Effects of endurance exercise intensity on acute signalling responses to subsequent resistance exercise in human skeletal muscle

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Incorporating both resistance (RE) and endurance (EE) exercise into a training regime, known as concurrent training, can attenuate muscle hypertrophy and strength gains compared with undertaking RE alone (Kraemer *et al*., 1995). This phenomenon may result from EE-mediated interference to skeletal muscle anabolic responses induced by RE (Hawley, 2009). However, most concurrent training studies performed to date have employed moderate-intensity continuous (CONT) cycling as the EE modality. Thus, whether EE intensity modulates any potential acute interference effect following concurrent exercise is unclear (Fyfe *et al*., 2014). We therefore compared the effects of high-intensity interval training (HIT) and lower-intensity CONT cycling on the phosphorylation of signalling proteins regulating muscle protein synthesis before and after subsequent RE, compared with RE performed alone.

Eight recreationally-active males (age, 27 ± 4 y; height, 178.3 ± 6.1 cm; body mass, 83.7 ± 13.7 kg; VO_{2peak}, 45.7 ± 9 ml•kg^{-1}•min^{-1}; mean ± SD) completed three experimental trials in a randomised order: 1) HIT cycling followed by RE (HIT+RE trial), 2) work-matched CONT cycling followed by RE (CONT+RE trial), and 3) RE alone (RE trial). For the RE trial, participants performed RE (8 × 5 unilateral leg press at 80% 1RM) alone. For HIT+RE and CONT+RE, RE was performed after 15 min of passive recovery following either HIT cycling (10 × 2 min at 120% lactate threshold [LT]/1 min passive recovery) or work-matched CONT cycling (30 min at 80% LT). Muscle biopsies were obtained from the *vastus lateralis* before RE (PRE), 1 h (+1 h) and 3 h (+3 h) after RE.

Compared with RE, muscle glycogen content was reduced for HIT+RE and CONT+RE at PRE (~50 % and ~40 %, respectively; $P < 0.05$), +1 h (~62 % and ~43 %; respectively; $P < 0.05$) and +3 h (~45 % and ~31 %; respectively; $P < 0.05$). For HIT+RE, ACC^{Ser79} phosphorylation was higher compared with RE at PRE (~530 %; $P < 0.05$), at +1 h (~133 %; $P < 0.05$) and at + 3 h (~458 %; $P < 0.05$), and higher only at PRE for CONT+RE (~451 %; $P < 0.05$) compared with RE. The phosphorylation of mTOR^{Ser2448} was ~105 % higher at PRE for HIT+RE compared with RE ($P < 0.05$) and ~44 % higher for HIT+RE compared with CONT+RE at PRE ($P < 0.05$). At +1 h, mTOR phosphorylation was ~128 % higher for HIT+RE compared with CONT+RE ($P < 0.05$), but was not different from RE. RE increased p70S6K^{Thr389} phosphorylation ~171 % above resting values at +1 h ($P < 0.05$) and ~65 % at +3 h ($P < 0.05$); however, prior HIT or CONT did not alter this response compared with RE. RE increased GSK-3α/β$^{Ser21/9}$ phosphorylation ~80 % above resting values at + 1 h ($P < 0.05$); in contrast, this response was lower at +1 h for both HIT+RE (~33 %; $P < 0.05$) and CONT+RE (~34 %; $P < 0.05$) compared with RE. Prior CONT reduced eEF2^{Thr56} phosphorylation at PRE compared with RE alone (~37 %; $P < 0.05$), whilst eEF2 phosphorylation was reduced ~16 % at + 1 h for HIT+RE compared with RE ($P = 0.05$).

Despite considerable muscle glycogen depletion and increased AMPK activity (indexed by ACC phosphorylation) induced by HIT and CONT, these protocols did not interfere with early anabolic responses to subsequent RE, including mTOR and p70S6K phosphorylation. It therefore appears that EE intensity does not modulate any putative molecular interference effect following acute concurrent exercise. Further work is required to determine the significance of altered anabolic signalling responses in human skeletal muscle by divergent EE intensities, and particularly the effect of EE intensity on chronic adaptations to long-term concurrent training.

Fyfe JJ, Bishop DJ, Stepto NK. (2014) *Sports Medicine*, **44**: 743-62.
Hawley JA. (2009) *Applied Physiology, Nutrition & Metabolism*, **34:** 355-61.
Kraemer WJ1, Patton JF, Gordon SE, Harman EA, Deschenes MR, Reynolds K, Newton RU, Triplett NT, Dziados JE. (1995) *Journal of Applied Physiology*, **78**: 976-89.