DESCRIPTION FOR THE GENERAL PUBLIC (IN ENGLISH)

Glioblastoma (GBM) is the most aggressive central nervous system tumor. Routine treatment, which consist of surgery and chemotherapy, results in patients' survival for not longer than 2 years and this parameter has not significantly improved for many years. Understanding the molecular mechanism underlying proliferation and migration of GBM cells is crucial for designing new, more effective therapies. Glutamine is a bioenergetic substrate essential for tumor development in different types of tissues, including brain, and glutaminase is the glutamine-metabolizing enzyme. Studies performed in Department of Neurotoxicology MMRC surprisingly revealed that insertion into GBM cells of one of the glutaminase isoforms, GAB, leads to suppression of proliferation and migration ability of GBM cells in culture. However, the exact mechanism by which GAB suppresses tumor growth still remains unknown. Preliminary studies indicate that GAB can modulate intracellular signaling pathway dependent on protein kinase B (AKT). Alterations of AKT signaling pathway seem to be one of the reasons, and/or consequence of progression of GBM, but details of the mechanism by which GAB modulates AKT signaling are unknown. The project will contribute to explore this mechanism.