The effects of short-term unloading and re-training on skeletal muscle function and Na^+,K^+-ATPase abundance in humans

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Introduction: The Na^+,K^+ -ATPase (NKA) in skeletal muscle is a vital protein for the maintenance of muscle excitability, and adapts to chronic physical activity. We investigated whether changes in muscle strength, size and endurance seen after 23 days of unilateral lower limb suspension (ULLS) in humans were accompanied by decreased abundance of skeletal muscle NKA, and whether such changes were reversible with short-term re-training.

Methods: Knee extensor voluntary strength, fibre cross sectional area (Immunofluoresence), venous $[K^+]$ during submaximal and maximal one-legged cycling, and one-legged cycling time to fatigue were measured in both the weight-bearing and unloaded legs at baseline, after ULLS, and after 4 weeks of subsequent resistance training (RT). At the same time points resting muscle biopsies were analysed for NKA isoform (α_2, α_3, β_1, β_2) abundance using immunoblotting.

Results: After ULLS, knee extensor isometric strength was decreased by 24% ± 7.3% (Mean ± SD), and by 23% ± 8.2% at 60°/s ($p < 0.05$) in the unloaded leg, while in the weight-bearing leg isometric strength decreased by 16% ± 14.5%, and by 15% ± 10% at 60°/s. Total thigh mass was reduced by 4.43% ± 4.1%, and there was a tendency for reduced average *vastus lateralis* muscle fibre cross sectional area by 20.1% ± 13.9% only in the unloaded leg ($p = 0.056$). Time to fatigue was reduced by 25% ± 19.1%, and no differences were found in venous $[K^+]$ at submaximal or maximal intensities. There was no difference in any of the NKA isoforms, although there was a trend ($p = 0.097$) for a ~1.4 fold increase in β_2 abundance . After four weeks RT, all variables altered by ULLS were returned to baseline, and there was no change in NKA isoform abundance in response to RT.

Conclusions: Despite maladaptations in skeletal muscle endurance and strength, short-term ULLS is not associated with alterations in skeletal NKA isoform abundance in healthy humans. Whether potential alterations in muscle NKA isoform abundance are masked by an increased ratio of sarcolemma to intracellular area due to fibre atrophy; and if a greater duration or severity of disuse is required to decrease NKA content in skeletal muscle are important considerations for future research.