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Clinical effects of chemical exposures on mitochondrial function

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Abstract

Mitochondria are critical for the provision of ATP for cellular energy requirements. Tissue and organ functions are dependent on adequate ATP production, especially when energy demand is high. Mitochondria also play a role in a vast array of important biochemical pathways including apoptosis, generation and detoxification of reactive oxygen species, intracellular calcium regulation, steroid hormone and heme synthesis, and lipid metabolism. The complexity of mitochondrial structure and function facilitates its diverse roles but also enhances its vulnerability. Primary disorders of mitochondrial bioenergetics, or Primary Mitochondrial Diseases (PMD) are due to inherited genetic defects in the nuclear or mitochondrial genomes that result in defective oxidative phosphorylation capacity and cellular energy production. Secondary mitochondrial dysfunction is observed in a wide range of diseases such as Alzheimer's and Parkinson's disease. Several lines of evidence suggest that environmental exposures cause substantial mitochondrial dysfunction. Whereby literature from experimental and human studies on exposures associated

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