



**Mitochondrial disease**

Primary mitochondrial diseases are caused by pathogenic variants affecting mitochondrial genes encoded by nuclear or mitochondrial DNA.

The ability of mitochondria to generate energy via oxidative phosphorylation (OXPHOS) is typically impaired in mitochondrial diseases.

Examples include: Leigh syndrome and MELAS.

**Neurodegenerative diseases**

Neurodegeneration involves progressive loss of neuronal function underpinned by a complex etiology.

Mitochondrial dysfunction and defective autophagy are often hallmarks of neurodegeneration.

Inflammation and mitochondria-specific autoimmunity is emerging as an important component of neurodegeneration, highlighting the interplay between the immune and nervous systems.

Examples include: Parkinson's disease, Huntington's disease, Alzheimer's disease and amyotrophic lateral sclerosis (ALS).

**Axonal transport defects**

Retrograde (towards cell body) and anterograde (towards synapses) transport of organelles and vesicles is essential for neuronal function.

Mitochondrial retrograde transport enables delivery of dysfunctional mitochondria to lysosomes. Anterograde transport enables their delivery to energetic synapses and dendritic terminals.

Impaired mitochondrial transport is associated with neuroinflammation, and is proposed to be both a cause and consequence of neurological disease.

Examples include: amyotrophic lateral sclerosis, Alzheimer's disease, and Charcot-Marie-Tooth (CMT) disease.

**Disorders of mitochondrial dynamics**

Mitochondria are dynamic organelles that undergo fission and fusion events in response to changing metabolic requirements and environmental cues.

Mitochondrial dynamics can regulate the distribution of metabolites, and enable individual mitochondria to be removed from the mitochondrial pool.

Disruption of dynamics is mainly caused by pathogenic variants in genes involved in fission and fusion (e.g. *DNM1L*, *MFN2* and *OPA1*).

**Neuromuscular junction defects**

Synaptic transmission at neuromuscular junctions underpins communication between the nervous system and muscles, and relies on mitochondria for calcium buffering, metabolite homeostasis and energy generation.

Defective mitochondrial activity at synapses may cause neuromuscular junction defects, leading to muscle weakness.

Examples include: mitochondrial myopathy and peripheral neuropathy.

# Cell Function And Disease

**RJ Shavelson**



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