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Mitochondria in the aging brain and neurodegeneration

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Aging is defined as a progressive time-related accumulation of changes responsible for or at least involved in the increased susceptibility to disease and death. The brain is a highly metabolic tissue, and neurons in the central nervous system have an intense demand for mitochondria. Mitochondria were placed at the center of the 'free-radical theory of aging', because these exceptional organelles are not only the main producers of energy in the cells, but also to main source of reactive oxygen species. Mitochondria provide most of the ATP for cellular reactions. ATP production in mitochondria is coupled to an electron transport system in which the passage of electrons down the various electron carriers is associated with the transport of protons from the matrix into the intermembrane space. The majority of these protons reenter the mitochondrial matrix by the ATP synthase, thereby generating ATP. However, approximately 20% of mitochondrial oxygen consumption is not coupled to ATP production, and protons enter the matrix through the phospholipid bilayer and through uncoupling proteins, generating heat. Mitochondrial metabolism is also responsible for the majority of the ROS production in cells. ROS is formed when unpaired electrons avoid the electron transport chain and react with molecular oxygen, which generate superoxide. Superxide can react with DNA, protein, and lipids; Intracellular plays an important role in signaling; And both are related to neurodigenerative diseases and aging. ROS can also react with NO, generating reactive nitrogen species (RNS). The repair of physiologic ROS levels is critical to normal cell functions, and thus prolonged increases in mitochondrial activity can increase ROS levels and alter intracellular physiologic set points. Studies of aging brain mitochondria consistently report reductions of complex I activity, complex IV activity, and increased ROS production. Other age-related mitochondrial changes include reduced membrane potential and increased size. mtDNA mutations may contribute to age-related mitochondrial decline. mtDNA deletions accumulate with age in many tissues, especially brain. Mitochondria are altered in brains of persons with certain neurodegenerative diseases. There are activity reductions of complex I in Parkinson's disease (PD), cytochrome oxidase in Alzheimer's disease (AD), and multiple electron transport chain (ETC) enzymes in Huntington's disease (HD). Mitochondrial dysfunction occurs in amyotrophic lateral sclerosis and progressive supranuclear palsy. Leber's hereditary optic neuropathy (LHON), a focal degeneration of the optic nerves, arises from mutations in mtDNA-encoded complex I genes and is associated with complex I dysfunction. The status of mitochondrial biogenesis in AD is unclear. In the brain, mitochondrial function decrease with age and this functional decrease associates with increased mitochondrial biogenesis.

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