Protective effects of Angelica sinensis extract on amyloid beta-peptide-induced neurotoxicity

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摘要.

Abstract

The protective effects of alcohol extract from the root of Angelica sinensis (AS) on beta-amyloid peptide (Abeta)-induced toxicity and the mechanism of these effects were investigated. Abeta is a pathological hallmark of Alzheimer's disease; it decreased viability of Neuro 2A cells in a concentration-dependent manner with IC(50) of 14.9 microM. AS extract resulted in dose-dependent anti-Abeta toxicity according to MTT assay. Reactive oxygen species (ROS) analysis revealed a significant production of hydrogen peroxide, decreased glutathione (GSH) levels and increased lipid peroxidation (TBARS value) in the Abeta-treated Neuro 2A cells. The Abeta-treated cells also showed a significant decline in the mitochondrial transmembrane potential (DeltaPsim) and increase in the mitochondrial volume, and portions of the cytoplasm were sequestered by a membrane-bound vacuole. The malfunctions of Neuro 2A cells caused by Abeta were attenuated using AS extract. The AS extract protected cell viability against Abeta-induced oxidative damage (ROS, TBARS, and GSH contents) and rescued the DeltaPsim levels in a dose-dependent manner: the dosages of 25, 50, 100, and 200 microg/ml recovered 77%, 87%, 102%, and 105% of DeltaPsim, respectively. AS extract also recovered the enlarged mitochondria mass with dosages from 25 to 200 microg/ml. The results of this study demonstrated that AS extract possessed the activity to prevent the neurotoxicity induced by Abeta-associated oxidative stress, implying that AS has a potential role in the prevention of Alzheimer's diseases