

Maternal infection is one of the risks for the development of neurodevelopmental disorders. Although this is an essential epidemiological issue, precise mechanisms underlying alteration of the brain function remain unclear.

Lipocalin2 (Lcn2) is an innate immune response protein, also known as protein implied in the modulation of neuronal morphology. Lcn2 is upregulated in the brain during multiple pathological conditions including bacterial infection, inflammation or chronic stress. Moreover, research conducted in our laboratory showed that the level of Lcn2 increases in the fetal brain after maternal infection. In this project, we hypothesize that Lcn2 is involved in the regulation of neuronal circuitry development during brain infection.

To test this hypothesis, we will check whether infection mediated increase in the Lcn2 level is protective or deleterious in the context of pathology. We will analyze the inflammatory responses, development of neuronal circuitry and behavior using a mouse model of maternal infection and animals without Lcn2 expression. Project will contribute to the better understanding of processes occurring in fetal brain during inflammation in the mother organism.