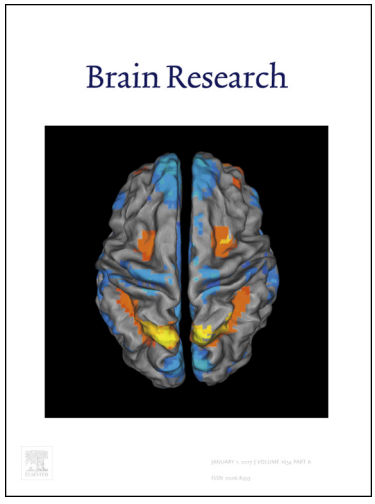

Research report

Time dependent neuroprotection of dexamethasone in experimental focal cerebral ischemia: the involvement of NF-κB pathways

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Time dependent neuroprotection of dexamethasone in experimental focal cerebral ischemia: the involvement of NF-κB pathways

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Abstract

We propose that the neuroprotective effect of glucocorticoid in ischemic damage may be time dependent. The present study was designed to test the proposal and its possible mechanism in cerebral ischemia/reperfusion (I/R) injury model. Reperfusion injury was induced after 120 minutes of middle cerebral artery occlusion (MCAO) in male Sprague-Dawley rats. At different time points after MCAO, rats were treated with high dose dexamethasone (10mg/kg), and neurological deficit and infarct sizes were measured 2 h, 24 h after MCAO. The expression of NF-κB target genes, including inducible nitric oxide synthase (iNOS), cyclooxygenase (COX-2), tumor necrosis factor-α (TNF-α) and interleukin-1β (IL-1β), were determined by western blot analysis and ELISA. Dexamethasone delivered 30 min (but not 60 min, 120 min) after MCAO markedly decreased the infarct size, improved neurological deficits in I/R injury model. Dexamethasone delivered 30 min (but not 60 min) after MCAO significantly inhibited NF-κB p65 expression and phosphorylation, compared with I/R group. The expression