

HDAC7 Antibody (N-term) Blocking Peptide

Synthetic peptide Catalog # BP1107b

Specification

HDAC7 Antibody (N-term) Blocking Peptide - Product Information

Primary Accession <u>Q8WUI4</u>
Other Accession <u>NP_056216</u>

HDAC7 Antibody (N-term) Blocking Peptide - Additional Information

Gene ID 51564

Other Names

Histone deacetylase 7, HD7, Histone deacetylase 7A, HD7a, HDAC7, HDAC7A

Target/Specificity

The synthetic peptide sequence used to generate the antibody AP1107b was selected from the N-term region of human HDAC7. A 10 to 100 fold molar excess to antibody is recommended. Precise conditions should be optimized for a particular assay.

Format

Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

Storage

Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

Precautions

This product is for research use only. Not for use in diagnostic or therapeutic procedures.

HDAC7 Antibody (N-term) Blocking Peptide - Protein Information

Name HDAC7

Synonyms HDAC7A

Function

Responsible for the deacetylation of lysine residues on the N-terminal part of the core histones (H2A, H2B, H3 and H4). Histone deacetylation gives a tag for epigenetic repression and plays an important role in transcriptional regulation, cell cycle progression and developmental events. Histone deacetylases act via the formation of large multiprotein complexes. Involved in muscle maturation by repressing transcription of myocyte enhancer factors such as MEF2A, MEF2B and MEF2C. During muscle differentiation, it shuttles into the cytoplasm, allowing the expression of myocyte enhancer factors (By similarity). May be involved in Epstein-Barr virus (EBV) latency, possibly by repressing the viral BZLF1 gene. Positively regulates the transcriptional repressor activity of FOXP3 (PubMed:<a href="http://www.uniprot.org/citations/17360565"



target="_blank">17360565). Serves as a corepressor of RARA, causing its deacetylation and inhibition of RARE DNA element binding (PubMed:28167758). In association with RARA, plays a role in the repression of microRNA-10a and thereby in the inflammatory response (PubMed:28167758).

Cellular Location

Nucleus. Cytoplasm. Note=In the nucleus, it associates with distinct subnuclear dot-like structures. Shuttles between the nucleus and the cytoplasm. Treatment with EDN1 results in shuttling from the nucleus to the perinuclear region. The export to cytoplasm depends on the interaction with the 14-3-3 protein YWHAE and is due to its phosphorylation

HDAC7 Antibody (N-term) Blocking Peptide - Protocols

Provided below are standard protocols that you may find useful for product applications.

Blocking Peptides

HDAC7 Antibody (N-term) Blocking Peptide - Images

HDAC7 Antibody (N-term) Blocking Peptide - Background

Histone deacetylase (HDAC) and histone acetyltransferase (HAT) are enzymes that regulate transcription by selectively deacetylating or acetylating the eta-amino groups of lysines located near the amino termini of core histone proteins (1). Eight members of HDAC family have been identified in the past several years (2,3). These HDAC family members are divided into two classes, I and II. Class I of the HDAC family comprises four members, HDAC-1, 2, 3, and 8, each of which contains a deacetylase domain exhibiting from 45 to 93% identity in amino acid sequence. Class II of the HDAC family comprises HDAC-4, 5, 6, and 7, the molecular weights of which are all about two-fold larger than those of the class I members, and the deacetylase domains are present within the C-terminal regions, except that HDAC-6 contains two copies of the domain, one within each of the N-terminal and C-terminal regions. Human HDAC-1, 2 and 3 were expressed in various tissues, but the others (HDAC-4, 5, 6, and 7) showed tissue-specific expression patterns (3). These results suggested that each member of the HDAC family exhibits a different, individualsubstrate specificity and function in vivo.

HDAC7 Antibody (N-term) Blocking Peptide - References

Meinke PT and Liberator P. Curr Med Chem, 8(2): 211-235 (2001). Nakayama T and Takami Y. J Biochem (Tokyo) 129 (4): 491-499 (2001). Cress, W.D. and Seto, E. J. Cell. Physiol. 184, 1-16 (2000).