

Product Information

Anti-Histone Deacetylase 5 (HDAC5) (PI-16)

Developed in Rabbit, Affinity Isolated Antibody

Product Number **H 9663**

Product Description

Anti-Histone Deacetylase 5 (HDAC5) (PI-16) is produced in rabbit using as immunogen a synthetic peptide corresponding to amino acid residues 4-19 of HDAC5 with C-terminal added lysine, conjugated to KLH. The corresponding sequence is similar in mouse and human. The antibody is affinity-purified using the immunizing peptide immobilized on agarose.

Anti-Histone Deacetylase 5 (HDAC5) (PI-16) recognizes mouse and human HDAC5. Applications include immunoblotting (~ 124 kDa), immunocytochemistry, and immunoprecipitation. Detection of the HDAC5 band by immunoblotting is specifically inhibited with the immunizing peptide. Additional weak bands may be detected by immunoblotting in some extract preparations.

Regulation of gene expression is mediated by several mechanisms; among them are DNA methylation, ATP-dependent chromatin remodeling, and posttranslational modifications of histones. These modifications include the dynamic acetylation and deacetylation of ϵ -amino groups of lysine residues present in the tail of core histones.¹ The enzymes responsible for this reversible acetylation/deacetylation process are histone acetyltransferases (HATs) and histone deacetylases (HDACs), respectively.² While HATs act as transcriptional coactivators, HDACs are part of transcriptional corepressor complexes.³ Mammalian HDACs can be divided into three classes according to sequence homology.⁴ Class I consists of the yeast Rpd3-like proteins HDAC1, HDAC2, HDAC3, and HDAC8. Class II consists of the yeast Hda1-like proteins HDAC4,⁵ HDAC5, HDAC6, HDAC7, HDAC9, and HDAC10.⁵

Class III comprises the yeast Sir2-like proteins. Whereas class I HDACs are ubiquitously expressed, most class II HDACs are tissue-specific.² The deacetylase activity of class II HDACs is regulated by sub-cellular localization.⁴ The localization of HDAC5 is both nuclear and cytoplasmic. Shuttling to the cytoplasm occurs during myocyte differentiation; the nuclear export being stimulated by CaMK phosphorylation at Ser²⁵⁹ and Ser⁴⁹⁸. HDAC5 activity is important for the differentiation of muscle cells by binding, through its N-terminal domain, to the MEF2 protein, thus repressing expression of MEF2 down stream genes.⁶ Over expression of HDAC5 in different cancer cells suppresses their growth by induction of apoptosis in a p53-independent manner.⁷

Reagent

The antibody is supplied as a solution in 0.01 M phosphate buffered saline, pH 7.4, containing 15 mM sodium azide.

Antibody Concentration: Approx. 1.0 mg/ml

Precautions and Disclaimer

Due to the sodium azide content, a material safety data sheet (MSDS) for this product has been sent to the attention of the safety officer of your institution. Consult the MSDS for information regarding hazards and safe handling practices.

Storage/Stability

For continuous use, store at 2-8 °C for up to one month. For prolonged storage, freeze in working aliquots at -20 °C. Repeated freezing and thawing is not recommended. Storage in frost-free freezers is also not recommended. If slight turbidity occurs upon prolonged storage, clarify the solution by centrifugation before use. Working dilutions should be discarded if not used within 12 hours.

Product Profile

For immunoblotting, a working antibody concentration of 1-2 µg/ml is recommended using a whole extract of mouse NIH-3T3 fibroblast cells and a chemiluminescent detection reagent.

For indirect immunofluorescence, a working antibody concentration of 1-2 µg/ml is recommended using cultured 293T cells expressing recombinant mouse HDAC5.

For immunoprecipitation, 1-2 µg of the antibody immunoprecipitates HDAC5 from an extract of 293T cells expressing recombinant mouse HDAC5.

Note: In order to obtain the best results using various techniques and preparations, we recommend determining the optimal working dilutions by titration.

References

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5. Fischle, W., et al., *J. Biol. Chem.*, **274**, 11713-11720 (1999).
6. McKinsey, T.A., et al., *Nature*, **408**, 106-111 (2000).
7. Huang, Y., et al., *Cancer Res.*, **62**, 2913-2922 (2002).

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