Hypoxia of the brain as well as the subsequent reperfusion can seriously alter the tissue microenvironment and result of the functional and structural changes of nerve and glial cells. Results of experimental studies show that some ions (e.g. Mg2+) can interfere with the brain development. To answer the question whether magnesium can modulate changes of neuronal circuits induced by hypoxia and reperfusion effect of magnesium administration on the density of nitrergic neurons (NO synthesising neurons) in the rats exposed to repeated hypoxia during the postnatal ontogeny (12, 25, and 35-day-old) was studied. NO synthesising neurons were identified according to the presence of NADPH-diaphorase (NADPH-d), the enzyme co-localized with NO synthase.

Results have shown that the long-lasting intermittent hypobaric hypoxia brings about the increase of the density of NADPH-diaphorase positive neurons in all studied regions of the hippocampus in 12-day-old animals, in 25-day-old only in the hilus of the dentate gyrus, and in 35-day-old in CA1, CA3 hippocampal regions and in the ventral blade of the dentate gyrus. Contrary to that, decreased density of nitrergic neurons was found in groups of animals exposed to hypoxia till the age of 25 days in both blades of the dentate gyrus and in 35 days in the ventral blade and hilus of the dentate gyrus.

Magnesium pre-treatment of animals exposed to hypoxia brought about decrease of nitrergic neurons in all age groups and in all areas studied, except the CA1 region in 35-dayoldrats, where magnesium had no effect on the density of NADPH-d positive neurons. When control animals were treated with magnesium, decrease of the density of NADPH-d positive neurons in all hippocampal regions of 35-day-old animals was observed. Results in 25-day-old rats were similar, except in the hilus of the dentate gyrus, where magnesium had no effect. In 18-day-old control rats magnesium treatment resulted in higher density of NADPH-d positive neurons in all hippocampal regions.

Presented results allow following conclusions

1. Hypoxia can both increase and decrease the density of NADPH-d positive neurons in the hippocampus (working hypothesis I was not fully confirmed).
2. Magnesium treatment in control rats resulted in both the decrease and increase of the NADPH-d positive neurons density in the hippocampus (working hypothesis II was not fully confirmed).
3. Magnesium treatment in rats exposed to long-term repeated hypoxia brought about decrease of the density of NADPH-d positive neurons in all hippocampal regions (working hypothesis IIII was confirmed).

Our experiments show that the repeated hypoxic stimulus in the immature brain increases the density of nitrergic neurons in the majority of cases. Simultaneous magnesium administration can balance the effect of hypoxia and bring the density of nitrergic neurons to control levels. Results indicate the neuroprotective effect of magnesium.