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CDK9 Antibody (C-term) Blocking peptide
Synthetic peptide
Catalog # BP13892b

Specification

CDK9 Antibody (C-term) Blocking peptide - Product Information

Primary Accession P50750

CDK9 Antibody (C-term) Blocking peptide - Additional Information

Gene ID 1025

Other Names
Cyclin-dependent kinase 9, C-2K, Cell division cycle 2-like protein kinase 4, Cell division protein kinase 9, Serine/threonine-protein kinase PITALRE, Tat-associated kinase complex catalytic subunit, CDK9, CDC2L4, TAK

Target/Specificity
The synthetic peptide sequence used to generate the antibody AP13892b was selected from the C-term region of CDK9. 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.

CDK9 Antibody (C-term) Blocking peptide - Protein Information

Name CDK9 {ECO:0000303|PubMed:10903437, ECO:0000312|HGNC:HGNC:1780}

Function
Protein kinase involved in the regulation of transcription (PubMed:10574912, PubMed:10757782, PubMed:11145967, PubMed:11575923, PubMed:11809800, PubMed:11884399, PubMed:14701750, PubMed:16109376, PubMed:16109377, PubMed:20930849, PubMed:<a

href="http://www.uniprot.org/citations/28426094" target="_blank">28426094, PubMed:29335245). 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 (PubMed:10574912, PubMed:10757782, PubMed:11145967, PubMed:11575923, PubMed:11809800, PubMed:11884399, PubMed:14701750, PubMed:16109376, PubMed:16109377, PubMed:20930849, PubMed:28426094, PubMed:30134174). This complex is inactive when in the 7SK snRNP complex form (PubMed:10574912, PubMed:10757782, PubMed:11145967, PubMed:11575923, PubMed:11809800, PubMed:11884399, PubMed:14701750, PubMed:16109376, PubMed:16109377, PubMed:20930849, PubMed:28426094). Phosphorylates EP300, MYOD1, RPB1/POLR2A and AR and the negative elongation factors DSIF and NELFE (PubMed:9857195, PubMed:10912001, PubMed:11112772, PubMed:12037670, PubMed:20081228, PubMed:20980437, PubMed:21127351). 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) (PubMed:17956865, PubMed:18362169). Promotes RNA synthesis in genetic programs for cell growth, differentiation and viral pathogenesis (PubMed:10393184, PubMed:11112772). P-TEFb is also involved in cotranscriptional histone modification, mRNA processing and mRNA export (PubMed:15564463, PubMed:19575011, PubMed:19844166). 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 (PubMed:15564463, PubMed:19575011, PubMed:19844166). 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 (PubMed:21127351). 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 (PubMed:20493174). In addition, probable function in DNA repair of isoform 2 via interaction with KU70/XRCC6 (PubMed:20493174). Promotes cardiac myocyte enlargement (PubMed:20081228). RPB1/POLR2A phosphorylation on 'Ser-2' in CTD activates transcription (PubMed:21127351). AR phosphorylation modulates AR transcription factor promoter selectivity and cell growth. DSIF and NELF phosphorylation promotes transcription by inhibiting their negative effect (PubMed:9857195, PubMed:10912001, PubMed:11112772). The phosphorylation of MYOD1 enhances its transcriptional activity and thus promotes muscle differentiation (PubMed:12037670). Catalyzes phosphorylation of KAT5, promoting KAT5 recruitment to chromatin and histone acetyltransferase activity (PubMed:29335245).

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

Tissue Location
Ubiquitous.

CDK9 Antibody (C-term) Blocking peptide - Protocols

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

- Blocking Peptides

CDK9 Antibody (C-term) Blocking peptide - Images

CDK9 Antibody (C-term) Blocking peptide - Background

The protein encoded by this gene is a member of thecyclin-dependent protein kinase (CDK) family. CDK family membersare highly similar to the gene products of S. cerevisiae cdc28, andS. pombe cdc2, and known as important cell cycle regulators. Thiskinase was found to be a component of the multiprotein complexTAK/P-TEFb, which is an elongation factor for RNA polymeraseII-directed transcription and functions by phosphorylating theC-terminal domain of the largest subunit of RNA polymerase II. Thisprotein forms a complex with and is regulated by its regulatorysubunit cyclin T or cyclin K. HIV-1 Tat protein was found tointeract with this protein and cyclin T, which suggested a possibleinvolvement of this protein in AIDS.

CDK9 Antibody (C-term) Blocking peptide - References

Dow, E.C., et al. J. Cell. Physiol. 224(1):84-93(2010)Liu, H., et al. Biochem. Biophys. Res. Commun. 397(2):245-250(2010)Pirngruber, J., et al. Oncogene 29(19):2853-2863(2010)Belakavadi, M., et al. Mol. Cell. Biol. 30(10):2437-2448(2010)Khiati, A., et al. J. Neurovirol. 16(2):150-167(2010)