SANTA CRUZ BIOTECHNOLOGY, INC.

HDAC9 (B-1): sc-398003

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

In the intact cell, DNA closely associates with histones and other nuclear proteins to form chromatin. The remodeling of chromatin is a critical component of transcriptional regulation and the acetylation of nucleosomal histones is a major source of this remodeling. Acetylation of lysine residues in the amino terminal tail domain of histone results in an allosteric change in the nucleosomal conformation and an increased accessibility to transcription factors by DNA. Several mammalian proteins function as nuclear histone acetylases, including GCN5, PCAF (p300/CBP-associated factor), p300/CBP, HAT1 and the TFIID subunit TAF II p250. Conversely, the deacetylation of histones is associated with transcriptional silencing. The histone deacetylases (HDAC) include HDAC1-9. HDAC9 and HDAC9a are two alternatively spliced isoforms of HDAC9. HDAC9a is 132 amino acids shorter than HDAC9, but both isoforms contain the HDAC catalytic domain, remain capable of deacetylase activity and repress myoctye enhancer-binding factor 2-mediated transcription. HDAC9 and HDAC9a are expressed in brain, skeletal muscle, kidney, placenta and pancreas.

CHROMOSOMAL LOCATION

Genetic locus: HDAC9 (human) mapping to 7p21.1; Hdac9 (mouse) mapping to 12 A3.

SOURCE

HDAC9 (B-1) is a mouse monoclonal antibody specific for an epitope mapping between amino acids 12-39 at the N-terminus of HDAC9 of human origin.

PRODUCT

Each vial contains 200 µg IgM kappa light chain in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

Blocking peptide available for competition studies, sc-398003 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% stabilizer protein).

APPLICATIONS

HDAC9 (B-1) is recommended for detection of HDAC9 isoforms 1-4 of mouse, rat and human origin by Western Blotting (starting dilution 1:100, dilution range 1:100-1:1000), immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)], immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

Suitable for use as control antibody for HDAC9 siRNA (h): sc-35550, HDAC9 siRNA (m): sc-35551, HDAC9 shRNA Plasmid (h): sc-35550-SH, HDAC9 shRNA Plasmid (m): sc-35551-SH, HDAC9 shRNA (h) Lentiviral Particles: sc-35550-V and HDAC9 shRNA (m) Lentiviral Particles: sc-35551-V.

Molecular Weight of HDAC9: 160 kDa.

Positive Controls: Ramos cell lysate: sc-2216 or NAMALWA cell lysate: sc-2234.

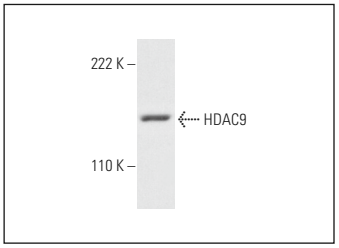
RESEARCH USE

For research use only, not for use in diagnostic procedures.

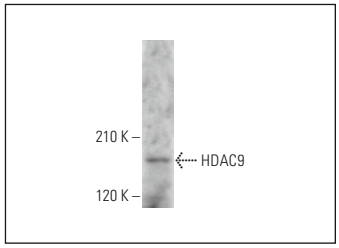
STORAGE

Store at 4° C, **DO NOT FREEZE**. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.

DATA



HDAC9 (B-1): sc-398003. Western blot analysis of HDAC9 expression in Ramos whole cell lysate.



HDAC9 (B-1): sc-398003. Western blot analysis of HDAC9 expression in NAMALWA whole cell lysate.

SELECT PRODUCT CITATIONS

1. Rastogi, B., et al. 2016. Overexpression of HDAC9 promotes oral squamous cell carcinoma growth, regulates cell cycle progression, and inhibits apoptosis. Mol. Cell. Biochem. 415: 183-196.
2. Hanigan, T.W., et al. 2017. Divergent JNK phosphorylation of HDAC3 in triple-negative breast cancer cells determines HDAC inhibitor binding and selectivity. Cell Chem. Biol. 24: 1356-1367.e8.
3. Lu, S., et al. 2018. HDAC9 promotes brain ischemic injury by provoking IκBα/NFκB and MAPKs signaling pathways. Biochem. Biophys. Res. Commun. 503: 1322-1329.
4. Li, C., et al. 2019. Nuclear receptor corepressor 1 represses cardiac hypertrophy. EMBO Mol. Med. 11: e9127.
5. Kang, D.W., et al. 2021. Phospholipase D1 is upregulated by vorinostat and confers resistance to vorinostat in glioblastoma. J. Cell. Physiol. 236: 549-560.
6. He, X., et al. 2022. Selective inhibition of histone deacetylase class IIa with MC1568 ameliorates podocyte injury. Front. Med. 9: 848938.
7. Yang, J.M., et al. 2022. NAC1 modulates autoimmunity by suppressing regulatory T cell-mediated tolerance. Sci. Adv. 8: eabo0183.
8. Dent, P., et al. 2022. AR12 increases BAG3 expression which is essential for Tau and APP degradation via LC3-associated phagocytosis and macroautophagy. Aging 14: 8221-8242.
9. Lei, M., et al. 2023. Molecular mechanism and therapeutic potential of HDAC9 in intervertebral disc degeneration. Cell. Mol. Biol. Lett. 28: 104.
10. Yeon, M., et al. 2024. HDAC9 and miR-512 regulate CAGE-promoted anti-cancer drug resistance and cellular proliferation. Curr. Issues Mol. Biol. 46: 5178-5193.

PROTOCOLS

See our web site at www.scbt.com for detailed protocols and support products.