E-E2-53

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

Ki Woong Kim[1], Hyun Soo Ju[1], KiHwan Bae[2] and Yeon Hee Seong[1*]
[1]College of Veterinary Medicine, Chungbuk National University, [2]College of Pharmacy, Chungnam National University

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 (Aβ) or Aβ 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 Aβ (25-35)-induced neurotoxicity was examined in primary cultured rat cortical neurons. To assess Aβ (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 Aβ (25-35)-induced increase of cytosolic Ca^{2+} concentration ($[Ca^{2+}]_c$), measured by fluorescent dye, fluo 4-AM. VA also blocked the Aβ (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 $[Ca^{2+}]_c$ and ROS and may have a promising role in prevention of AD.
*Vepharm@chungbuk.ac.kr

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

Soon Ock Cho[1], Kyung-Sik Song[2] and Yeon Hee Seong[1*]
[1]College of Veterinary Medicine, Chungbuk National University
[2]College of Agriculture and Life-Science, Kyungpook National University

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 (Aβ), which is involved in the pathology of alzheimer's disease (AD), was used in the present study. We demonstrated that GR (25 and 50 μg/ml) prevented the Aβ (25-35) (10 μM)-induced neuronal cell death, as assessed by a MTT assay. GR significantly inhibited Aβ (25-35)-induced elevation of the cytosolic Ca^{2+} concentration ($[Ca^{2+}]_c$). GR inhibited generation of reactive oxygen species. From these results, it is concluded that the protection against Aβ-induced neurotoxicity by GR may help to provide the pharmacological basis of its clinical usage in treatment of neurodegeneration such as AD.
*vepharm@chungbuk.ac.kr