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The neuroprotective effect of glutamate receptors group II agonists in an animal model of birth asphyxia is connected with inhibition of caspase independent apoptosis

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Statement of the Problem: Hypoxic-ischemic encephalopathy is one of the leading causes of neonatal mortality and permanent neurological disability worldwide. It was shown recently that mGluR2/3 activation before or after ischemic insult results in neuroprotection, but the exact mechanism of this effect is not clear.

Aim: The aim of present study was to investigate whether glutamate receptors group II agonists (mGluR2/3) activation after hypoxia-ischemia reduces brain damage and inhibits apoptotic processes.

Methodology: We used an animal model of Hypoxia-Ischemia (H-I) on 7-day old rat pups. Animals underwent unilateral common carotid artery ligation combined with 75 min hypoxia at 7.4% oxygen. Control pups were sham-operated (anaesthetized and left common carotid artery dissected, but not ligated). Animals were injected intraperitoneally with mGluR2 (LY 379268) and mGluR3 (NAAG) agonists, 1 hour or 6 hours after H-I (5 mg/kg of body weight). We examined the weight deficit of the ischemic brain hemisphere and the expression of caspase independent apoptosis factors (AIF, HTR/OMI and endonuclease G). The expression of trophic factors GDNF, BDNF and TGF-beta was also measured.

Results: Our results show that application of each agonist decreased brain tissue weight loss in ischemic hemisphere independently on the time of application (from 40% in H-I to 15-20% in treated). Both agonists of mGluR2/3 applied 1 hour or 6 hours after H-I decreased expression of AIF, HTR/OMI and endonuclease G proteins compared to untreated H-I. The mGluR2/3 agonists application decreased expression of TGF-beta and increased BDNF and GDNF in the ischemic hemisphere compared to H-I.

Conclusion: This study demonstrated the neuroprotective effect of mGluR 2/3 agonists on neonatal hypoxic-ischemic brain injury. Presented data suggest that this effect is connected with decreasing apoptosis.

Biography

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