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Neuroprotective effect of astaxanthin on H(2)O(2)-induced neurotoxicity in vitro and on focal cerebral ischemia in vivo.

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Abstract

Astaxanthin (AST) is a powerful antioxidant that occurs naturally in a wide variety of living organisms. Much experimental evidence has proved that AST has the function of eliminating oxygen free radicals and can protect organisms from oxidative damage. The present study was carried out to further investigate the neuroprotective effect of AST on oxidative stress induced toxicity in primary culture of cortical neurons and on focal cerebral ischemia-reperfusion induced brain damage in rats. AST, over a concentration range of 250-1000nM, attenuated 50μM H(2)O(2)-induced cell viability loss. 500nM AST pretreatment significantly inhibited H(2)O(2)-induced apoptosis measured by Hoechst 33342 staining and restored the mitochondrial membrane potential (MMP) measured by a fluorescent dye, Rhodamine 123. In vivo, AST prevented cerebral ischemic injury induced by 2h middle cerebral artery occlusion (MCAO) and 24h reperfusion in rats. Pretreatment of AST intragastrically twice at 5h and 1h prior to ischemia dramatically diminished infarct volume and improved neurological deficit in a dose-dependent manner. Nissl staining showed that the neuronal injury was significantly improved by pretreatment of AST at 80mg/kg. Taken together, these results suggest that pretreatment with AST exhibits noticeable neuroprotection against brain damage induced by ischemia-reperfusion and the antioxidant activity of AST maybe partly responsible for it.

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