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Multiform Glioblastoma (GBM) is an aggressive, malignant cancer which affects glial cells, most commonly in the brain. There are currently no curative treatments available for GBM. It is thought that GBM tumor initiating cells (TICs) strongly contribute to GBM's aggressive and malignant nature. Tumor initiating cells are known for their plasticity, resistance to treatment, self-renewing capabilities, and tumorigenic properties. Knowing more about the specific genes involved in the genetic pathways common to GBM could provide information beneficial to the eventual development of effective GBM therapies. Previous experiments targeting genes amplified in GBM have shown that the knock down of PIK3C2G results in decreased relative cell counts. Additionally, previous experiments have shown that suppression of PIK3C2G in TICs resulted in cell differentiation and cell death. Other previous experiments have focused on PIK3C2G's role in insulin signaling in hepatic cells. Little is currently known about PIK3C2G's role in the brain or elsewhere in the body outside of in the insulin signaling pathway in hepatic cells. In order to investigate the potential proto-oncogenic properties of PIK3C2G, we designed knock down and over expression experiments in a transformed cancer cell line and murine NIH 3T3 cell lines respectively. The designed experiments involve immunoblotting, immunoprecipitation, quantitative PCR, sub-cloning DNA, and shRNA-mediated gene suppression. Initially, we chose to use the gateway system for the overexpression experiment due to its flexibility. However, after concerns regarding potentially low levels of expression when using the gateway system's destination vectors, we settled on using the retroviral vector pBABE-puro instead. Following previous complications, the final stages of this experiment are still underway.