

Mouse Cdk9 Antibody (C-term) Blocking Peptide
Synthetic peptide
Catalog # BP16162b**Specification**

Mouse Cdk9 Antibody (C-term) Blocking Peptide - Product InformationPrimary Accession [Q99J95](#)**Mouse Cdk9 Antibody (C-term) Blocking Peptide - Additional Information****Gene ID** 107951**Other Names**

Cyclin-dependent kinase 9, Cell division protein kinase 9, Cdk9

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.

Mouse Cdk9 Antibody (C-term) Blocking Peptide - Protein Information**Name** Cdk9**Function**

Protein kinase involved in the regulation of transcription. Member of the cyclin-dependent kinase pair (CDK9/cyclin-T) complex, also called positive transcription elongation factor b (P-TEFb), which facilitates the transition from abortive to productive elongation by phosphorylating the CTD (C-terminal domain) of the large subunit of RNA polymerase II (RNAP II) POLR2A, SUPT5H and RDBP. This complex is inactive when in the 7SK snRNP complex form. Phosphorylates EP300, MYOD1, RPB1/POLR2A and AR and the negative elongation factors DSIF and NELFE. Regulates cytokine inducible transcription networks by facilitating promoter recognition of target transcription factors (e.g. TNF-inducible RELA/p65 activation and IL-6-inducible STAT3 signaling). Promotes RNA synthesis in genetic programs for cell growth, differentiation and viral pathogenesis. P-TEFb is also involved in cotranscriptional histone modification, mRNA processing and mRNA export. Modulates a complex network of chromatin modifications including histone H2B monoubiquitination (H2Bub1), H3 lysine 4 trimethylation (H3K4me3) and H3K36me3; integrates phosphorylation during transcription with chromatin modifications to control co-transcriptional histone mRNA processing. The CDK9/cyclin-K complex has also a kinase activity towards CTD of RNAP II and can substitute for CDK9/cyclin-T P-TEFb in vitro. Replication stress response protein; the CDK9/cyclin-K complex is required for genome integrity maintenance, by promoting cell cycle recovery from replication arrest and limiting single-stranded DNA amount in response to replication stress, thus reducing the breakdown of stalled replication forks and avoiding DNA

damage. In addition, probable function in DNA repair of isoform 2 via interaction with KU70/XRCC6. Promotes cardiac myocyte enlargement. RPB1/POLR2A phosphorylation on 'Ser-2' in CTD activates transcription. AR phosphorylation modulates AR transcription factor promoter selectivity and cell growth. DSIF and NELF phosphorylation promotes transcription by inhibiting their negative effect. The phosphorylation of MYOD1 enhances its transcriptional activity and thus promotes muscle differentiation. Catalyzes phosphorylation of KAT5, promoting KAT5 recruitment to chromatin and histone acetyltransferase activity.

Cellular Location

Nucleus. Cytoplasm. Nucleus, PML body. Note=Accumulates on chromatin in response to replication stress. Complexed with CCNT1 in nuclear speckles, but uncomplexed form in the cytoplasm. The translocation from nucleus to cytoplasm is XPO1/CRM1-dependent Associates with PML body when acetylated (By similarity)

Tissue Location

Expressed at high levels in brain and kidney.

Mouse Cdk9 Antibody (C-term) Blocking Peptide - Protocols

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

- [Blocking Peptides](#)

Mouse Cdk9 Antibody (C-term) Blocking Peptide - Images**Mouse Cdk9 Antibody (C-term) Blocking Peptide - Background**

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Mouse Cdk9 Antibody (C-term) Blocking Peptide - References

Yokoyama, S., et al. Dev. Cell 17(6):836-848(2009)Alarcon, C., et al. Cell 139(4):757-769(2009)Takaya, T., et al. Circ. J. 73(8):1492-1497(2009)Kohoutek, J., et al. Mol. Cell. Biol. 29(12):3280-3285(2009)Elagib, K.E., et al. Blood 112(13):4884-4894(2008)