# ABSTRACT

**Background:** The complement system is involved in neuroprotection and brain repair after brain damage. To understand the molecular mechanisms of these processes, we performed gene expression profiling using quantitative real-time polymerase chain reaction (qPCR), which is the most accurate modern strategy for gene expression analysis.

**Project:** Our project was directly aimed at expression profiling of selected genes potentially involved in loss and rescue of neural tissue during three weeks after hypoxic-ischemic brain injury, an experimental model of perinatal asphyxia. Recent experiments have shown that over-expression of C3a under the control of the GFAP promoter (C3a/GFAP) reduced hippocampal injury after left common carotid artery ligation in neonatal mice by 50%, compared to wild type mice. Here, we assessed how the local expression of C3a/GFAP transgene affects gene expression profiles. Gene expression was measured on samples from hippocampus ipsilateral and contralateral to the injury and ipsilateral part of cortex, taken at the time of injury, 6 and 24 hours; 3, 7 and 21 days after the injury.

**Results**: Our data showed that the regulation of gene expression after hypoxic-ischemic injury differs in timing and intensity and may also be region dependent. The analysed genes belong to families related to neuromediator release and deactivation (SYN II, SYN III, GS), proliferation (NES, SOX2, DMN), apoptosis (Bax, Bad, BclXL, 14-3-3 eta), neuronal maturation (ENO2, TubB3), microglia activation (Aif 1, CD68 antigen); to families of inflammatory cytokines (Il1β, Il6, TNFα), complement anaphylatoxin receptors (C3aR, C5aR, C5L2), growth factors (GAP43, NGF) and genes related to the transgene function (C3a/GFAP, GFAP, C3).

**Conclusion**: Our study showed selected processes of the brain response to the hypoxic-ischemic injury within three weeks after the insult, which might help to uncover potential therapeutic targets for reducing brain damage.

We showed slight differences in trends in expression of the complement receptors and inflammatory cytokines. However, our study did not find any significant differences in gene expression profiles of C3a/GFAP over-expressing mice and wild type mice and as such, we still do not understand the mechanisms involved in C3a neuroprotection.