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CDK7 Antibody (C-term) Blocking Peptide
Synthetic peptide
Catalog # BP7523b

Specification

CDK7 Antibody (C-term) Blocking Peptide - Product Information

Primary Accession P50613

CDK7 Antibody (C-term) Blocking Peptide - Additional Information

Gene ID 1022

Other Names
Cyclin-dependent kinase 7, 39 kDa protein kinase, p39 Mo15, CDK-activating kinase 1, Cell division protein kinase 7, Serine/threonine-protein kinase 1, TFIIH basal transcription factor complex kinase subunit, CDK7, CAK, CAK1, CDKN7, MO15, STK1

Target/Specificity
The synthetic peptide sequence used to generate the antibody AP7523b was selected from the C-term region of human CDK7 . 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.

CDK7 Antibody (C-term) Blocking Peptide - Protein Information

Name CDK7

Synonyms CAK, CAK1, CDKN7, MO15, STK1

Function
Serine/threonine kinase involved in cell cycle control and in RNA polymerase II-mediated RNA transcription. Cyclin-dependent kinases (CDKs) are activated by the binding to a cyclin and mediate the progression through the cell cycle. Each different complex controls a specific transition between 2 subsequent phases in the cell cycle. Required for both activation and complex formation of CDK1/cyclin-B during G2-M transition, and for activation of CDK2/cyclins during G1-S transition (but not complex formation). CDK7 is the catalytic subunit of the CDK-activating kinase (CAK) complex. Phosphorylates SPT5/SUPT5H, SF1/NR5A1, POLR2A, p53/TP53, CDK1, CDK2, CDK4, CDK6 and CDK11B/CDK11. CAK activates the cyclin-associated

kinases CDK1, CDK2, CDK4 and CDK6 by threonine phosphorylation, thus regulating cell cycle progression. CAK complexed to the core-TFIIH basal transcription factor activates RNA polymerase II by serine phosphorylation of the repetitive C- terminal domain (CTD) of its large subunit (POLR2A), allowing its escape from the promoter and elongation of the transcripts (PubMed:9852112). Phosphorylation of POLR2A in complex with DNA promotes transcription initiation by triggering dissociation from DNA. Its expression and activity are constant throughout the cell cycle. Upon DNA damage, triggers p53/TP53 activation by phosphorylation, but is inactivated in turn by p53/TP53; this feedback loop may lead to an arrest of the cell cycle and of the transcription, helping in cell recovery, or to apoptosis. Required for DNA-bound peptides-mediated transcription and cellular growth inhibition.

Cellular Location
Nucleus. Cytoplasm. Cytoplasm, perinuclear region. Note=Colocalizes with PRKCI in the cytoplasm and nucleus (PubMed:15695176). Translocates from the nucleus to cytoplasm and perinuclear region in response to DNA-bound peptides (PubMed:19071173).

Tissue Location
Ubiquitous.

CDK7 Antibody (C-term) Blocking Peptide - Protocols

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

- Blocking Peptides

CDK7 Antibody (C-term) Blocking Peptide - Images

CDK7 Antibody (C-term) Blocking Peptide - Background

CDK7 is a member of the cyclin-dependent protein kinase (CDK) family. CDK family members are highly similar to the gene products of Saccharomyces cerevisiae cdc28, and Schizosaccharomyces pombe cdc2, and are known to be important regulators of cell cycle progression. This protein forms a trimeric complex with cyclin H and MAT1, which functions as a Cdk-activating kinase (CAK). It is an essential component of the transcription factor TFIIH, that is involved in transcription initiation and DNA repair. This protein is thought to serve as a direct link between the regulation of transcription and the cell cycle.

CDK7 Antibody (C-term) Blocking Peptide - References

Kino, T., et al., Biochem. Biophys. Res. Commun. 298(1):17-23 (2002).Schneider, E., et al., Oncogene 21(33):5031-5037 (2002).Bochar, D.A., et al., J. Biol. Chem. 274(19):13162-13166 (1999).Akoulitchev, S., et al., Genes Dev. 12(22):3541-3550 (1998).Yee, A., et al., Cancer Res. 55(24):6058-6062 (1995).