Abstract

High energy deamand and insulation via the blood-bain barrier are the main reasons for neuronal sensitivity to oxygen or energy deficiency. Even short or mild periods of hypoxia/ischemia (H/I) could fataly impact the CNS enviroment. The area on the edge of the tissue affected by H/I and adjacent unaffected tissue is called the penumbra. Here, we can observe additional H/I related processes – gliosis allied with sterile inflammation and censecutive apoptosis. Opioid receptors attenuate H/I impact on CNS in both acute and consecutive phases. In acute phases, opioid receptors regulate ion homeostasis and attenuate glutamate toxicity; in consecutive phases, lower gliosis manifestation. Both these actions have significant neuroprotective effects. Ability of opioid receptor to lower sterile inflammation in CNS could be used in a series of neurodegenerative diseases, eg. Alzheimer disease or amyotrophic lateral screlosis. Glial cells participate on ion homeostasis, glutamate uptake, and production of antiinflammatory substances; one can, therefore, assume that a significant part of neuroprotective effects of OR is related to glial cells. The opioid system and its signaling pathways has not been fully elucidated yet. I present global overview of this phenomenon and describe some recent findings regarding opioid receptors and their signaling pathways in this bachelor‘s thesis.

Keywords: Opioids, opioid receptors, neuroprotection, glial cells, astrocytes, microglia