

Effects of hypoxia on skeletal muscle molecular adaptations to heavy resistance training

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We have previously shown that heavy resistance training in hypoxia (IHRT) results in greater strength improvements compared to the same training in normoxia (Inness *et al.*, 2016). However, the mechanisms responsible for enhanced strength gains with IHRT have not been investigated. We therefore determined the effects of IHRT on skeletal muscle molecular adaptations that contribute to muscle strength.

A pair-matched, placebo-controlled, single-blind study included 18 resistance-trained participants assigned to either IHRT or placebo (PLA) (n = 9 per group). Both groups performed 20 sessions over 7 weeks with either IHRT (FiO₂ 0.143) or PLA (FiO₂ 0.20). Groups were matched for body mass (mean ± S.D.; 83.1 ± 7.5, 80.2 ± 12.0 kg), height (1.83 ± 0.05, 1.81 ± 0.06 m), one-repetition maximum (1-RM) squat (121.4 ± 22.1, 125.5 ± 30.7 kg) and training history. Resting *vastus lateralis* muscle biopsies were taken following an overnight fast before and after the training programme. Biopsies were analysed for muscle fibre cross-sectional area (CSA), mTOR signalling, proteins involved in sarcoplasmic reticulum (SR) calcium uptake and release, and proteins involved in force transfer between the sarcomeres and extracellular matrix.

Training increased Type II fibre CSA in both groups (mean ± 90% confidence limits (CL), effect size (ES); IHRT: 16.0 ± 25.2%, ES 0.50; PLA: 22.0 ± 31.8%, ES 0.42). Type I CSA only increased in PLA (16.1 ± 23.3%, ES 0.48); however, the changes in Type I or II fibre CSA were not different between groups. Training caused no substantial change in total p70S6K in either group. Training caused a possibly trivial decrease in total mTOR in PLA, and a possibly small increase in IHRT, resulting in a greater increase in mTOR for IHRT compared to PLA (18.9 ± 27.3%, ES 0.65). The content of SR-associated proteins dihydropyridine receptor, SERCA1, and calsequestrin did not change in either group. In contrast, SERCA2 increased in IHRT only (23.5 ± 18.7%, ES 0.33), and this increase was greater compared to PLA (42.6 ± 52.2%, ES 0.63). The content of force transfer protein dystrophin did not change in either group; however, alpha-actinin increased only in IHRT (47.8 ± 67.5%, ES 0.67), and this was greater compared to PLA (63.1 ± 79.5%, ES 1.10).

The greater strength increases following heavy resistance training in hypoxia compared to normoxia are possibly due to enhanced SR calcium regulation and force transfer between sarcomeres and the extracellular matrix.

Inness M, Billaut F, Walker E, Petersen A, Sweeting A, & Aughey R (2016). Heavy resistance training in hypoxia enhances 1RM squat performance. *Front Physiol* **7**, 502.