Histone deacetylase 1

Histone deacetylase 1 (HDAC1) is thought to play pivotal roles in neurogenesis and neurodegeneration.

The role of HDAC1 in neuronal growth and structural plasticity in the developing brain in vivo remains unclear.

Histone deacetylase 1 (HDAC1) plays a crucial role in cancer progression and development. This enzyme has been confirmed to be a key regulator of tumor biology functions, such as tumor cell proliferation, migration and invasion. However, HDAC1 expression in glioma remains controversial, and its specific function and molecular mechanism in glioblastoma is poorly understood.

Findings demonstrated that protein and mRNA levels of HDAC1 were increased in glioma cell lines and glioma tissues compared to normal glial cell lines and non-neoplastic brain tissues, respectively. Furthermore, HDAC1 knockdown cells displayed decreased proliferation and invasion capabilities, whereas HDAC1 overexpressing glioblastoma cells displayed more proliferation and invasion capabilities in vitro. These novel outcomes suggested that knockdown of HDAC1 possibly suppressed the expression of phosphorylated AKT (p-AKT) and phosphorylated ERK (p-ERK) proteins, while overexpression of HDAC1 significantly increased p-AKT and p-ERK protein in glioblastoma cells. In addition, knockdown of HDAC1 repressed subcutaneous tumor growth in vivo, and led to down-regulation of p-AKT and p-ERK protein in U87 MG xenograft tumors. For the first time, we have demonstrated that HDAC1 promotes proliferation and invasion in glioblastoma cells by activating PI3K/AKT and MEK/ERK signaling pathways in vitro and in vivo. These results suggest that HDAC1 may be a novel biomarker and potential therapeutic target in glioblastoma [1)].

Ruan et al showed that in the optic tectum of Xenopus laevis, HDAC1 knockdown dramatically decreased the frequency of AMPAR-mediated synaptic currents and increased the frequency of GABAAR-mediated currents, whereas HDAC1 overexpression significantly decreased the frequency of GABAAR-mediated synaptic currents. Both HDAC1 knockdown and overexpression adversely affected dendritic arbor growth and visual experience-dependent structural plasticity. Furthermore, HDAC1 knockdown decreased BDNF expression via a mechanism that involves acetylation of specific histone H4 residues at lysine K5. In particular, the deficits in dendritic growth and visually guided avoidance behavior in HDAC1-knockdown tadpoles could be rescued by acute tectal infusion of BDNF. These results establish a relationship between HDAC1 expression, histone H4 modification and BDNF signaling in the visual-experience dependent regulation of dendritic growth, structural plasticity and function in intact animals in vivo [2)].

1)
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2)
Ruan H, Gao J, Qi X, Tao Y, Guo X, Guo Z, Zheng L, Song Y, Liao Y, Shen W. Visual Experience Dependent Regulation of Neuronal Structure and Function by Histone Deacetylase 1 in Developing

Xenopus Tectum in vivo. Dev Neurobiol. 2016 Dec 29. doi: 10.1002/dneu.22480. [Epub ahead of print] PubMed PMID: 28033671.