Concurrent training further enhances markers of skeletal muscle ribosome biogenesis, but not associated signalling responses, versus single-mode resistance training

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Previous work (Apro *et al.*, 2013; Fyfe *et al.*, 2016) suggests attenuated skeletal muscle hypertrophy with concurrent training is not mediated *via* blunted post-exercise increases in translational efficiency (*i.e.* mTORC1 signalling or rates of muscle protein synthesis). Whether increased translational capacity (*i.e.* ribosome biogenesis), which occurs following resistance training (RT) in humans (Figueiredo *et al.*, 2015), is instead attenuated with concurrent training is unclear. We therefore examined markers of ribosome biogenesis in human skeletal muscle after concurrent training [incorporating either high-intensity interval training (HIT) or moderate-intensity continuous training (MICT)], *versus* RT performed alone.

Recreationally-active men (n = 23; VO_{2peak}, 44 ± 11 $mL \cdot kg^{-1} \cdot min^{-1}$; mean ± SD) underwent 8 wk (3 sessions/wk) of either 1) HIT cycling and RT (HIT+RT group, n=8), 2) MICT cycling and RT (MICT+RT group, n=7) or 3) RT only (RT group, n=8). *Vastus lateralis* biopsies were obtained at rest both pre- and post-training to evaluate basal training-induced responses, and 1 h and 3 h after the final training bout to examine post-exercise skeletal muscle molecular responses in a training-accustomed state.

Basal training-induced changes in markers of ribosome biogenesis in skeletal muscle, including expression of the 45S rRNA (ribosomal RNA) precursor (~75%), and mature rRNA species 5.8S (~125%) and 28S (~75%) were greater for both MICT+RT and HIT+RT *versus* RT alone, mirroring the larger increases in total RNA content (~27-47%). During the final training session, RT further increased the phosphorylation of $p70S6K1^{Thr389}$ (~50%) and regulators of 45S rRNA transcription [$TIF\text{-}1A^{Ser649}$ (~52-75%) and UBF^{Ser388} (~49-64%)] *versus* concurrent exercise. Training-induced increases in type I muscle fibre cross-sectional area were attenuated following HIT+RT *versus* RT alone (~34%).

Training-induced changes in mature rRNA expression and total RNA content in skeletal muscle were greater with concurrent training *versus* RT alone, suggesting enhanced ribosome biogenesis. This occurred despite RT further inducing both mTORC1 and ribosome biogenesis-related signalling responses following the final training bout. Attenuated changes in skeletal muscle translational capacity therefore do not appear to mediate interference to RT adaptations with short-term concurrent training; however, blunted mTORC1 and ribosome biogenesis-related signalling may play a role following longer-term training.

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