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Estrogen Receptor Beta Signaling: A Novel Therapeutic Target for Gliomas

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Gliomas are the most common and devastating central nervous system neoplasms. A gender bias exists in their development: females are at lower risk than males, implicating estrogen-mediated protective effects. Estrogen functions are mediated by two ER subtypes: ERα, that functions as tumor promoter and ERβ that function as tumor suppressor. We examined the potential use of ERβ agonists as a novel therapeutic to curb the growth of gliomas. Western analysis of six glioma model cells showed detectable expression of ERβ with little or no ERa. Treatment of glioma cells with ERb agonists resulted in significant decrease in proliferation. IHC analysis of tumor tissues revealed that ERb expression is down regulated in high-grade gliomas. We found that ERb agonists promote both expression and tumor suppressive functions of ERb in glioma cells. Liquiritigenin, a plant-derived ERβ agonist significantly reduced in vivo tumor growth in a xenograft model. Compared to control mice, animals treated with liquiritigenin had greater than 50% reduction in tumor volume and size. IHC analysis of tumors revealed a significant increase in the nuclear ERβ expression with a concomitant decrease in cell proliferation in the liquiritigenin-treated group. Our results suggest that ERb signaling has a tumor suppressive function in gliomas. Since ERβ agonists are currently in clinical trials and are well tolerated with fewer side effects, identification of an ERb agonist as a therapeutic agent can be readily extended to clinical use with current chemotherapies, providing an additional tool for enhancing survival in glioma patients.

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