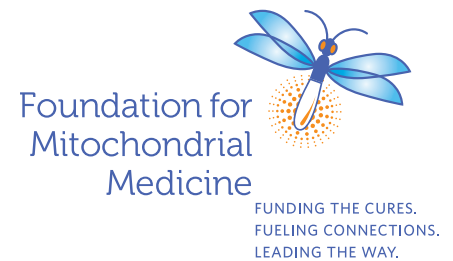


Parkinson's & Mitochondrial Disease

Mitochondrial disease can look like a number of better known diseases: Autism, Parkinson's, Alzheimer's, Lou Gehrig's disease (ALS), muscular dystrophy and chronic fatigue, among others. And it's this web of complexity and connectivity that makes mitochondrial disease research valuable to so many. Research shows that mitochondrial dysfunction is often at the crux of these more commonly recognized diseases.



Recent findings implicate mitochondrial dysfunction, oxidative damage, abnormal protein accumulation and protein phosphorylation as key molecular mechanisms compromising dopamine neuronal function and survival as the underlying cause of pathogenesis in both sporadic and familial PD.

Parkinson's Disease May Stem From an Energy Crisis in the Brain

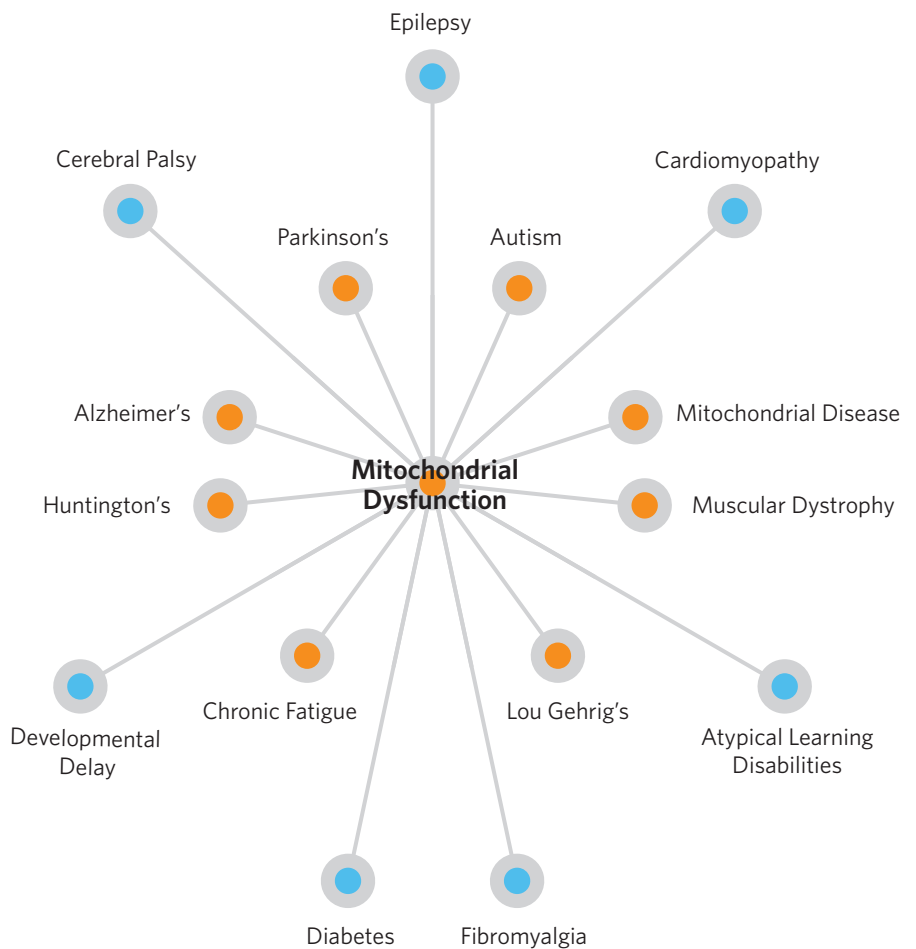
Parkinson's disease may stem from an energy crisis in the brain, years before symptoms appear.

If the research pans out, it points to a possible new approach for Parkinson's: Giving a boost to a key power switch inside brain cells in hopes of slowing the disease's inevitable march instead of just treating symptoms. At issue are little power factories inside cells, called mitochondria. Increasingly, scientists suspect that malfunctioning mitochondria play some role in a list of degenerative brain diseases.

After all, brain cells are energy hogs, making up about 2 percent of body weight yet consuming about 20 percent of the body's energy.

FAMILIAR CONNECTIONS

Mitochondrial dysfunction is a central element of familiar diseases.



Research shows that mitochondrial dysfunction is often a central element of these more commonly recognized diseases. Studies and reports indicate the "orange" ones are more influenced.

A cure for mitochondrial disease could impact cures for Autism, Parkinson's, Alzheimer's and Muscular Dystrophy

So a power drain could trigger some serious long-term consequences. “It could be a root cause” of Parkinson’s,

says Dr. Clemens Scherzer of Boston’s Brigham and Women’s Hospital and Harvard University.

The Huffington Post, Feb 15, 2011

Mitochondrial dysfunction in Parkinson’s disease.

Many lines of evidence suggest that mitochondrial dysfunction plays a central role in the pathogenesis of Parkinson’s disease

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Abstract

It is clear from a striking convergence of human tissue studies, neurotoxin models, and genetic models that mitochondrial dysregulation plays a central pathogenic role in Parkinson’s disease (PD) and related neurodegenerative conditions. Impaired mitochondrial quality could result from both increased damage and decreased ability to repair or clear damaged mitochondria. In particular, common deficits in mitochondrial respiratory chain function, oxidative stress, morphology/dynamics, and calcium handling capacities have been described in multiple PD model systems employing complex I inhibitors, 6-hydroxydopamine and molecular manipulation of Parkinsonian genes including alpha-synuclein, PTEN-

induced kinase 1, Parkin, DJ-1, and, to a lesser extent, leucine rich repeat kinase 2. The most recent and exciting work implicates alterations in the regulation of macroautophagy and likely of selective mitophagic clearance of damaged mitochondria, although additional studies are needed to resolve some issues in this area. Future studies emphasizing the normal mitoprotective function(s) of proteins associated with recessive loss-of-function causes of familial PD, as well as compensatory mechanisms operating in their absence, may offer particularly valuable insights into strategies to enhance mitochondrial health.

J Alzheimers Dis. 2010;20 Suppl 2:S325-34. 

Mitochondrial dysfunction in Parkinson’s disease: pathogenesis and neuroprotection.

by: Ross B. Mounsey, Peter Teismann

Abstract

Mitochondria are vitally important organelles involved in an array of functions. The most notable is their prominent role in energy metabolism, where they generate over 90% of our cellular energy in the form of ATP through oxidative phosphorylation. Mitochondria are involved in various other processes including the regulation of calcium homeostasis and stress response. Mitochondrial complex I impairment and subsequent oxidative stress have been identified as modulators of cell death in experimental models of Parkinson’s disease (PD). Identification of specific genes which are involved in the rare familial forms of PD has further augmented the understanding and elevated the role mitochondrial dysfunction is thought to have in disease pathogenesis. This paper provides a review of the role mitochondria may play in idiopathic PD through

the study of experimental models and how genetic mutations influence mitochondrial activity. Recent attempts at providing neuroprotection by targeting mitochondria are described and their progress assessed.

Parkinson’s disease, Vol. 2011(2010) doi:10.4061/2011/617472
Key: citeulike:8637550 