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Category: Windows-only software

The long-term goal of this project is to identify the molecular and cellular changes that occur during the development and progression of glioblastoma multiforme (GBM), a malignant brain tumor. In recent years, multiple lines of evidence have established that GBM is a cancer of neural stem cells, and the characterization of the molecular changes that regulate the transformation of normal stem cells into GBM cells could lead to the identification of new therapeutic targets for this disease. A better understanding of the molecular changes that regulate the transformation of stem cells into cancer stem cells could lead to the identification of new therapeutic targets for GBM. The transforming growth factor-beta (TGF- $\beta$ ) pathway is the best-characterized signaling pathway that regulates the transformation of neural stem cells into GBM cells. The role of TGF- $\beta$  in the pathogenesis of GBM is complex and is still not fully understood. TGF- $\beta$  is both a tumor suppressor and tumor promoter in different contexts. TGF- $\beta$  is known to activate signal transducer and activator of transcription 5 (Stat5) and suppress transcription factor 4 (TCF4) in the glial lineage. Stat5 and TCF4 are known to be involved in the regulation of cell proliferation and differentiation. Stat5, TCF4 and the TGF- $\beta$  signaling pathway are aberrantly activated in GBM cells and the effects of Stat5 and TCF4 are believed to be mediated by TGF- $\beta$  in GBM. The hypothesis of this project is that the abnormal activation of the TGF- $\beta$  signaling pathway by aberrant Stat5 and TCF4 is a molecular mechanism by which neural stem cells become transformed to GBM cells. In this project, we will test this hypothesis and further characterize the role of Stat5 and TCF4 in the pathogenesis of GBM. We will (1) determine the role of Stat5 and TCF4 in the pathogenesis of GBM; (2) establish the role of the TGF- $\beta$  signaling pathway in GBM in vivo; and (3) investigate the mechanisms by which Stat5 and TCF4 promote the transformation of neural stem cells to GBM cells. We will use glioblastoma cell lines, patient-derived tumor samples and the murine glioblastoma model to study the role of Stat5 and TCF4 in the pathogenesis of GBM. We will use genetic and pharmacological approaches and immunohistochemistry to

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