ± 6.5%) and lactate threshold power (5.7 ± 7.0% vs 9.4 ± 10.1%) were also seen in both groups. There were also significant increases in citrate synthase activity in the alkaline group (22.3 ± 26.7&) and VO_2peak in the placebo group (9.2 ± 5.6%). Trends for improvements were also seen in citrate synthase activity in the placebo group (12.6 ± 19.3%) and VO_2peak in the alkaline group (4.5 ± 6.9%), however these did not reach significance ($P = 0.089$ and 0.066 respectively). Despite the significant changes in performance within a group, there were no significant differences between groups.

Discussion: Both training groups showed substantial changes in performance and physiological measures following the training period, however, suppressing exercise-induced acidosis during training did not significantly improve mitochondrial adaptations or performance in comparison to a placebo condition. There was a large degree of individual variation in the responses of physiological and performance measures to the treatments, however there were trends towards greater local metabolic and muscular adaptations when exercise-induced acidosis was reduced.

Conclusion: The relationship between acid/base balance and mitochondrial adaptations in humans is complex. Individual differences in training responses at a local muscular and central level in this study highlight the need to gain a better understanding of how alterations in exercise-induced acidosis affect specific cellular events leading to skeletal muscle adaptation.

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