DESCRIPTION FOR THE GENERAL PUBLIC (IN ENGLISH)

Hypoxia during delivery is a complication that can disrupt the infant brain development, leading to various types of neurological disorders throughout life. According to global statistics, four million newborns die each year, a quarter of these deaths is due to perinatal hypoxia. In previous studies many cases pointed out the correlation between body temperature and subsequent neonatal mortality and the incidence of complications. These studies have provided undeniable evidence that a lower temperature has outstanding protective role, significantly minimizing the severity of adverse reactions destroying the neurons. It is worth noting that, so far was carried out hundreds, if not thousands of studies at different levels of biological organization on the mechanism of damage induced by hypoxia. However, so far the results does not enable for an unambiguous resolve of the problem. That is why we decided to focus on research aimed at detailed knowledge of endogenous substances with extremely high neuroprotective potential, which may be targeted to develop modern therapeutic strategies. Neural tissue damage is accompanied by increased levels of neurotrophic factors, from which the most crucial role in neuronal plasticity is attributed to brain-derived neurotrophic factor (BDNF). According to our knowledge, to date there have been no reports on the relationship between body temperature during hypoxia and the level of BDNF. Therefore, the main objective of the project is to answer the question whether there is a relationship between body temperature during hypoxia and the level described neurotrophin.

For the study we chose the animal model of simulated preinatal asphyxia, in which two-day newborn rats are exposed to anoxia. This model has been commonly accepted for several years and used in the world's research institutions in the study of perinatal hypoxia. Neonatal rats should be regarded as an excellent model to study the pathophysiology of preterm infants, because level of brain development is comparable to that achieved of human infants born in the seventh month of pregnancy. In order to answer to the questions in this project we plan to demonstrate a correlation between body temperature during hypoxia and the level of BDNF in the hippocampus and cortex of the brain of newborn rat in early life, namely 2 hours after hypoxia, 3, 7 and 14 days later. Analysis of the level of the protein will carry out the techniques used in molecular biology for the detection of specific molecules by Western blotting, enzyme linked immunosorbent assay ELISA and immunofluorescence assay. These techniques also apply to check whether a change in the profile of described factor in early life affects the process of apoptosis (cell death) in the brain after perinatal hypoxia simulated under different conditions. In the central nervous system highly active BDNF are found in the hippocampus and cerebral cortex, and thus the areas involved in the regulation of processes related to memory or learning. After 6 week we plan check whether the changes in the two forms of factor BDNF due to perinatal hypoxia under different conditions thermal effect on spatial memory in 6-weeks old rats. For this purpose, we conduct a Morris water maze test, which is widely used to study the related spatial memory.

The results will complement the existing knowledge on the pathophysiology of perinatal hypoxia. It is extremely important, because due to the variety of factors that may lead to the onset of hypoxia, and the heterogeneity of brain tissue, the process has not yet been fully elucidated. Therefore, detailed knowledge of endogenous mechanisms of neuroprotection can contribute to mitigating the effects of hypoxic-ischemic damage on the immature brain. Although clinical trials are crucial for developing new therapeutic approaches, however, studies in animal models provide more detailed data on the course of pathological processes than is possible in the case of research involving human newborns.