Evaluation of anti-angiogenic agent F16 for targeting glioblastoma xenograft tumors	
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Glioblastoma Multiforme (GBM) is one of the most aggressive and lethal types of all cancers, with an average 5-year survival rate of 5%. Since GBM tumors are highly vascularized tumors, and their growth is angiogenesis-dependent, antagonizing tumor angiogenesis by using angiogenesis inhibitors were considered as one of the promising approaches. In this context, intensive preclinical evaluation of a novel small molecule named F16 has exhibited potent anti-angiogenic and anti-tumor activities by selectively antagonizing Vascular Endothelial Growth Factor Receptor (VEGFR). Also, recent pharmacokinetic evaluation of F16 with tissue distribution analysis has shown that this molecule is transported across the blood-brain barrier (BBB) and accumulates in the brain regions with no signs of neurotoxicity. Therefore, further studies were conducted to determine the efficacy of F16 in delaying glioblastoma progression via inhibiting tumor angiogenesis. Our in vitro studies have clearly demonstrated the ability of F16 to inhibit migration and invasion of U87MG cells and also confirmed a potent cytotoxic effect against these cells in comparison to Temozolomide (TMZ). Our in vivo studies with the subcutaneously implanted (s.c.) xenograft tumor model and in vitro studies have clearly demonstrated the ability of F16 to delay tumor growth and inhibit migration and invasion.