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Mitochondrial alterations in Alzheimer's disease

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Abstract

Morphological alterations of mitochondria may be related to metabolic and energy deficiency in neurons in Alzheimer's disease and other neurodegenerative disorders. Mitochondrial dysfunction is also a hallmark of beta peptide induced neuronal toxicity in Alzheimer's disease. A general change in glucose utilization, increased oxidative stress, and Ca;{2+} deregulation are additional metabolic defects in the AD brain that may also be associated with defective mitochondrial function the result is a cycle of increased mitochondrial dysfunction causing increased oxidative damage until the cellular energy supply falls below the threshold for cellular survival. In a series of studies on the morphological and morphometric estimation of mitochondria in Alzheimer's disease, by electron microscopy we noticed substantial morphological and morphometric changes in the neurons of the hippocampus, the acoustic cortex, the frontal cortex, the cerebellar cortex, the climbing fibers, the thalamus, the globus pallidus, the red nucleus and the locus coeruleus. The morphological alterations consisted of considerable changes of the mitochondrial cristae, accumulation of osmiophilic material, and decrease of their size, in comparison with the normal controls. Mitochondrial alterations were particularly prominent in neurons, which showed loss of dendritic spines and abbreviation of the dendritic arborization. The ultrastructural study of large number of neurons in the thalamus and the red nucleus revealed that the mitochondrial alterations did not coexist with cytoskeletal pathology and accumulation of amyloid deposits, though they were prominent in neurons, which demonstrated fragmentation of the cisternae of the Golgi apparatus. Morphometric analysis showed that mitochondria are significantly reduced in Alzheimer's disease. The relationship between the site and extent of mitochondrial abnormalities and the synaptic alterations suggests an intimate and early association between these features in Alzheimer's disease.

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