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Neuroprotective effects of metabotropic glutamate receptors group II (mGluR2/3) agonists in an animal model of birth asphyxia

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Hypoxic-ischemic encephalopathy is an abnormal neurobehavioral state which results fromimpaired cerebral blood flow at the time around birth. This condition may cause neonatal death or be manifested later as cerebral palsy or mental deficiency. Recent investigations have not provided us with promising neuroprotective compounds to reduce perinatal hypoxic-ischemic (HI) brain injury. 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. The aim of present study was to investigate whether mGluR2/3 activation after hypoxia-ischemia reduces brain damage and if the activation of antioxidant enzymes and decrease of oxidative stress participate in observed effects.We used an animal model of hipoxia-ischemia (H-I) on 7-day old rat pups. Animals were anesthetized and the left common carotid artery was isolated and double – ligated and then cut between the ligatures. After completion of the surgical procedure the pups were subjected to hypoxia(7.2% -7.4% oxygen in nitrogen for 75 min at 35 ˚C). Control pups were sham-operated (anaesthetized and left c.c.a. dissected, but not ligated). Animals were injected intra peritoneal with specific mGluR2 (LY 379268) and mGluR3 (NAAG) agonists 1 h or 6 h after H-I (5 mg/kg of body weight). Weight deficit of the ischemic brain hemisphere, radical oxygen species (ROS) content, activity of antioxidant enzymes (superoxide dismutase - SOD, Glutatione peroxidase - GPx, catalase - CAT) and level of reduced glutathione (GSH) were measured.

Our results show a neuroprotective effect of mGluR 2/3 agonists. Both agonists applied decreased brain tissue weight loss in ischemic hemisphere independently on the time of application (from 40% in H-I to 15 - 20% in treated). Our results show that both mGluR2/3 antagonists reduce ROS level in the injured hemisphere. H-I resulted inincreased activity of SOD in the injured hemisphere, which was reduced by mGluR2/3 agonists application 1h or 6 h after H-I. The activity of glutathione peroxidase (GPX) was also increased by H-I and its activity was decreased by both agonists. The decrease of GSH level observed in ischemic hemisphere was not observed after agonists application, the effect was better expressed when agonists were administered 1 h after H-I. Application of agonists resulted also in decrease in CATactivity.

Conclusions: The results show that activation of mGluR2 and mGluR3 in a short time after H-I insult triggered neuroprotective mechanisms, which probably partly engage defence against oxidative stress and ROS production. The effect is more distinct when agonists are applied in a short time after H-I

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