Histone deacetylases (HDACs) can regulate the progression of various cancers, while their roles in glioblastoma multiforme (GBM) are not well known.

A study investigated the expression of class I HDACs (HDAC1, 2, 3, 8) in GBM U87, A172, U251, and LN229 cells and compared their levels with that in primary normal human astrocytes (NHA) cells. It showed that HDAC2 expression is significantly up-regulated in GBM cells. Silencing of HDAC2 via its specific siRNAs can suppress the in vitro proliferation, migration, and invasion of GBM U87 and A172 cells. Furthermore, silencing of HDAC2 can increase the sensitivity of GBM cells to temozolomide (TMZ), a standard-of-care during clinical GBM treatment. This might be due to that si-HDAC can significantly down-regulate the mRNA and protein expression of MRP1, while has no effect on ABCB1 and ABCG2. Schisandrin B (Sch B), a specific inhibitor of MRP1, can further increase the TMZ sensitivity in HDAC2-knocked down GBM cells. Collectively, data revealed that targeted HDAC2 can suppress the malignancy of GBM cells and increase their sensitivity of TMZ via down-regulation of MRP1. It suggested that HDAC2 might be a potential target for GBM therapy and improvement in TMZ therapy efficiency [1].

1)
Zhang Z, Wang Y, Chen J, Tan Q, Xie C, Li C, Zhan W, Wang M. Silencing of histone deacetylase 2 suppresses malignancy for proliferation, migration, and invasion of glioblastoma cells and enhances temozolomide sensitivity. Cancer Chemother Pharmacol. 2016 Nov 10. [Epub ahead of print] PubMed PMID: 27832326.