

EZH2 Antibody (Center) Blocking Peptide
Synthetic peptide
Catalog # BP2512c**Specification**

EZH2 Antibody (Center) Blocking Peptide - Product InformationPrimary Accession [Q15910](#)**EZH2 Antibody (Center) Blocking Peptide - Additional Information****Gene ID** 2146**Other Names**

Histone-lysine N-methyltransferase EZH2, ENX-1, Enhancer of zeste homolog 2, Lysine N-methyltransferase 6, EZH2, KMT6

Target/Specificity

The synthetic peptide sequence used to generate the antibody [AP2512c](/product/products/AP2512c) was selected from the Center region of human EZH2. 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.

EZH2 Antibody (Center) Blocking Peptide - Protein Information**Name** EZH2 ([HGNC:3527](#))**Synonyms** KMT6**Function**

Catalytic subunit of the PRC2/EED-EZH2 complex, a Polycomb group (PcG) complex that methylates 'Lys-9' (H3K9me) and 'Lys-27' (H3K27me) of histone H3, leading to transcriptional repression of the affected target gene (PubMed: [14532106](http://www.uniprot.org/citations/14532106), PubMed: [15225548](http://www.uniprot.org/citations/15225548), PubMed: [15385962](http://www.uniprot.org/citations/15385962), PubMed: [16618801](http://www.uniprot.org/citations/16618801), PubMed: [16936726](http://www.uniprot.org/citations/16936726), PubMed: [17344414](http://www.uniprot.org/citations/17344414)).

[22323599](http://www.uniprot.org/citations/22323599), PubMed: [24474760](http://www.uniprot.org/citations/24474760), PubMed: [26581166](http://www.uniprot.org/citations/26581166), PubMed: [30026490](http://www.uniprot.org/citations/30026490), PubMed: [30923826](http://www.uniprot.org/citations/30923826)). Able to mono-, di- and trimethylate 'Lys-27' of histone H3 to form H3K27me1, H3K27me2 and H3K27me3, respectively (PubMed: [15231737](http://www.uniprot.org/citations/15231737), PubMed: [17210787](http://www.uniprot.org/citations/17210787), PubMed: [18285464](http://www.uniprot.org/citations/18285464), PubMed: [22323599](http://www.uniprot.org/citations/22323599), PubMed: [30923826](http://www.uniprot.org/citations/30923826)). Displays a preference for substrates with less methylation, loses activity when progressively more methyl groups are incorporated into H3K27, H3K27me0 > H3K27me1 > H3K27me2 (PubMed: [22323599](http://www.uniprot.org/citations/22323599), PubMed: [30923826](http://www.uniprot.org/citations/30923826)). Compared to EZH1-containing complexes, it is more abundant in embryonic stem cells and plays a major role in forming H3K27me3, which is required for embryonic stem cell identity and proper differentiation (PubMed: [19026781](http://www.uniprot.org/citations/19026781)). The PRC2/EED-EZH2 complex may also serve as a recruiting platform for DNA methyltransferases, thereby linking two epigenetic repression systems (PubMed: [16357870](http://www.uniprot.org/citations/16357870), PubMed: [17200670](http://www.uniprot.org/citations/17200670)). Genes repressed by the PRC2/EED- EZH2 complex include HOXC8, HOXA9, MYT1, CDKN2A and retinoic acid target genes (PubMed: [16179254](http://www.uniprot.org/citations/16179254), PubMed: [18086877](http://www.uniprot.org/citations/18086877), PubMed: [20935635](http://www.uniprot.org/citations/20935635)). EZH2 can also methylate non-histone proteins such as the transcription factor GATA4 and the nuclear receptor RORA (PubMed: [23063525](http://www.uniprot.org/citations/23063525)). Regulates the circadian clock via histone methylation at the promoter of the circadian genes (PubMed: [16717091](http://www.uniprot.org/citations/16717091)). Essential for the CRY1/2-mediated repression of the transcriptional activation of PER1/2 by the CLOCK- BMAL1 heterodimer; involved in the di and trimethylation of 'Lys-27' of histone H3 on PER1/2 promoters which is necessary for the CRY1/2 proteins to inhibit transcription (By similarity).

Cellular Location

Nucleus. Note=Localizes to the inactive X chromosome in trophoblast stem cells.
{ECO:0000250|UniProtKB:Q61188}

Tissue Location

In the ovary, expressed in primordial follicles and oocytes and also in external follicle cells (at protein level) (PubMed:31451685). Expressed in many tissues (PubMed:14532106) Overexpressed in numerous tumor types including carcinomas of the breast, colon, larynx, lymphoma and testis (PubMed:14532106)

EZH2 Antibody (Center) Blocking Peptide - Protocols

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

- [Blocking Peptides](#)

EZH2 Antibody (Center) Blocking Peptide - Images

EZH2 Antibody (Center) Blocking Peptide - Background

EZH2, SUZ12, and EED form a complex that methylates nucleosomal histone H3 at Lys27. EZH2

contains a SET domain, a signature motif for all known histone lysine methyltransferases except the H3-K79 methyltransferase DOT1, and is therefore likely to be the catalytic subunit. Consequently, EZH2 is thought to regulate gene expression by controlling chromatin structure. Several lines of evidence suggested a critical role for the EZH2 protein during normal and perturbed development of the hematopoietic and central nervous systems. The EZH2 protein has been shown to associate with the VAV1 protooncogene protein and with the XNP protein, the product of a gene associated with mental retardation. Additionally, due to mapping of EZH2 to the 7q35-q36 chromosomal region associated with myeloid disorders, this protein is suggested to participate in the genetic events triggering myeloid leukemia.

EZH2 Antibody (Center) Blocking Peptide - References

Cardoso, C., et al., Hum. Mol. Genet. 7(4):679-684 (1998). Laible, G., et al., EMBO J. 16(11):3219-3232 (1997). Hobert, O., et al., Mol. Cell. Biol. 16(6):3066-3073 (1996). Chen, H., et al., Genomics 38(1):30-37 (1996).