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Hsp60 expression in skeletal muscle increases after endurance training

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Adult skeletal muscle is remarkably plastic. Increased contractile activity, such as endurance exercise, elicits multiple signals to activate a large set of genes, leading to phenotypic changes in skeletal muscle, including IIb-to-IIa fiber type switching, enhanced mitochondrial biogenesis, and angiogenesis, to match physiologic capability to functional demand.

Heat shock protein (Hsp) 60 is a mitochondrial protein which plays a key role in the translocation of proteins from the cytoplasm to mitochondria. Apart from its mitochondrial localization, Hsp60 has been detected in the cytoplasm, in the cellular membrane and inside exosomes [1]. In the skeletal muscle the expression of Hsp60 is fibre type specific, being expressed more in type IIa and I fibers compared to IIx and IIb. Upon endurance training Hsp60 increases particularly in type I fibers (unpublished data).

To investigate the role of Hsp60 in skeletal muscle plasticity and mitochondrial biogenesis, we used three experimental models: 1) Forty-eight trained young healthy male mice; 2) in vitro C2C12, where Hsp60 was over expressed by plasmids or repressed by siRNAs; 3) in vivo transfected muscles where Hsp60 was over expressed by plasmids and inhibited by siRNAs.

References

[1] Campanella et al. (2012) The odyssey of Hsp60 from tumor cells to other destinations includes plasma membrane-associated stages and Golgi and exosomal protein-trafficking modalities. PLoS One 7: e42008.

Keywords

Heat shock protein 60, skeletal muscle, endurance exercise, mitochondrial biogenesis.

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