

# Phospho-Cdk7-T170 Antibody Blocking Peptide

Synthetic peptide Catalog # BP3696a

## **Specification**

# Phospho-Cdk7-T170 Antibody Blocking Peptide - Product Information

**Primary Accession** 

P50613

## Phospho-Cdk7-T170 Antibody 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

#### **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.

# Phospho-Cdk7-T170 Antibody 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:<a href="http://www.uniprot.org/citations/9852112" target="\_blank">9852112</a>/a>). 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.

## Phospho-Cdk7-T170 Antibody Blocking Peptide - Protocols

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

## • Blocking Peptides

Phospho-Cdk7-T170 Antibody Blocking Peptide - Images

## Phospho-Cdk7-T170 Antibody Blocking Peptide - Background

This gene encodes a member of the syntaxin superfamily. Syntaxins are nervous system-specific proteins implicated in the docking of synaptic vesicles with the presynaptic plasma membrane. Syntaxins possess a single C-terminal transmembrane domain, a SNARE [Soluble NSF (N-ethylmaleimide-sensitive fusion protein)-Attachment protein REceptor] domain (known as H3), and an N-terminal regulatory domain (Habc). Syntaxins bind synaptotagmin in a calcium-dependent fashion and interact with voltage dependent calcium and potassium channels via the C-terminal H3 domain. This gene product is a key molecule in ion channel regulation and synaptic exocytosis.

### Phospho-Cdk7-T170 Antibody Blocking Peptide - References

Yoshida, T., et al. Int. J. Mol. Med. 24(2):233-246(2009)Hamdan, F.F., et al. Ann. Neurol. 65(6):748-753(2009)Corominas, R., et al. Neurosci. Lett. 455(2):105-109(2009)Chen, C.S., et al. J. Biol. Chem. 284(11):6877-6884(2009)Ramakrishnan, N.A., et al. J. Biol. Chem. 284(3):1364-1372(2009)Tian, J.H., et al. J. Biol. Chem. 278(28):26265-26274(2003)