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Protective effect of *Actinidia arguta* on Glutamate-induced neurotoxicity in cultured neurons

Jae hee Cho, Yeon hee Seong*

College of Veterinary Medicine, Chungbuk National University

Objectives

Glutamate known as the major excitatory neurotransmitter in the central nervous system plays an important role in fast excitatory synapses in the brain. The activation of glutamate receptors in a normal condition is involved in significant signaling function of brain. It acts for synaptic plasticity and neuronal gene expression, outgrowth, and survival through activation of its receptors. Four main subtypes of glutamate receptors are NMDA, AMPA, kainate and metabotropic receptors. Especially, NMDA receptor and its related channels are highly permeable to Ca^{2+}. Excessive extracelluar glutamate or overactivation of NMDA receptor can result in excitotoxicity and it can lead to a massive influx of Ca^{2+}, changes in the mitochondrial membrane potential, increase in the production of reactive oxygen species (ROS), and initiation of apoptosis, which eventually cause neuronal death. Therefore, excessively released glutamate play a critical role to cause neurotoxicity in neurodegenerative disease such as stroke, Alzheimer's disease and Parkinson's disease. *Actinidia arguta*, generally called hardy kiwifruit, has been reported to possess anti-inflammatory, anti-allergic, and antioxidative properties. The present study examined whether ethanol extract of leaf and stem of *A. arguta* can protect primary culture of rat cortical neurons against glutamate-induced neurotoxicity.

Materials and Methods

Primary cortical neuronal cultures were prepared using Sprague-Dawley (SD) rat fetuses on embryonic days 15 to 16. Experiments of glutamate-induced neurotoxicity were performed on neurons after 5 days in culture. Cultured neurons were treated with 500 μM glutamate for 12 h. Viability of cultured cells was measured by 3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyl-tetrazolium bromide (MTT) assay and Hoechst 33342 staining. The expression level of apoptosis-associated protein was measured by Western blot analysis.

주저자 연락처 (Corresponding author) : 성연희 E-mail : vepharm@cbnu.ac.kr Tel : 043-261-2968

Results

A. arguta, over a concentration range of 1-50 μg/ml, inhibited glutamate-induced neuronal cell death, as assessed by MTT assay (Fig. 1A) and the number of neuronal apoptotic death, evidenced by Hoechst 33342 staining (Fig. 1B). *A. arguta* inhibited the expression level of pro-apoptotic protein, which was increased by treatment with glutamate. These results suggest that *A. arguta* showed the neuroprotective effect on glutamate-induced neuronal toxicity. In conclusion, the present study suggest that *A. arguta* may have a therapeutic role in treatment of neurodegenerative disease such as stroke.

(A) (B)

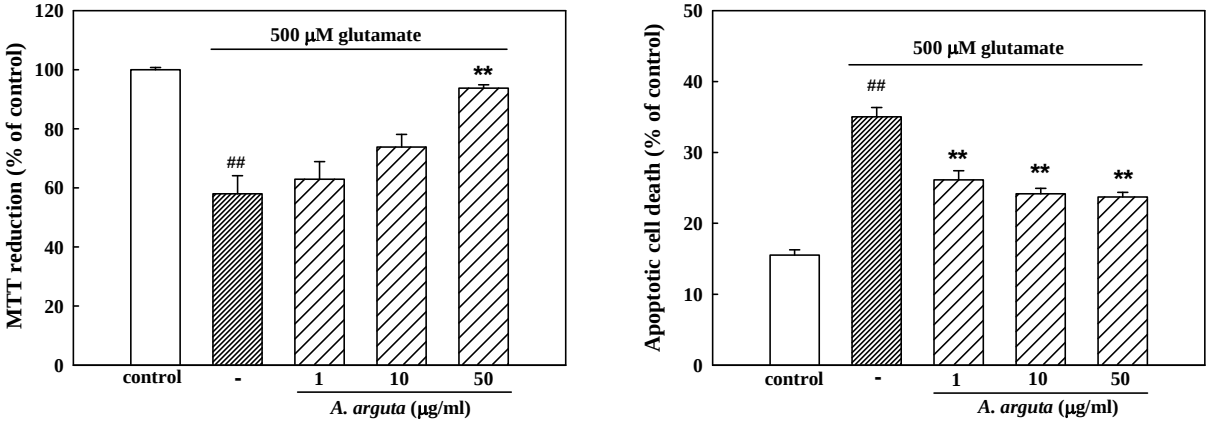

Fig. 1. Inhibitory effect of *A. arguta* on glutamate-induced neuronal apoptotic death in cultured cortical neurons.