

## JMJD5 Antibody (C-term 2) Blocking Peptide Synthetic peptide

Catalog # BP1031a

## Specification

# JMJD5 Antibody (C-term 2) Blocking Peptide - Product Information

Primary Accession

## <u>Q8N371</u>

# JMJD5 Antibody (C-term 2) Blocking Peptide - Additional Information

Gene ID 79831

#### **Other Names**

Lysine-specific demethylase 8, JmjC domain-containing protein 5, Jumonji domain-containing protein 5, KDM8, JMJD5

### Target/Specificity

The synthetic peptide sequence used to generate the antibody <a href=/products/AP1031a>AP1031a</a> was selected from the C-term region of human JMJD5. 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.

# JMJD5 Antibody (C-term 2) Blocking Peptide - Protein Information

## Name KDM8

## Function

Bifunctional enzyme that acts both as an endopeptidase and 2- oxoglutarate-dependent monooxygenase (PubMed:<a href="http://www.uniprot.org/citations/28847961" target="\_blank">28847961</a>, PubMed:<a href="http://www.uniprot.org/citations/29459673" target="\_blank">29459673</a>, PubMed:<a href="http://www.uniprot.org/citations/28982940" target="\_blank">28982940</a>, PubMed:<a href="http://www.uniprot.org/citations/28982940" target="\_blank">29563586</a>). Endopeptidase that cleaves histones N-terminal tails at the carboxyl side of methylated arginine or lysine residues, to generate 'tailless nucleosomes', which may trigger transcription elongation (PubMed:<a

href="http://www.uniprot.org/citations/28847961" target="\_blank">28847961</a>, PubMed:<a href="http://www.uniprot.org/citations/29459673" target="\_blank">29459673</a>, PubMed:<a href="http://www.uniprot.org/citations/28982940" target="\_blank">28882940</a>). Preferentially



recognizes and cleaves monomethylated and dimethylated arginine residues of histones H2, H3 and H4. After initial cleavage, continues to digest histones tails via its aminopeptidase activity (PubMed:<a href="http://www.uniprot.org/citations/28847961" target="\_blank">28847961</a>, PubMed:<a href="http://www.uniprot.org/citations/29459673" target="\_blank">29459673</a>). Upon DNA damage, cleaves the N-terminal tail of histone H3 at monomethylated lysine residues, preferably at monomethylated 'Lys-9' (H3K9me1). The histone variant H3F3A is the major target for cleavage (PubMed:<a href="http://www.uniprot.org/citations/28982940" target=" blank">28982940</a>). Additionally, acts as a Fe(2+) and 2-oxoglutarate- dependent monooxygenase, catalyzing (R)-stereospecific hydroxylation at C-3 of 'Arg-137' of RPS6 and 'Arg-141' of RCCD1, but the biological significance of this activity remains to be established (PubMed:<a href="http://www.uniprot.org/citations/29563586" target=" blank">29563586</a>). Regulates mitosis through different mechanisms: Plays a role in transcriptional repression of satellite repeats, possibly by regulating H3K36 methylation levels in centromeric regions together with RCCD1. Possibly together with RCCD1, is involved in proper mitotic spindle organization and chromosome segregation (PubMed:<a href="http://www.uniprot.org/citations/24981860" target=" blank">24981860</a>). Negatively regulates cell cycle repressor CDKN1A/p21, which controls G1/S phase transition (PubMed:<a href="http://www.uniprot.org/citations/24740926" target=" blank">24740926</a>). Required for G2/M phase cell cycle progression. Regulates expression of CCNA1/cyclin-A1, leading to cancer cell proliferation (PubMed:<a href="http://www.uniprot.org/citations/20457893" target=" blank">20457893</a>). Also, plays a role in regulating alpha-tubulin acetylation and cytoskeletal microtubule stability involved in epithelial to mesenchymal transition (PubMed:<a href="http://www.uniprot.org/citations/28455245" target=" blank">28455245</a>). Regulates the circadian gene expression in the liver (By similarity). Represses the transcriptional activator activity of the CLOCK-BMAL1 heterodimer in a catalytically-independent manner (PubMed: <a href="http://www.uniprot.org/citations/30500822" target="\_blank">30500822</a>). Negatively regulates the protein stability and function of CRY1; required for AMPK-FBXL3-induced CRY1 degradation (PubMed:<a href="http://www.uniprot.org/citations/30500822"

target="\_blank">30500822</a>).

**Cellular Location** Nucleus. Chromosome Note=Colocalizes with trimethylated 'Lys-9' of histone H3 (H3K9me3)

**Tissue Location** 

Weakly expressed in most cells. Highly expressed in breast cancer cells (PubMed:20457893). Expressed in embryonic stem cells (PubMed:24740926).

# JMJD5 Antibody (C-term 2) Blocking Peptide - Protocols

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

### <u>Blocking Peptides</u>

JMJD5 Antibody (C-term 2) Blocking Peptide - Images

## JMJD5 Antibody (C-term 2) Blocking Peptide - Background

Covalent modification of histones plays critical role in regulating chromatin structure and transcription. While most covalent histone modifications are reversible, only recently has it been established that methyl groups are subject to enzymatic removal from histones. A family of novel JmjC domain-containing histone demethylation (JHDM) enzymes have been identified that perform this specific function. Histone demethylation by JHDM proteins requires cofactors Fe(II) and alpha-ketoglutarate. Family members include JHDM1 (demethylating histone 3 at lysine 36), and JHDM2A as well as JMJD2CH3K9 (both of which demethylate histone 3 at lysine 9). Contributions of histone demethylase activity to tumor development, decreases in cell proliferation, and hormone-dependent transcriptional activation have been observed.



# JMJD5 Antibody (C-term 2) Blocking Peptide - References

Ota, T., et al., Nat. Genet. 36(1):40-45 (2004).