CDK7&CCNH&MNAT1 Protein, Human (sf9, His)

Cat. No.:	HY-P75361
Synonyms:	CAK1 Protein; CDKN7 Protein; HCAK Protein; MO15 Protein; p39MO15 Protein; STK1 Protein
Species:	Human
Source:	Sf9 insect cells
Accession:	P50613 (A2-F346)&P51946 (Y2-L323)&P51948 (D2-S309)
Gene ID:	1022&902&4331
Molecular Weight:	Approximately 25&38&44 kDa

PROPERTIES

Biological Activity	The enzyme activity of this recombinant protein is testing in progress, we cannot offer a guarantee yet.
Appearance	Solution.
Formulation	Supplied as a 0.2 μm filtered solution of PBS, pH 7.4.
Endotoxin Level	<1 EU/μg, determined by LAL method.
Reconsititution	N/A.
Storage & Stability	Stored at -80°C for 1 year. It is stable at -20°C for 3 months after opening. It is recommended to freeze aliquots at -80°C for extended storage. Avoid repeated freeze-thaw cycles.
Shipping	Shipping with dry ice.

DESCRIPTION

Background	CDK7, in conjunction with its regulatory partners CCNH and MNAT1, functions as a serine/threonine kinase with pivotal roles in cell cycle regulation and RNA polymerase II-mediated transcription. As a catalytic subunit of the CDK-activating kinase (CAK) complex, CDK7 plays a critical role in the activation and formation of CDK1/cyclin-B during the G2-M transition and CDK2/cyclins during the G1-S transition. Its phosphorylation targets include SPT5/SUPT5H, SF1/NR5A1, POLR2A, p53/TP53, CDK1, CDK2, CDK4, CDK6, and CDK11B/CDK11. Through threonine phosphorylation, CAK activates cyclin-associated kinases, thereby regulating cell cycle progression. Moreover, CDK7, when complexed with the core-TFIIH basal transcription factor, facilitates RNA polymerase II activation by serine phosphorylation of the C-terminal domain (CTD) of its large subunit (POLR2A). This activation enables RNA polymerase II to escape the promoter and initiate transcript elongation. CDK7's consistent expression and activity throughout the cell cycle contribute to its involvement in DNA-bound peptides-mediated transcription and cellular growth inhibition. Additionally, in response to DNA damage, CDK7 triggers the activation of p53/TP53, forming a feedback loop where p53/TP53 can subsequently inactivate CDK7, potentially leading to cell cycle arrest, transcriptional shutdown, and cellular recovery or apoptosis.

Caution: Product has not been fully validated for medical applications. For research use only.

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