Yu et al. Molecular Neurodegeneration 2012, 7(Suppl 1):S33
http://www.molecularneurodegeneration.com/content/7/S1/S33

MOLECULAR NEURODEGENERATION

MEETING ABSTRACT Open Access

Protective effect of Curcumin on chronic cerebral ischemia by altering expression of α- synuclein in 2VO model

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

Background

Previous studies have shown that natural compound Curcumin can improve some biological effects induced by chronic cerebral hypoperfusion and may represent a target for treatment. α- synuclein oligomerization and aggregation are considered to have a role in the pathogenesis of brain ischemia/reperfusion. However, the effects of Curcumin on α- synuclein in chronic cerebral hypoperfusion are poorly understood. This study aims to observe the effect of Curcumin on chronic cerebral ischemia model in rats and investigate change of α-synuclein induced by Curcumin.

Methods

The chronic cerebral ischemia was produced in male Sprague-Dawley rats by permanent occlusion of bilateral common carotid arteries (2VO). Animals were randomly divided into 5 groups: normal control group, sham-operated group, 2VO+DMSO group, 2VO+Curcumin 100mg/kg group, 2VO+Curcumin 50mg/kg group. After surgery, all animals were injected intraperitoneally with DMSO solution of Curcumin or a same volume of normal DMSO. Each group was injected once daily for four consecutive weeks. After the completion of the behavioral testing, rats were sacrificed. Hematoxylin-eosin staining and Nissl staining were carried out in section. The expressions of α- synuclein protein in hippocampus were detected by immunohistochemistry.

Results

The chronic cerebral ischemia in rats resulted in a significant pathological change in the hippocampus CA1 area, including: loss of pyramidal cell, shrinkage of nuclei, dark staining of neurons, loss of Nissl body and glial proliferation as compared with sham-operated rats. The administration of different doses of Curcumin attenuated neuronal injury in rats induced by chronic cerebral ischemia along with the concomitant increased the numbers of α-synuclein -positive cells.

Conclusion

Our data demonstrated that the neuroprotective effect of Curcumin involved in increasing the protein levels of α-synuclein induced by ischemia.

Acknowledgments

This study was supported by the National Natural Science Foundation of China (No.30973154), Chongqing Science and Technology Commission Foundation (No.2009BB5270), and Chongqing Municipal Education Commission Foundation (No.KJ090301).

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Published: 7 February 2012

doi:10.1186/1750-1326-7-S1-S33
Cite this article as: Yu *et al.*: Protective effect of Curcumin on chronic cerebral ischemia by altering expression of α- synuclein in 2VO model. *Molecular Neurodegeneration* 2012 **7**(Suppl 1):S33.

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