Abstract

Members of the large family of G proteins and their coupled receptors are involved in a variety of transduction processes the cell uses to respond to a received signal. Depending on their specific structure and function, they influence a wide range of effector molecules. A large body of research has shown that many neurodegenerative diseases have a negative impact on the signal pathways controlled by G proteins. Due to ageing population, neurodegenerative diseases are currently imposing a risk for growing numbers of people. The sequelae observed in the pathological development of such diseases include especially changes in membrane receptors representation or receptor uncoupling from G protein, which inhibits G subunits activation. The undesirable inhibition or over-stimulation of G proteins results in the increase or decrease in effector activity, which subsequently impacts the production of second messengers and the activity of subsequent members of the signal cascade. As a result, these alterations lead to an increase in intracellular concentration of Ca2+ ions, which then influence receptors responsible for excitotoxicity, and contribute to apoptosis and necrosis of neuronal population. The thesis summarizes the defects of signalling pathways controlled by trimeric G proteins in association with the most common neurodegenerative diseases.

Key words:
GPCRs, G proteins, signalization, neurodegeneration, Alzheimer´s disease, Parkinson´s disease, Huntington´s disease, amyotrophic lateral sclerosis