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Vitis Amurensis protects amyloid β protein-induced neurotoxicity in cultured neurons

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Vitis amurensis (VA; Vitaceae) has long been used for oriental herbal medicine. Studies have reported that roots and stems of VA have anti-inflammatory and antioxidative effects. Amyloid β protein $(A\beta)$ or $A\beta$ peptide fragments have been suggested to play an important role in the pathogenesis of Alzheimer's disease (AD). In the present study, the protective effect of ethanol extract of stems and leaves of VA against AB (25-35)-induced neurotoxicity was examined in primary cultured rat cortical neurons. To assess AB (25-35) (10 µM)-induced neuronal cell death, 3-[4,5-dimethylthiazol-2-yl]-2,5-diphenyl-tetrazolium bromide (MTT) assay and Hoechst 33342 staining were performed. VA (1-10 µg/ml) showed concentration-dependent inhibitory effect on A β (25-35)-induced neuronal cell death. VA (10 µg/ml) completely inhibited 10 µM A β (25-35)-induced increase of cytosolic Ca²⁺ concentration ([Ca²⁺]_c), measured by fluorescent dye, fluo 4-AM VA also blocked the A β (25-35)-induced generation of reactive oxygen species (ROS). In addition, viniferin (0.1-3 μM), isolated from VA as an active component, inhibited Aβ (25-35)-induced neuronal cell death. These results suggest that VA prevents Aβ (25-35)-induced neurotoxicity by inhibiting the increase of [Ca²⁺]_c and ROS and may have a promising role in prevention of AD.

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Neuroprotective effect of *Glycyrrhizae* Radix against AB (25-35)-induced neuronal cell death in cultured rat cortical neurons.

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Glycyrrhizae Radix (GR), the root of Glycyrrhiza uralensis (Lycophodiaceae), has been widely used in Chinese medicine and food and has a history of consumption for the past 6000 years. Many compounds showing various bioactivities have been isolated from GR. Especially, anti-dementia and anti-ischemic effects of its components have been reported in animal models. However, the neuroprotective effects of GR have not been studied in in vitro. To observe neuroprotective effects of GR in the cellular level, amyloid β protein (A β), which is involved in the pathology of alzheimer's disease (AD), was used in the present study. We demonstrated that GR (25 and 50 μ g/ml) prevented the A β (25-35) (10 μ M)-induced neuronal cell death, as assessed by a MTT assay. GR significantly inhibited AB (25-35)-induced elevation of the cytosolic Ca2+ concentration ([Ca2+]c). GR inhibited generation of reactive oxygen species. From these results, it is concluded that the protection against AB-induced neurotoxicity by GR may help to provide the pharmacological basis of its clinical usage in treatment of neurodegeneration such as AD.

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