Impaired age-associated mitochondrial translation is mitigated by exercise and PGC-1α

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Author(s) de Smalen, Laura M.; Börsch, Anastasiya; Leuchtmann, Aurel B.; Gill, Jonathan F.; Ritz, Danilo; Zavolan, Mihaela; Handschin, Christoph
Author(s) at UniBasel Handschin, Christoph ; Zavolan, Mihaela ; Börsch, Anastasiya ;
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Sarcopenia, the age-related loss of skeletal muscle mass and function, can dramatically impinge on quality of life and mortality. While mitochondrial dysfunction and imbalanced proteostasis are recognized as hallmarks of sarcopenia, the regulatory and functional link between these processes is underappreciated and unresolved. We therefore investigated how mitochondrial proteostasis, a crucial process that coordinates the expression of nuclear- and mitochondrial-encoded mitochondrial proteins with supercomplex formation and respiratory activity, is affected in skeletal muscle aging. Intriguingly, a robust mitochondrial translation impairment was observed in sarcopenic muscle, which is regulated by the peroxisome proliferator-activated receptor γ coactivator 1 α (PGC-1α) with the estrogen-related receptor α (ERRα). Exercise, a potent inducer of PGC-1α activity, rectifies age-related reduction in mitochondrial translation, in conjunction with quality control pathways. These results highlight the importance of mitochondrial proteostasis in muscle aging, and elucidate regulatory interactions that underlie the powerful benefits of physical activity in this context.
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