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Neuroprotective effects of honokiol against beta-amyloid-induced neurotoxicity via GSK-3β and β-catenin signaling pathway in PC12 cells.

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Abstract

Beta-amyloid (Aβ) accumulation, one of the most important pathogenic traits of Alzheimer's disease (AD), has been reported to induce neurotoxicity in vitro as well as in vivo. Honokiol, isolated from the bark of Magnolia officinalis, has neuroprotective effects in different models of AD in vivo and in vitro. However, the exact mechanism for its neuroprotective effect is not well understood. The present study aimed to investigate the molecular mechanisms underlying the protective action of honokiol against Aβ1-42-induced neurotoxicity in cultured rat pheochromocytoma (PC12) cells. The results revealed that honokiol protected PC12 cells from Aβ1-42 induced cytotoxicity with increases in cell viability, GSH production and Bcl-2 expression, but decreases in the release of lactate dehydrogenase and cytochrome c, the amount of DNA fragmentation and MDA level, as well as Bax expression. Mechanistic study showed that honokiol could inhibit the activation of glycogen synthase kinase (GSK)-3β, attenuate the nuclear accumulation of β-catenin and suppress the phosphorylation of β-catenin (Ser33/Ser37/Thr41 site) in the Aβ1-42-treated PC12 cells. These results indicate that the anti-oxidative and anti-apoptotic effects of honokiol in Aβ1-42-treated PC12 cells may be mediated, at least in part, by regulation the GSK-3β and β-catenin signaling pathways.

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Publication type, MeSH terms, Substances

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