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Curcuma Longae Radix, *Phellinus Linteus* 및 *Scutellariae* Radix 혼합추출물의 산화적 신경세포손상에 대한 보호효과

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Protective effect of Ethanol Extract of a Mixture of *Curcuma Longae* Radix, *Phellinus Linteus*, and *Scutellariae* Radix on Oxidative Neuronal Damage

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Objectives

Ischemic stroke is induced by a transient or permanent reduction of cerebral blood flow resulting from the occlusion of a cerebral artery. Many researches suggested that ischemic brain damage causes elevation of the intracellular Ca^{2+} concentration ($[Ca^{2+}]_i$), NMDA receptor modulation induced by glutamate release and reactive oxygen species (ROS) generation, resulting in apoptotic neuronal cell death. *Curcuma longae* radix from the root of *Curcuma longa* has antioxidant and anti-inflammatory effects. *Phellinus linteu*s has been used for anti-cancer, anti-diabetes and antioxidant activities. *Scutellariae* radix from *Scutellaria baicalensis* Gergi (Labiatae) has been demonstrated to possess antipyretic, antibacterial and anti-inflammatory properties. We previously reported that an ethanol extract of a mixture of *Curcuma longae* radix, *Phellinus linteus*, and *Scutellariae* radix, which was named as HS0608, inhibited Aβ (25-35)-induced neuronal cell damage and memory impairment due to antioxidant activity. The present study investigated the neuroprotective effect of HS0608 on hydrogen peroxide (H_2O_2)-induced neuronal damage in cultured rat cortical neurons and ischemia-induced brain damage following middle cerebral artery occlusion (MCAO)/reperfusion in rats.

Materials and Methods

Primary cortical neuron cultures were prepared using embryonic day 15 to 16 Sprague-Dawley (SD) rat fetuses. Experiments of H_2O_2-induced neurotoxicity were performed on neurons after 5-6 days in culture. Cultured neurons were treated with 100 μM H_2O_2 for 15 min. Viability of cultured cells was measured by 3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyl-tetrazolium bromide (MTT) assay and Hoechst 33342 staining. H_2O_2-induced elevation of $[Ca^{2+}]_i$ and ROS were measured by fluorescence dyes using laser scanning confocal microscopy. Cerebral ischemia was induced by 2-h MCAO and 24-h reperfusion in SD rats. After 24-h reperfusion, rats were sacrificed and brains were dissected out for 2,3,5-triphenyltetrazolium chloride (TTC) staining.

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Results

HS0608, over a concentration range of 10-100 μg/ml, inhibited H_2O_2-induced neuronal cell death, as assessed by MTT assay (Fig. 1A) and the number of neuronal apoptotic death, evidenced by Hoechst 33342 staining (Fig. 2B). H_2O_2-induced elevation of $[Ca^{2+}]_i$ and generation of ROS were also inhibited by HS0608. In vivo, HS0608 (200mg/kg) inhibited the formation of infarction and edema in ischemic brains induced by MCAO/reperfusion. These results suggest that HS0608 showed the neuroprotective effect on H_2O_2- and ischemia-induced neuronal damage via antioxidative mechanism. In conclusion, the present study provides further evidence that HS0608 can be a useful promising agent for the treatment of neurodegeneration in stroke.

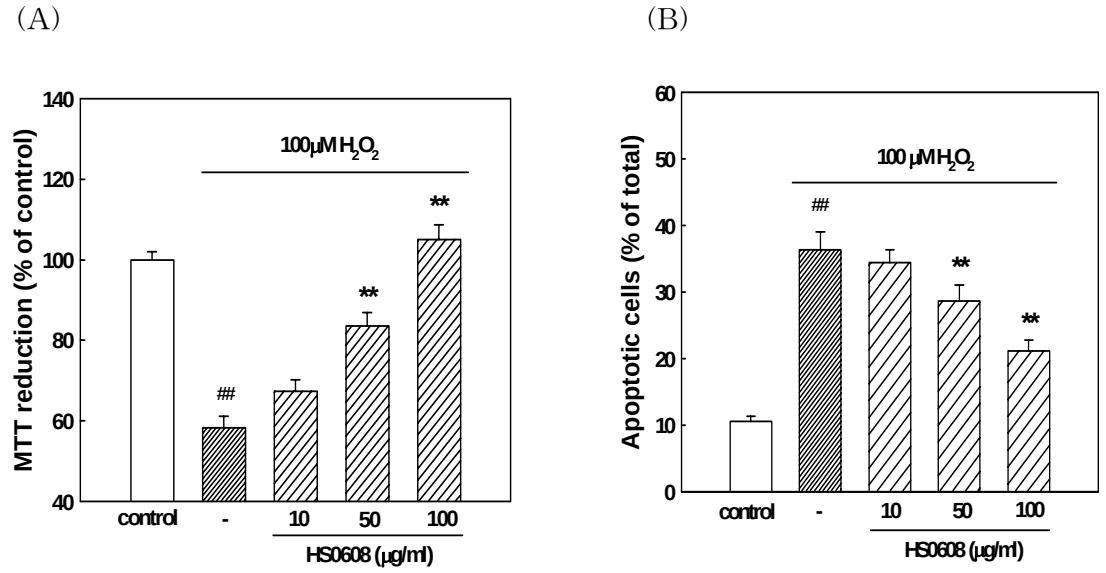

Fig. 1. Inhibitory effect of HS0608 on H_2O_2-induced neuronal death in cultured cerebral cortical neurons.