



A service of the [U.S. National Library of Medicine](#)
and the [National Institutes of Health](#)

Select **19713741**

1: [Cell Cycle](#). 2009 Sep 15;8(18):2936-44. Epub 2009 Sep 13.



The ABCG2 resistance network of glioblastoma.

[Bleau AM](#), [Huse JT](#), [Holland EC](#).

Department of Cancer Biology and Genetics and Brain Tumor Center, Memorial Sloan-Kettering Cancer Center, New York, NY, USA.

Glioblastoma multiforme (GBM) is the most common and lethal primary brain tumor variant. It exhibits heterogeneity at both the morphologic and genetic levels, with a complex combination of somatic alterations rendering the tumor class difficult to adequately treat. The role of so-called "cancer stem-cells" (CSCs) in the resistance of high-grade gliomas like GBM to conventional treatment regimens has received much recent attention, especially with regard to the enhanced ability of stem-like cells to activate survival pathways, repair DNA damage, and expel cytotoxic drugs. Furthermore, the biology of GBM is actually even more convoluted, characterized by a constantly changing microenvironment that greatly influences tumor growth and response to therapy. Herein we review the most recently reported genetic events in human glioma patients and their influence on treatment response, particularly in relation to O6-methylguanine-DNA methyltransferase methylation status. We aim to present evidence for a role for cancer stem-like cells and their unique microenvironment in therapy resistance and forward our views on glioma initiation and origin. Finally, given the recent interest in "side population" (SP) cells as a model of CSCs in gliomagenesis, we further describe the function of ABCG2 transporters, the mediators of the SP phenotype, at both the blood-brain barrier and in stem-like tumor cells.

PMID: 19713741 [PubMed - in process]
