EFFECT OF ANTIOXIDANTS IN CULTURED CEREBRAL NEURONS OF FETAL BRAIN

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It is well known that oxygen radicals and lipid peroxidation in ischemic process induce the neuronal dysfunction and cell death in cerebral ischemia-anoxia. To understand the pathogenetic role of oxygen radicals in cerebral ischemia, cerebral neurons from human fetal brain(12-20 weeks' gestation) were treated with oxygen radicals, and the cell injury and cell death were evaluated. Oxygen radicals were produced enzymatically by xanthin oxidase (10-20 mU/ml) and hypoxanthine(0.1 mM). Cultures with 20mU/ml xanthine oxidase for 4 hrs resulted in cell death at more than 50%. The neuroprotective effects of various oxygen radical scavengers, iron chelators, and NMDA receptor antagonists also were investigated. In cerebral neuron cultures with xanthine oxidase, the following reagents blocked the neurotoxic effects of oxygen radicals: allopurinol(a xanthine oxidase inhibitor), glutathione, vitamin E(antioxidants), superoxide dismutase, catalase(oxygen radicals catalyzing enzymes), TPEN(a metal chelator), and NMDA receptor antagonists(MK-801, APV, CKA). Cultured human cerebral neurons are valuable experimental tool in which putative neuroprotective agents could be evaluated for neuronal damage caused by cerebral ischemia-reperfusion injury.