

## **Abstract**

Depression is a complex systemic disorder with exhibiting biological, cognitive and psychopathological symptoms. There are changes in hormonal regulation, immune changes and disturbance in function of especially monoamine and indoleamine neurotransmitter circuits in the CNS. Pharmacologic intervention with antidepressants is treating depressive syndrome as well as homeostatic imbalance. These neurotransmitter systems use in the signal transduction from membrane into the cell receptors coupled with heterotrimeric G proteins (GTP binding proteins). This thesis studies changes in G protein subunit levels induced by stress and psychotropic drugs in the CNS and immune system of experimental mice and antidepressant induced changes *in vitro* in the C6 glioma cell line and *in vivo* in the rat tissue. Immobilization stress induces prominent changes in the G protein subunit levels in the spleen and CNS of experimental mice with analogical profile of response. Results show importance of stress dopaminergic component in the leukocyte function regulation and also demonstrate importance of dopaminergic regulation without stress exposition. Findings obtained in the glioma C6 cell line show antidepressant induced drug specific changes of the G subunit profiles and demonstrate thus mode of action independent on the monoamine reuptake blockade, which can participate in the modulation of the signal transduction cascades initiated by G proteins in the neuronal and glial cell populations