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Oxidative stress, mitochondrial dysfunction and Alzheimer's disease

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In general, oxidative stress is the overpowering of anti-oxidant defense system by oxidative system caused by overproduction of reactive oxygen species (ROS). At present, many lines of evidence from animal and human studies suggest that mitochondrial dysfunction is the main source of ROS that have a central role in pathogenesis of neurodegenerative diseases. Mitochondria are the only intracellular organelles that contain their own DNA (mitochondrial DNA; mtDNA) in multiple copies. Although most mitochondrial proteins are encoded by nuclear DNA, mtDNA encodes 13 polypeptide components of the respiratory chain that is located in mitochondrial inner membrane. Biochemical, ultrastructural and genetic studies confirmed the mitochondrial dysfunction / oxidative stress in patients with Alzheimer's disease. Moreover, it has been shown that Aβ is present in mitochondria and through interaction with mitochondrial proteins promotes oxidative stress and apoptosis. In addition, γ-secretase complex (containing PS1) has been recently localized to mitochondria and shown to have the ability to actively cleave APP in the mitochondrial membrane. According to recent animal data oxidative stress is present at very early stage of disease prior to the appearance of Aβ plaques suggesting that oxidative stress is a primary rather a secondary event in AD pathogenesis and anti-oxidant therapies hold great promise.