ABSTRACT

About 14 million people worldwide suffer from **Alzheimer's Disease** (AD). This is a problem not only for patients, but also their families and society. The cause of AD is poorly understood. In the amyloid hypothesis, the fundamental cause of AD is amyloid beta (A_β) deposits mainly in the hippocampus. There is also other theory, which postulates excessive glutamate release in that illness. Drugs, which are used in AD, such as memantine, belong to antagonists of glutamategic system. A natural antagonist of glutamatergic NMDA receptor is zinc, which attenuates excessive glutamate release. Zinc acts in the central nervous system (CNS) via GPR39 receptor, which activation leads to increased expression of proteins, that are associated with neuroplasticity. In this project we ask about the role of the GPR39 in neuroprotection/neuroplasticity, which is required in prevention, as well as treatment of AD.

THE AIM OF THIS PROJECT is investigation of neuroprotective role of GPR39, as well as its role in Alzheimer's disease. In this project it is planned to investigate behavior and hippocampal protein expression associated with AD, of GPR39 knockout animals. It is also planned to investigate behavior linked to learn and memory processes after chronic drug administration, which acts via GPR39.

Alzheimer's disease is incurable but preventable. It is very important to find proper pathomechanism of that illness. Research on neurodegeneration or AD should be extensively directed on finding a cure or preventive drugs.
Studies proposed in this project should answer the question about the role of GPR39 in neuroprotection. Administration of drug acting via GPR39 will show whether GPR39 may be a target for pharmacotherapy of neurodegeneration and/or Alzheimer's disease.