

MEETING ABSTRACT

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Curcumin protects SH-SY5Y cells from oxidative stress by up-regulating HO-1 via Phosphatidylinositol 3 Kinase/Akt/Nrf-2 and down-regulating HO-2

Wenke Yin^{1,2,3†}, Xiong Zhang^{1,2†}, Xiaodong Shi^{1,2,3}, Yu Li^{1,2,3*}

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Background

Oxidative stress is considered to have a causative role in the development of central nervous system diseases, such as Alzheimer's disease, Parkinson's disease, tumor, etc. Curcumin, a polyphenol extracted from rhizomes of the plant *Curcuma longa*, is widely reported to have diverse anti-oxidative stress effects, but the underlying mechanism has not been fully elucidated. We investigated the mechanism underlying the neuroprotective properties of curcumin in human neuroblastoma SH-SY5Y cells subjected to oxidative stress.

Methods

The cells were treated with curcumin at different concentration (0, 1.25, 5, 20 μ M) for 24, 48, 72 h, and/or with PI3K inhibitor LY294002 or Nrf-2 siRNA, the concentration- and time-dependent protection of curcumin against H₂O₂-induced toxicity was measured as ROS production and reduced cell growth. RT-PCR and Western blot were applied for detecting the expression of HO-1, HO-2, PI3K, AKT and Nrf-2 at mRNA and protein levels, also, the production of ferritin was measured by WB.

Results

The expression of HO-1 and the production of ferritin were significantly increased, but the expression of HO-2 was decreased. Furthermore, curcumin could significantly induce the protein and mRNA expression of

PI3K, AKT and Nrf-2 ($P < 0.05$), while the protective effects of curcumin were prevented by PI3K inhibitor LY294002 or Nrf-2 siRNA.

Conclusion

Taken together, the data show that the cytoprotection of curcumin against oxidative stress in SH-SY5Y cells is by up-regulating HO-1 expression via PI3K/AKT/Nrf-2 intracellular signaling pathway and down-regulating HO-2 expression. Which suggests that curcumin is beneficial in the prevention and treatment of some CNS disease.

Author details

¹Department of Pathology, Chongqing, Chongqing Medical University, 400016, China. ²Chongqing Key Laboratory of Neurobiology, Chongqing Medical University, Chongqing, 400016, China. ³Institute of Neuroscience, Chongqing Medical University, Yuzhong District Yuanjiagang Yixueyuan Road No.1, Chongqing, 400016, China.

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* Correspondence: liyu100@163.com

† Contributed equally

¹Department of Pathology, Chongqing, Chongqing Medical University, 400016, China

Full list of author information is available at the end of the article