

LECTURE PRESENTATION



Identifying an APP-binding protein in neuronal cell death

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From 2011 International Conference on Molecular Neurodegeneration Shanghai, China. 22-24 September 2011

Background

Apoptosis is an essential cellular process involved in multiple diseases and a major pathway for neuronal death in neurodegeneration. The detailed signaling events/pathways that lead to apoptosis, especially in neurons, require further elucidation.

Results

Here we find that a mitochondrial solute carrier family protein, appoptosin, induces reactive oxygen species release and intrinsic caspase-dependent apoptosis. The physiological function of appoptosin is to transport/ exchange glycine/5-amino-levulinic acid across the mitochondrial membrane for heme synthesis. Alzheimer's β -amyloid precursor protein interacts with appoptosin and modulates appoptosin-induced apoptosis. Levels of appoptosin are upregulated in brain samples from Alzheimer's disease and infarct patients and in rodent stroke models, as well as in cells treated with β -amyloid $(A\beta)$ and glutamate. Downregulation of appoptosin prevents the cell death and caspase activation caused by glutamate or $A\beta$ insults.

Conclusion

Our study identifies appoptosin as a crucial player in apoptosis and a novel proapoptotic protein involved in neuronal cell death, providing a possible new therapeutic target for neurodegenerative disorders and cancers.

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doi:10.1186/1750-1326-7-S1-L22 Cite this article as: Zhang and Xu: Identifying an APP-binding protein in neuronal cell death. Molecular Neurodegeneration 2012 7(Suppl 1):L22.

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