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Catechin regulates intracellular calcium concentration and parvalbumin expression in ischemic brain injury

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Calcium is an essential factor that involved in modulation of cellular functions, such as cell differentiation, survival, and apoptosis. Parvalbumin is a calcium buffering protein that modulates intracellular calcium concentration. Catechin has an excellent antioxidant property and exerts a neuroprotective effect. This study investigated whether catechin can regulate parvalbumin expression and intracellular calcium concentration in middle cerebral artery occlusion (MCAO)-induced cell damage and glutamate toxicity-induced neuronal cell death. Male Sprague-Dawley rats were treated with vehicle or catechin (50 mg/kg) immediately before MCAO and cerebral cortical tissues were collected 24 h after MCAO. Catechin alleviated MCAO-induced infarction and neuronal movement deficit. MCAO induced a decrease of parvalbumin expression in cerebral cortex. However, catechin administration prevented MCAO-induced a decrease of parvalbumin. Glutamate excitotoxicity dramatically increased the intracellular calcium concentration in cultured hippocampal cells, whereas catechin attenuated an increase of intracellular calcium concentration. We observed a reduction of parvalbumin expression in glutamate-exposed cells. Catechin prevented glutamate-induced this decrease. These findings suggest that catechin exerts a neuroprotective effect through regulation of intracellular calcium concentration and parvalbumin expression in ischemic brain injury.

Biography

Phil-Ok Koh has completed her PhD at Gyeongsang National University and Postdoctoral studies from University of Maryland at Baltimore, USA. She is the Professor of College of Veterinary Medicine at Gyeongsang National University. She has published more than 180 papers in reputed journals and has been serving as an Editorial Board Member of reputed journals.

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