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NEUROPROTECTIVE EFFECTS OF THE CYCLOPHILIN D DEFICIENCY IN B-AMYLOID MOUSE MODEL

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Text of the abstract

INTRODUCTION: The accumulation of amyloid β (A β) and tau proteins in the brain is the fundamental of Alzheimer disease. In the early phase of the disease the A β binds to the mitochondrial proteins and cause bioenergetic failure and increased oxidative stress, which can induce neuronal death. The mitochondrial cyclophilin D (CypD) protein is a major component and regulator of mitochondrial permeability transition pore. In our experiments the gene deletion of CypD (CypD-KO) protein has a neuroprotective effect in the present of

Aß.

AIM: Our goal was to cross transgenic mice overexpressing a mutant human form of amyloid precursor protein and mutated presenilin1 with CypD-KO. In our experiments mitochondrial functions were examined.

METHODS: Our experiments were done on isolated brain mitochondria from 4 different mice (wild-type, Alzheimer's mouse model (Alz), CypD-KO and crossed Alz/CypD). The mitochondrial respiration was measured by Clark-type electrode. The mitochondrial H₂O₂ production with Amplex UltraRed fluorimetry. The ATP production was measured with a linked spectrophotometric assay.

RESULTS: During our measurements the Alz mice showed decreased O₂ consumption, ATP production and elevated H₂O₂ production versus the wild type control. The bioenergetic parameters of Alz/CypD samples were improved compared to Alz. The Alz/CypD results were comparable to CypD.

CONCLUSIONS: From our investigations of Alzheimer's mouse model we were led to conclude that mitochondrial functions were impaired, but knocking out the CypD protein improved bioenergetic parameters. Our results showed that inhibitors of CypD protein can be potential drug candidates in neurodegenerative diseases.

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