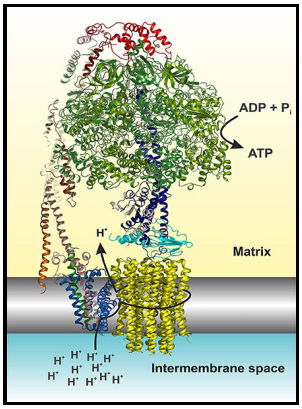
Mitochondrial ATPase - biochemical and molecular genetic analysis

typescript - Mechanisms of muscle atrophy and hypertrophy: implications in health and disease



Description: -
-Mitochondrial ATPase - biochemical and molecular genetic analysis
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Notes: Thesis (Ph.D.) - University of Warwick, 1989.
This edition was published in 1989

Tags: #TSC1 #TSC #complex #subunit #1 #[Homo #sapiens #(human)]

Mechanisms of muscle atrophy and hypertrophy: implications in health and disease

Mechanistically, Atrolnc-1 modulates an inhibitor of NF-kB named A20-binding inhibitor of NF-kB ABIN-1 resulting in increased MuRF1 expression when this lncRNA is overexpressed. Hearing impairment is common in various phenotypes of the mitochondrial DNA A3243G mutation. In the pulsed state, cyanide binds slowly, but with high affinity.

Mechanisms of muscle atrophy and hypertrophy: implications in health and disease

Seattle WA : University of Washington, Seattle; 1993-2021.

2021 ASBMB Annual Meeting

Mitochondrial dysfunction In many catabolic conditions, mitochondrial quality control and the mitochondrial fusion and fission proteins are dysregulated. The ULK1 and the Beclin1 complexes are negatively or positively modulated by mTOR and AMPK, respectively.

Mechanisms of muscle atrophy and hypertrophy: implications in health and disease

Two novel pathogenic mitochondrial DNA mutations affecting organelle number and protein synthesis. The mitochondrion: a perpetrator of acquired hearing loss. NF-kB and the atrophy program are also positively modulated by tumor necrosis factor-like weak inducer of apoptosis TWEAK , which is a member of the TNF superfamily.

Mitochondrial DNA: MedlinePlus Genetics

Researchers have not determined how mutations in these genes lead to hearing loss. Indeed, ACL is phosphorylated and activated by AKT resulting in an improvement of mitochondrial respiration and ATP production.

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The MT-TL1, MT-TK, and MT-TE genes provide instructions for making tRNA molecules, which are essential for protein production within mitochondria. Altogether these findings suggest that mTORC1 has a major role in muscle homeostasis but may not be the exclusive regulator of protein synthesis and that autophagy in muscles is controlled by mTORC1 independent and dependent pathways.

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In the ubiquitin—proteasome system, proteins are targeted for degradation by the 26S proteasome through covalent attachment of a chain of ubiquitin molecules.

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