

Determinants of muscle buffer capacity

D. Bishop and J. Edge, School of Human Movement and Exercise Science, The University of Western Australia, 35 Stirling Highway, Crawley, WA 6009, Australia.

Little is known about the stimulus required to increase muscle buffer capacity ($\beta_{in-vitro}$). It has been hypothesised that it is important that training is: (1) of high intensity; and (2) is performed under conditions of high skeletal muscle hydrogen ion (H^+) accumulation (Weston et al., 1996). We tested this first hypothesis by investigating the effects on $\beta_{in-vitro}$ of two training protocols of different intensity, but matched for total work. It has previously been shown that increasing the extracellular buffer concentration can reduce the skeletal muscle H^+ accumulation during high-intensity exercise (Costill et al., 1984). We therefore tested the second hypothesis by experimentally manipulating the extracellular buffer concentration during training.

For the first study, 18 untrained females (mean \pm SD: age 19 ± 1 y, mass 59.8 ± 5.8 kg) were randomly assigned to high-intensity interval training (INT-5) or moderate intensity continuous (CON-5) training. Training was matched for total work and consisted of 6 - 10 \times 2 min intervals (1 min rest) at 130 - 160% of lactate threshold (LT) (INT-5) or 20 - 35 min of continuous cycling at 85 - 95% of LT (CON-5), 3 \times per week for 5 weeks. For the second study, 10 untrained females (mean \pm SD: age 20 ± 3 y, mass 62.3 ± 10.0 kg) were also randomly assigned to one of two training groups, matched for total work. One group (BIC-8) ingested sodium bicarbonate ($NaHCO_3$, 0.4 g \cdot kg $^{-1}$) while the control group (INT-8) ingested a placebo ($NaCl$, 0.2 g \cdot kg $^{-1}$) prior to each training session. Training consisted of 6 - 12 \times 2 min intervals (1 min rest) at 130 - 180% of LT, 3 \times per week for eight weeks. Muscle biopsies (vastus lateralis) were taken at rest to determine muscle lactate ($[La^-]_m$), pH_m and $\beta_{in-vitro}$.

Training responses are summarised in the table. All training programs resulted in a significant improvement in $O_{2\ peak}$ and LT with no significant difference between groups. However, relative to CON-5, INT-5, INT-8 and BIC-8 had a significantly greater improvement in $\beta_{in-vitro}$. The pooled data revealed a significant negative correlation between initial $\beta_{in-vitro}$ and percent change with training ($r=0.58$; $P<0.05$).

Training	Peak O_2		LT		$\beta_{in-vitro}$	
	Pre	Post	Pre	Post	Pre	Post
CON-5	41.3 ± 7.3	$45.6 \pm 5.7^*$	137 ± 33	$152 \pm 29^*$	123 ± 32	125 ± 19
INT-5	42.8 ± 6.6	$48.1 \pm 7.4^*$	141 ± 27	$149 \pm 27^*$	126 ± 15	$150 \pm 19^*$
INT-8	40.7 ± 5.6	$47.7 \pm 6.1^*$	113 ± 18	$130 \pm 21^*$	140 ± 32	$161 \pm 19^*$
BIC-8	35.2 ± 7.1	$43.0 \pm 6.4^*$	109 ± 21	$137 \pm 20^*$	129 ± 32	$156 \pm 19^*$

* significantly different to pre-training ($p<0.05$)

Despite similar changes in aerobic fitness, INT-5 had a significantly greater increase in $\beta_{in-vitro}$ than CON-5. This suggests that it is the intensity of training, not the total work performed, that is the stimulus for change in $\beta_{in-vitro}$. We have also shown that ingesting $NaHCO_3$ and therefore altering the likely accumulation of H^+ during training, did not affect these adaptations.

Costill, D.L., Verstappen, F., Kuipers, H., Janssen, E. and Fink, W. (1984) *International Journal of Sports medicine* 5, 228-231.

Weston, A.R., Wilson, G.R., Noakes, T.D. and Myburgh, K.H. (1996) *Acta Physiologica Scandinavica* 157, 211-216.