

### **CDK7 Antibody**

Purified Rabbit Polyclonal Antibody (Pab) Catalog # AP51071

### **Specification**

### **CDK7 Antibody - Product Information**

Application WB, IHC-P, E
Primary Accession P50613
Reactivity Human
Host Rabbit
Clonality Polyclonal
Calculated MW 40 KDa

## **CDK7 Antibody - 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

## **Dilution**

WB~~1:1000 IHC-P~~N/A E~~N/A

#### Format

0.01M PBS, pH 7.2, 0.09% (W/V) Sodium azide, Glycerol 50%

### **Storage**

Store at -20 °C.Stable for 12 months from date of receipt

# **CDK7 Antibody - 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 (PubMed:<a href="http://www.uniprot.org/citations/9852112" target="\_blank">9852112</a>, PubMed:<a href="http://www.uniprot.org/citations/19136461" target="\_blank">19136461</a>, PubMed:<a href="http://www.uniprot.org/citations/26257281" target="\_blank">26257281</a>, PubMed:<a href="http://www.uniprot.org/citations/28768201" target="\_blank">28768201</a>, PubMed:<a href="http://www.uniprot.org/citations/28768201" target="\_blank">28768201</a>). 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 (PubMed: <a href="http://www.uniprot.org/citations/9372954" target=" blank">9372954</a>, PubMed:<a href="http://www.uniprot.org/citations/9840937" target="\_blank">9840937</a>, PubMed:<a href="http://www.uniprot.org/citations/19136461" target=" blank">19136461</a>, PubMed:<a href="http://www.uniprot.org/citations/26257281" target="blank">26257281</a>, PubMed:<a href="http://www.uniprot.org/citations/28768201" target="blank">28768201</a>). Initiates transcription by RNA polymerase II by mediating phosphorylation of POLR2A at 'Ser-5' of the repetitive C- terminal domain (CTD) when POLR2A is in complex with DNA, promoting dissociation from DNA and initiation (PubMed: <a href="http://www.uniprot.org/citations/19136461" target=" blank">19136461</a>, PubMed:<a href="http://www.uniprot.org/citations/26257281" target="blank">26257281</a>, PubMed:<a href="http://www.uniprot.org/citations/28768201" target="blank">28768201</a>). 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 CTD of 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>). 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 - Protocols**

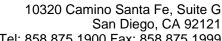
Provided below are standard protocols that you may find useful for product applications.

- Western Blot
- Blocking Peptides
- Dot Blot
- Immunohistochemistry
- Immunofluorescence
- <u>Immunoprecipitation</u>
- Flow Cytomety
- Cell Culture

# CDK7 Antibody - Images

# CDK7 Antibody - Background

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





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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-terminus domain (CTD) of its large subunit (POLR2A), allowing its escape from the promoter and elongation of the transcripts. 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.

## **CDK7 Antibody - References**

Tassan J.-P., et al.J. Cell Biol. 127:467-478(1994). Levedakou E.N., et al. Oncogene 9:1977-1988(1994). Darbon J.-M., et al. Oncogene 9:3127-3138(1994). Wu L., et al. Oncogene 9:2089-2096(1994). Kobelt D., et al. Oncol. Rep. 1:1269-1275(1994).