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Epigallocatechin gallate improves neuronal damage in animal model of ischemic stroke and glutamate-exposed neurons via modulation of hippocalcin expression

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Abstract

Epigallocatechin gallate (EGCG) is a polyphenolic component of green tea that has anti-oxidative and anti-inflammatory effects in neurons. Ischemic stroke is a major neurological disease that causes irreversible brain disorders. It increases the intracellular calcium concentration and induces apoptosis. The regulation of intracellular calcium concentration is important to maintain the function of the nervous system. Hippocalcin is a neuronal calcium sensor protein that controls intracellular calcium concentration. We investigated whether EGCG treatment regulates the expression of hippocalcin in stroke animal model and glutamate-induced neuronal damage. We performed middle cerebral artery occlusion (MCAO) to induce cerebral ischemia. EGCG (50 mg/kg) or phosphate buffered saline was injected into the abdominal cavity just before MCAO surgery. The neurobehavioral tests were performed 24 h after MCAO surgery and cerebral cortex tissue was collected. MCAO damage induced severe neurobehavioral disorders, increased infarct volume, and decreased the expression of hippocalcin in the cerebral cortex. However, EGCG treatment improved these deficits and alleviated the decrease in hippocalcin expression in cerebral cortex. In addition, EGCG dose-dependently alleviated neuronal cell death and intracellular calcium overload in glutamate-exposed neurons. Glutamate exposure reduced hippocalcin expression, decreased Bcl-2 expression, and increased Bax expression. However, EGCG treatment mitigated these changes caused by glutamate toxicity. EGCG also attenuated the increase in caspase-3 and cleaved caspase-3 expressions caused by glutamate exposure. The effect of EGCG was more pronounced in non-transfected cells than in hippocalcin siRNA-transfected cells. These findings demonstrate that EGCG protects neurons against glutamate toxicity through the regulation of Bcl-2 family proteins and caspase-3. It is known that hippocalcin exerts anti-apoptotic effect through the modulation of apoptotic pathway. Thus, we can suggest evidence that EGCG has a neuroprotective effect by regulating hippocalcin expression in ischemic brain damage and glutamate-exposed cells.

Figures

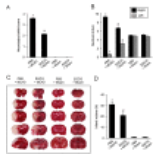

Fig 1. Neurobehavioral tests and infarct volume...

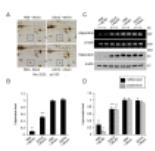

Fig 2. Expression change of hippocalcin in...

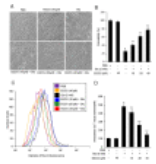

Fig 3. The measurement of cell viabilty...

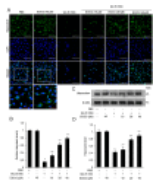

Fig 4. Expression change of hippocalcin in...

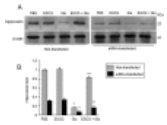

Fig 5. Western blot analysis of hippocalcin...

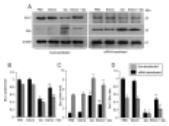

Fig 6. Western blot analysis of Bcl-2...

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