Heat stress treatment rescues denervation-induced mitochondrial loss in skeletal muscle

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Purpose: Traumatic nerve injury or motor neuron disease including ALS results in skeletal muscle denervation and subsequent mitochondrial loss. The purpose of this study is to test whether heat stress treatment can be an effective therapy to counteract denervation-induced mitochondrial loss.

Methods: ICR mice were employed and divided into non-heat stress group or heat stress group. All mice were undergone 10 days of unilateral sciatic nerve transection as surgical denervation. Mice in heat stress group were exposed into a hot environment chamber (40°C, 30 min/day, from day 3 to 9 during the 10 days denervation period). Twenty-four hour after final heat stress treatment, mice were killed and gastrocnemius muscles were collected.

Results: Denervation 1) activated autophagy dependent mitochondrial clearance machinery (mitophagy; increased Parkin and LC3-II in mitochondrial fraction) and 2) decreased mitochondrial content and its related oxidative capacity (protein content of UQCRC2 and enzyme activity of CS). However, daily heat stress treatment rescues denervation-activated mitophagy and subsequent mitochondrial loss.

Discussion: Our observations imply that heat stress may be an effective treatment for nerve injury and disease.

Key words: Mitochondria, Heat stress, Denervation, Mitophagy, Autophagy

Resistance training increases mitochondrial fusion proteins in rat skeletal muscle

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Purpose: to investigate the effect of resistance training, which induces muscle hypertrophy, on the expression of proteins related to mitochondrial dynamics in rat skeletal muscle.

Methods: Resistance training consisted of maximum isometric contraction, which was induced by percutaneous electrical stimulation of the gastrocnemius muscle.

Results: Four weeks of resistance training (three times/week) increased Mfn2 (P<0.05) and Opa1 (P<0.01) protein levels without altering mitochondrial oxidative phosphorylation proteins.

Discussion: These observations suggest that resistance training have little effect on mitochondrial biogenesis, but alters the expression of proteins involved in mitochondrial fusion, which may contribute to mitochondrial quality control.

Key words: resistance training, skeletal muscle, mitochondrial dynamics