Feng Jinghan, J Neurol Neurophysiol 2017, 8:5 (Suppl)
DOI: 10.4172/2155-9562-C1-056

Neuroprotection of naringin against cerebral ischemia-reperfusion injury through attenuating reactive nitrogen species-mediated autophagic cell death

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Aims: Reactive nitrogen species (RNS) largely generate at the reperfusion phase after cerebral ischemia, triggering a series of cascades to exacerbate brain damage. Emerging evidences indicate that RNS are sufficient to induce excessive autophagy under pathological condition. Unlike basal level autophagy, over-activated autophagy can lead to cell death at least partly attributed to energy depletion. In this study, we tested whether naringin, a non-toxic bioflavonoid could attenuate brain damage by inhibiting RNS-mediated autophagic cell death during cerebral ischemia-reperfusion.

Methods: Middle cerebral artery occlusion (MCAO) for 2 hours followed by reperfusion (RP) for 22 hours in vivo and oxygen-glucose-deprivation (OGD) for 10 hours and reoxygenation (RO) for 14 hours in vitro were used to mimic cerebral ischemia-reperfusion injury. The effects of naringin on reducing cell death, decreasing peroxynitrite and autophagy level were investigated.

Results: Naringin reduced SH-SY5Y cell death and decreased superoxide anion, nitric oxide and peroxynitrite level after OGD/RO in vitro. Naringin dose-dependently reduced neurological deficit score, infarct volume and apoptotic cell death compared with vehicle-treated MCAO/RP group in vivo. Naringin decreased nitric oxide level in serum, inhibited NADPH oxidase activity, the expression of p47phox and p67phox, and also reduced inducible nitric oxide synthase and 3-nitrotyrosine level of brains in vivo. Naringin significantly reversed autophagy activation in MCAO/reperfused rat brains, similar to peroxynitrite decomposition catalyst-FeTMPyP.

Conclusion: Naringin might be a promising neuroprotective agent against cerebral ischemia-reperfusion injury and its mechanisms could be at least partially attributed to attenuating RNS-mediated autophagic cell death.

Biography

Feng Jinghan is currently a PhD student from The University of Hong Kong, Hong Kong. Her research focuses to explore the roles and in-depth mechanisms of autophagy in ischemic stroke and aims to raise some potential therapeutic targets. Further, she has made efforts to screen some effective natural compounds to regulate autophagic process for better outcomes. She has more than 3 years of experience on establishing rat/mouse middle cerebral artery occlusion model and performing a series of molecular experiments.

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