

5-Aza-dC ([Decitabine](#)) treatment combines with anti-PD-1 immunotherapy to efficiently suppress the progression of GL261 gliomas. Data support a mechanism of [epigenetic modifications of AP-2 \$\alpha\$](#) that contributes to [tumor immune evasion](#), and reactivation of AP-2 α synergizes with anti-PD-1 antibodies to increase [antitumor](#) efficacy, which may be a broadly applicable strategy in [solid tumors](#)¹⁾

Guo et al. showed that [Transforming growth factor Beta](#) induced the downregulation of [MST1](#) expression in U87 and U251 glioma cells. Treatment of glioma cells with the DNA methylation inhibitor [Decitabine](#) (5-aza-2'-deoxycytidine 5-AzadC) prevented the loss of MST1 expression. Addition of 5-AzadC also reduced the TGF- β -stimulated proliferation, migration and invasiveness of glioma cells. Furthermore, Knockdown of [DNMT1](#) upregulated MST1 expression in gliomas cells. In addition, the inhibition of DNMT1 blocked TGF- β -induced proliferation, migration and invasiveness in glioma cells. These results suggest that TGF- β promotes glioma malignancy through DNMT1-mediated loss of MST1 expression²⁾.

¹⁾

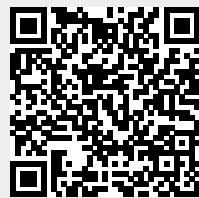
Long S, Huang G, Ouyang M, Xiao K, Zhou H, Hou A, Li Z, Zhong Z, Zhong D, Wang Q, Xiang S, Ding X. Epigenetically modified AP-2 α by DNA methyltransferase facilitates glioma immune evasion by upregulating PD-L1 expression. *Cell Death Dis.* 2023 Jun 17;14(6):365. doi: 10.1038/s41419-023-05878-x. PMID: 37330579.

²⁾

Guo Z, Li G, Bian E, Ma CC, Wan J, Zhao B. TGF- β -mediated repression of MST1 by DNMT1 promotes glioma malignancy. *Biomed Pharmacother.* 2017 Aug 9;94:774-780. doi: 10.1016/j.biopha.2017.07.081. [Epub ahead of print] PubMed PMID: 28802229.

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