

hCdk7-T170

Purified Rabbit Polyclonal Antibody (Pab) Catalog # AP22377a

Specification

hCdk7-T170 - Product Information

| Application | WB,E |
|-------------------|---------------|
| Primary Accession | <u>P50613</u> |
| Other Accession | <u>Q03147</u> |
| Reactivity | Human |
| Predicted | Mouse |
| Host | Rabbit |
| Clonality | polyclonal |
| Isotype | Rabbit IgG |
| Calculated MW | 39038 |

hCdk7-T170 - Additional Information

Gene ID 1022

Other Names

Cyclin-dependent kinase 7, 2.7.11.22, 2.7.11.23, 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

This antibody is generated from a rabbit immunized with a KLH conjugated synthetic peptide between amino acids from human.

Dilution WB~~1:1000 E~~Use at an assay dependent concentration.

Format

Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is purified through a protein A column, followed by peptide affinity purification.

Storage

Maintain refrigerated at 2-8°C for up to 2 weeks. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.

Precautions hCdk7-T170 is for research use only and not for use in diagnostic or therapeutic procedures.

hCdk7-T170 - 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:<u>9852112</u>, PubMed:<u>19136461</u>, PubMed:<u>26257281</u>, PubMed:<u>28768201</u>). 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: 9372954, PubMed: 9840937, PubMed: 19136461, PubMed: 26257281, PubMed:28768201). 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:19136461, PubMed:26257281, PubMed:28768201). 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:<u>9852112</u>). 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.

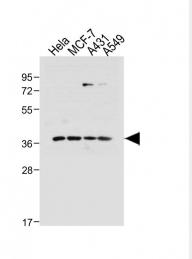
hCdk7-T170 - Protocols

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

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

hCdk7-T170 - Images





All lanes : Anti-hCdk7-T170 at 1:1000 dilution Lane 1: Hela whole cell lysate Lane 2: MCF-7 whole cell lysate Lane 3: A431 whole cell lysate Lane 4: A549 whole cell lysate Lysates/proteins at 20 µg per lane. Secondary Goat Anti-Rabbit IgG, (H+L), Peroxidase conjugated at 1/10000 dilution. Predicted band size : 39 kDa Blocking/Dilution buffer: 5% NFDM/TBST.

hCdk7-T170 - 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 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. 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.

hCdk7-T170 - 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).