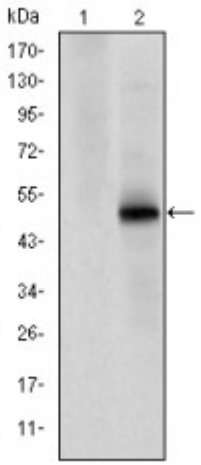
Anti-Cdk9 antibody



Description	Mouse monoclonal to Cdk9.

Model	STJ97949
Host	Mouse
Reactivity	Human
Applications	ELISA, IHC, WB
Immunogen	Purified recombinant fragment of human Cdk9 expressed in E. Coli.
Gene ID	1025
Gene Symbol	CDK9
Dilution range	WB 1:500-1:2000IHC 1:200-1:1000ELISA 1:10000
Specificity	Cdk9 Monoclonal Antibody detects endogenous levels of Cdk9 protein.
Tissue Specificity	Ubiquitous.
Purification	Affinity purification
Clone ID	1B5A7
Note	For Research Use Only (RUO).
Protein Name	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
Clonality	Monoclonal
Conjugation	Unconjugated

Isotype	IgG1
Formulation	Ascitic fluid containing 0.03% sodium azide.
Storage Instruction	Store at -20°C, and avoid repeat freeze-thaw cycles.
Database Links	HGNC:1780OMIM:603251
Alternative 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
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 NELF. 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.
Cellular Localization	Nucleus. Cytoplasm. Nucleus, PML body. 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.
Post-translational Modifications	Autophosphorylation at Thr-186, Ser-347, Thr-350, Ser-353, Thr-354 and Ser-357 triggers kinase activity by promoting cyclin and substrate binding (e.g. HIV TAT) upon conformational changes. Thr-186 phosphorylation requires the calcium Ca(2+) signaling pathway, including CaMK1D and calmodulin. This inhibition is relieved by Thr-29 dephosphorylation. However, phosphorylation at Thr-29 is inhibitory within the HIV transcription

initiation complex. Phosphorylation at Ser-175 inhibits kinase activity. Can be phosphorylated on either Thr-362 or Thr-363 but not on both simultaneously . Dephosphorylation of Thr-186 by PPM1A and PPM1B blocks CDK9 activity and may lead to CDK9 proteasomal degradation. However, PPP1CA-mediated Thr-186 dephosphorylation is required to release P-TEFb from its inactive P-TEFb/7SK snRNP complex. Dephosphorylation of C-terminus Thr and Ser residues by protein phosphatase-1 (PP1) triggers CDK9 activity, contributing to the activation of HIV-1 transcription.; N6-acetylation of Lys-44 by CBP/p300 promotes kinase activity, whereas acetylation of both Lys-44 and Lys-48 mediated by PCAF/KAT2B and GCN5/KAT2A reduces kinase activity. The acetylated form associates with PML bodies in the nuclear matrix and with the transcriptionally silent HIV-1 genome; deacetylated upon transcription stimulation. Polyubiquitinated and thus activated by UBR5. This ubiquitination is promoted by TFIIS/TCEA1 and favors 'Ser-2' phosphorylation of RPB1/POLR2A CTD.