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CK2-Alpha and EGFRvIII are Co-Expressed in Primary Glioblastoma Tumors

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Abstract

Glioblastoma (GBM) is the most common and fatal primary brain tumor with an overall life expectancy of 14 months. Since no cure exists it is essential novel therapies are developed to treat this devastating disease. Previous research has shown that an oncogenic kinase, casein kinase 2 (CK2) may be a promising therapeutic target for GBMs. CK2 has enhanced expression or activity in a wide variety of cancers including GBM and it was demonstrated that inhibitors to CK2 regressed tumor growth in GBM xenograft mouse models. Through our own work we demonstrate that GBM patients with high expression of CK2 had a much worse prognosis than patients with low levels. Currently the mechanisms enabling enhanced expression or activity of CK2 is still unknown. Our studies demonstrate that a deletion mutant of the EGF receptor (EGFRvIII) is involved in CK2 dependent tumorigenesis in GBM cell lines. We have generated GBM cell lines (U87-MG, U138) that stably express EGFRvIII and have shown that these cells are more sensitive to depletion of CK2. Initial studies showed that siRNAs specific to a specific CK2 subunit (CK2alpha) decreased GBM cell growth ~2-fold in the control GBM cells. Interestingly, expression of EGFRvIII sensitized the GBM cells to growth arrest since cell growth was reduced 5-7 fold with reduced expression of CK2alpha. In addition, inhibition of CK2alpha activity using commercially available inhibitors (TBB, TBBz) also reduced GBM cell growth (1.5-2 fold), but we observed a more dramatic reduction (4-5 fold) in EGFRvIII overexpressing cells. We have also conducted preliminary studies demonstrating that CK2alpha kinase activity in vivo is enhanced with EGFRvIII expression. Together our study suggests that EGFRvIII may play an important role in GBM tumorigenesis by regulating CK2 activity and that a combination treatment targeting both EGFRvIII and CK2alpha might be more efficacious than each one individually.

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