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Inhibitory effect of rice bran oil on glutamate- and ischemia- induced neurotoxicity : *in vitro* and *in vivo*

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글루타메이트와 허혈성 신경독성에 대한 미강유의 억제효과 : *in vitro* and *in vivo*

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Objectives

Stroke is the leading cause of morbidity and mortality worldwide. The hallmark of stroke is a sudden loss of brain function caused by a blockage or rupture of a blood vessel to the brain. Many pathophysiology studies have shown that hypoxic or ischemic injury of brain causes an excessive release of glutamate followed by overstimulation of glutamate receptors, particularly the N-methyl-D-aspartate (NMDA) receptor subtype. This type of receptor is largely permeable to calcium leading to a massive influx of this ion into the cells, resulting in neuronal death. Rice bran, the major byproduct of the rice milling industry, is the source of a high quality vegetable oil, rice bran oil (RBO). RBO has attracted much medicinal attention due to its strong hypocholesterolemic properties known to be attributable to its balanced fatty acid composition and high levels of antioxidant phytochemicals such as oryzanols, tocopherols and tocotrienols. RBO contains phytocerimides, which was demonstrated to have also anti-ischemic effect in our previous experiment. Therefore, in the present study, we investigated the inhibitory effect of RBO on glutamate- and ischemia -induced neurotoxicity using cultured neurons and middle cerebral artery occlusion (MCAo)/reperfusion rats, respectively.

Materials and Methods

Materials

Rice bran oil (RBO), glutamate, SD rats

Methods

Neuronal cells, cultured from 16-day-old fetuses of SD rats, were treated with glutam ate (8 h). Neuronal viability was measured by 3-[4,5-dimethylthiazole-2-yl]-2,5-diphe nyl-tetrazolium bromide (MTT) assay. Cerebral ischemic injury was induced by 2-h MCAo and 24-h reperfusion in SD rats.

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Results

RBO (0.01, 0.1 μl/ml) inhibited glutamate-induced neuronal cell death. RBO (5, 10 ml/kg) prevented cerebral ischemic injury induced by 2-h MCAo and 24-h reperfusion. Ischemic rats showed neurological signs, such as circling movement and decreased grip of contralateral forelimb and RBO (10 ml/kg) significantly prevented such behavioral deficits. Rats received RBO (5, 10 ml/kg) showed significantly improved behavior in rotarod test performed 24 h after the reperfusion, compared with rats received corn oil (5 ml/kg) as control. The present study provides an evidence that RBO might be available to protect neurodegeneration in stroke. Furthermore, the protective effect of RBO on ischemia-induced neurotoxicity might be attributable to phytoceramide, as an active principle.

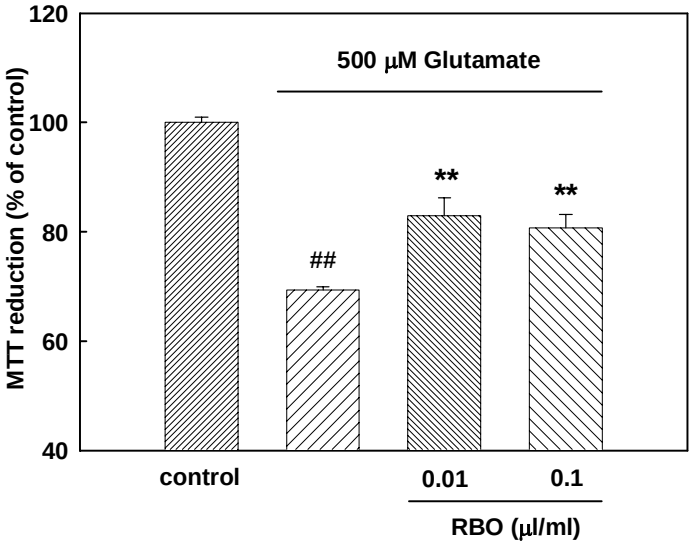

Fig 1. Inhibitory effect of RBO on glutamate-induced neuronal cell death in cultured cortical neurons.