SANTA CRUZ BIOTECHNOLOGY, INC.

HDAC5 (G-18): sc-5250

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 believed to be a critical component of transcriptional regulation and a major source of this remodeling is brought about by the acetylation of nucleosomal histones. 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. Conversely, the deace-tylation of histones is associated with transcriptional silencing. Several mammalian proteins have been identified as nuclear histone acetylases, including GCN5, PCAF (for p300/CBP-associated factor), p300/CBP and the TFIID subunit TAF II p250. Mammalian HDAC1 (also designated HD1) and HDAC2 (also designated mammalian RPD3), both of which are related to the yeast transcriptional regulator Rpd3p, have been identified as histone deacetylases.

REFERENCES

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2. Braunstein, M., et al. 1993. Transcriptional silencing in yeast is associated with reduced nucleosome acetylation. Genes Dev. 7: 592-604.
3. Bauer, W.R., et al. 1994. Nucleosome structural changes due to acetylation. J. Mol. Biol. 236: 685-690.
4. Brownell, J.E., et al. 1996. Tetrahymena histone acetyltransferase A: a homolog to yeast GCN5p linking histone acetylation to gene activation. Cell 84: 843-851.
5. Yang, X.J., et al. 1996. A p300/CBP-associated factor that competes with the adenoviral oncoprotein E1A. Nature 382: 319-324.

CHROMOSOMAL LOCATION

Genetic locus: HDAC5 (human) mapping to 17q21.31; Hdac5 (mouse) mapping to 11 D.

SOURCE

HDAC5 (G-18) is an affinity purified goat polyclonal antibody raised against a peptide mapping at the N-terminus of HDAC5 of human origin.

PRODUCT

Each vial contains 200 µg IgG in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

Blocking peptide available for competition studies, sc-5250 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

STORAGE

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

RESEARCH USE

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

APPLICATIONS

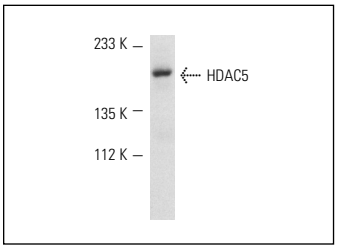
HDAC5 (G-18) is recommended for detection of HDAC5 of mouse, rat and human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)], immunofluorescence (starting dilution 1:50, dilution range 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 HDAC5 siRNA (h): sc-35542, HDAC5 siRNA (m): sc-35543, HDAC5 shRNA Plasmid (h): sc-35542-SH, HDAC5 shRNA Plasmid (m): sc-35543-SH, HDAC5 shRNA (h) Lentiviral Particles: sc-35542-V and HDAC5 shRNA (m) Lentiviral Particles: sc-35543-V.

Molecular Weight of HDAC5: 140-150 kDa.

Positive Controls: KNRK nuclear extract: sc-2141, IMR-32 cell lysate: sc-2409 or Jurkat nuclear extract: sc-2132.

DATA



HDAC5 (G-18): sc-5250. Western blot analysis of HDAC5 expression in KNRK nuclear extract.

SELECT PRODUCT CITATIONS

1. Ajamian, F., et al. 2004. Selective regulation of class I and class II histone deacetylases expression by inhibitors of histone deacetylases in cultured mouse neural cells. Neurosci. Lett. 365: 64-68.
2. Sucharov, C.C., et al. 2008. YY1 protects cardiac myocytes from pathologic hypertrophy by interacting with HDAC5. Mol. Biol. Cell 19: 4141-4153.
3. Fioravante, D., et al. 2008. The ubiquitin-proteasome system is necessary for long-term synaptic depression in Aplysia. J. Neurosci. 28: 10245-10256.
4. Seo, H.W., et al. 2009. Transcriptional activation of hypoxia-inducible factor-1α by HDAC4 and HDAC5 involves differential recruitment of p300 and FIH-1. FEBS Lett. 583: 55-60.
5. Chandrasekaran, S., et al. 2009. Histone deacetylases facilitate sodium/calcium exchanger up-regulation in adult cardiomyocytes. FASEB J. 23: 3851-3864.
6. Govindan, M.V. 2010. Recruitment of cAMP-response element-binding protein and histone deacetylase has opposite effects on glucocorticoid receptor gene transcription. J. Biol. Chem. 285: 4489-4510.
7. Suzuki, A., et al. 2010. Down-regulation of PROS1 gene expression by 17β-estradiol via estrogen receptor α (ERα)-Sp1 interaction recruiting receptor-interacting protein 140 and the corepressor-HDAC3 complex. J. Biol. Chem. 285: 13444-13453.