Mitochondrial Mechanisms Of Degeneration And Repair In Parkinsons Disease

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Mitochondrial Mechanisms Of Degeneration And
Mechanisms of mitochondrial dysfunction and their impact on age-related macular degeneration. Oxidative stress-induced damage to the retinal pigment epithelium (RPE) is considered to be a key factor in age-related macular degeneration (AMD) pathology.

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Mechanisms of mitochondrial dysfunction and their impact on age-related macular degeneration 1. Introduction to RPE degeneration and development of AMD. AMD is associated with several environmental and genetic... 2. RPE mitochondria damage and dysfunction during normal aging. Mitochondria are ...

Mechanisms of mitochondrial dysfunction and their impact ...
Mitochondrial Mechanisms of Degeneration and Repair in Parkinson's Disease. Editors: Buhlman, Lori M. (Ed.) Free Preview. Summarizes theories of how poor mitochondrial function and morphology contribute to neurodegeneration; Discusses both sporadic and familial forms of Parkinson's disease ...

Mitochondrial Mechanisms of Degeneration and Repair in ...
Although impaired mitochondrial function is implicated in degeneration of aging muscle, the majority of research to date has focused upon changes within the muscle fibers, and generally, our thinking about how changes in mitochondria within the motoneurons impact aging muscle is less evolved.

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In this respect, mitochondria are implicated in aging and age-related neurodegenerative disorders, and are also likely key to aging muscle changes through their direct effects in muscle fibers and through secondary effects mediated by mitochondrial impairments in motoneurons.

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In fact, the most common approach to the generation of animal and cell-culture models of idiopathic Parkinson’s disease involves exposure to mitochondrial toxins. Even more compelling is the fact that most familial patients harbor genetic mutations that cause disruptions in normal mitochondrial morphology and function.

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systems, and biogenesis of mitochondria. The precise cause of RPE degeneration and the onset and progression of AMD are not fully understood. However, mitochondria dysfunction, increased reactive oxygen species (ROS) production, and mitochondrial DNA (mtDNA) damage are observed together with increased protein

Mechanisms of mitochondrial dysfunction and their impact ...
Manifestations of mitochondrial dysfunction with aging include impaired respiratory function, elevated reactive oxygen species production, and increased susceptibility to permeability transition,...

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Although mitochondrial dysfunction is intimately related to axonal degeneration following nerve injury, the molecular mechanisms of mitochondrial swelling and its mechanistic relation to axonal degeneration are largely unknown. Previous studies have demonstrated that axonal degeneration in the injured peripheral nerves shows two morphologically distinct phases: (1) A latency period (~24h), in which the morphology of axonal cytoskeletons seems unchanged, followed by (2) an execution period ...

Mitochondrial swelling and microtubule depolymerization ...
Oxidative stress-induced damage to the retinal pigment epithelium (RPE) is considered to be a key factor in age-related macular degeneration (AMD) pathology. RPE cells are constantly exposed to...

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Age-related macular degeneration is a complex disease, and for the majority of complex traits, the mechanisms underlying their pathogenesis are not exactly known . These mechanisms likely include interactions between genetic, environmental and lifestyle risk factors that may lead to aberrant processes occurring in the retina . Advanced age is by definition the main AMD risk factor.

Role of Mitochondrial DNA Damage in ROS-Mediated ...
A newly discovered self-destructive mechanism in mitochondria, the cells’ powerhouses, may be one of the first deficits leading to motor neuron degeneration associated with toxic TDP-43 clumps — a hallmark of amyotrophic lateral sclerosis (ALS).

Mitochondrial 'Suicide' May Trigger Neurodegeneration in ...
Here, we show that disruption of mitochondrial membrane potential leads to axonal NMNAT2 depletion in mouse sympathetic neurons, increasing the substrate-to-product ratio (NMN/NAD) of this NAD-synthesising enzyme, a metabolic fingerprint of Wallerian degeneration. The mechanism appears to involve both impaired NMNAT2 synthesis and reduced axonal transport.

Mitochondrial impairment activates the Wallerian pathway ...
The specific degeneration of neurons in the substantia nigra pars compacta causes rigidity, tremor, and bradykinesia, all of which are hallmark symptoms of Parkinson’s disease. One of the potential molecular factors that might induce the loss of dopaminergic neurons is an elevated level of reactive oxygen species which causes cell damage over ...

Dopamine Metabolism and Reactive Oxygen Species Production ...
K. Kaarniranta, H. Uusitalo, J. Blasiak et al., “Mechanisms of mitochondrial dysfunction and their impact on age-related macular degeneration,” Progress in Retinal and Eye Research, pp. 100858–100858, 2020. View at: Publisher Site | Google Scholar

Autophagy in Age-Related Macular Degeneration: A ...
Fen induces oxidative stress in primary neurons in vitro. As a first step toward elucidating the mechanisms of neuronal degeneration by Fen in vivo, primary cultured neurons from C57BL/6 mice were ...

Fenpropathrin induces degeneration of dopaminergic neurons ...
This study is designed to understand mechanisms of retinal degeneration in patients with Bietti’s crystalline dystrophy (BCD) by using pluripotent stem cells and genomic editing approaches. It revealed that excessive poly-unsaturated fatty acid (PUFA) is the key to damaging retinal mitochondrial functions. Application of gene therapy can effectively alleviate BCD.

PSCs Reveal PUFA-Provoked Mitochondrial Stress as a ...
Our results show that gp120 causes neuronal apoptosis and axonal degeneration through two, independent and spatially separated mechanisms of action: (i) an indirect insult to cell bodies, requiring the presence of Schwann cells, results in neuronal apoptotic death and subsequent axonal degeneration; (ii) a direct, local toxicity exerted on axons through activation of mitochondrial caspase pathway that is independent of cell body.