Editorial

There has been a trend of use of conventional or non-conventional therapies against cancer such as targeting "king pin" tumor cells itself, targeting tumor vasculature lined by endothelial cells, targeting tumor associated macrophages and recently, targeting inhibitory signals on T cells through immunotherapies in tumor microenvironment (TME). However, most of the cases have witnessed therapy resistance owing a short-term or transient benefit. Despite of several failures in clinical trials targeting tumors and their microenvironments, our understanding is improving every day. It is evident that mutations in tumor cell compartment play a critical role in cancer development as well as therapy resistance. Here, we have discussed studies representing therapy resistance, through p53 as a model mutation and glioblastoma (GBM) as a model tumor.

Targeting tumor cells in glioblastoma

GBM, a grade IV glioma classified by World Health Organization, is considered highly malignant, vascular and invasive subtype [1]. Hypoxia and neovascularization are signature histopathologic features of GBM [2], which is most lethal during first year after initial diagnosis despite surgical resection and other standard therapies [1,3]. Temozolomide chemotherapy and radiotherapy against GBM tumor cells have led to a significant improvement in tumor growth and patient survival in newly diagnosed and recurrent GBM [4,5]. The survival advantage conferred

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18. Achyut BR, Bader DA, Robles AI, Wangsa D, Harris CC, et al. (2013)