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Long-term melatonin administration protects brain mitochondria from aging.

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Abstract

We tested whether chronic melatonin administration in the drinking water would reduce the brain mitochondrial impairment that accompanies aging. Brain mitochondria from male and female senescent prone (SAMP8) mice at 5 and 10 months of age were studied. Mitochondrial oxidative stress was determined by measuring the levels of lipid peroxidation and nitrite, glutathione/glutathione disulfide ratio, and glutathione peroxidase and glutathione reductase activities. Electron transport chain activity and oxidative phosphorylation capability of mitochondria were also determined by measuring the activity of the respiratory chain complexes and the ATP content. The results support a significant age-dependent mitochondrial dysfunction with a diminished efficiency of the electron transport chain and reduced ATP production, accompanied by an increased oxidative/nitrosative stress. Melatonin administration between 1 and 10 months of age completely prevented the mitochondrial impairment, maintaining or even increasing ATP production. There were no major age-dependent differences between males in females, although female mice seemed to be somewhat more sensitive to melatonin treatment than males. Thus, **melatonin administration as a single therapy maintained fully functioning brain mitochondria during aging**, a finding with important consequences in the pathophysiology of brain aging.

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