

CDK2 Antibody (C-term) Blocking Peptide

Synthetic peptide Catalog # BP7518b

Specification

CDK2 Antibody (C-term) Blocking Peptide -Product Information

Primary Accession P24941

CDK2 Antibody (C-term) Blocking Peptide -Additional Information

Gene ID 1017

Other Names

Cyclin-dependent kinase 2, Cell division protein kinase 2, p33 protein kinase, CDK2, CDKN2

Target/Specificity

The synthetic peptide sequence used to generate the antibody AP7518b was selected from the C-term region of human CDK2 . 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.

CDK2 Antibody (C-term) Blocking Peptide -Protein Information

Name CDK2

CDK2 Antibody (C-term) Blocking Peptide -Background

CDK2 is a member of the Ser/Thr protein kinase family. This protein kinase is highly similar to the gene products of S. cerevisiae cdc28, and S. pombe cdc2. It is a catalytic subunit of the cyclin-dependent protein kinase complex, whose activity is restricted to the G1-S phase, and essential for cell cycle G1/S phase transition. This protein associates with and regulated by the regulatory subunits of the complex including cyclin A or E, CDK inhibitor p21Cip1 (CDKN1A) and p27Kip1 (CDKN1B). Its activity is also regulated by its protein phosphorylation.

CDK2 Antibody (C-term) Blocking Peptide -References

Moshinsky, D.J., et al., Biochem. Biophys. Res. Commun. 310(3):1026-1031 (2003).Chow, J.P., et al., J. Biol. Chem. 278(42):40815-40828 (2003).O'Nions, J., et al., Oncogene 22(46):7181-7191 (2003).Yun, J., et al., J. Biol. Chem. 278(38):36966-36972 (2003).Izumiya, Y., et al., J. Virol. 77(17):9652-9661 (2003).



Synonyms CDKN2

Function

Serine/threonine-protein kinase involved in the control of the cell cycle; essential for meiosis, but dispensable for mitosis. Phosphorylates CTNNB1, USP37, p53/TP53, NPM1, CDK7, RB1, BRCA2, MYC, NPAT, EZH2. Triggers duplication of centrosomes and DNA. Acts at the G1-S transition to promote the E2F transcriptional program and the initiation of DNA synthesis, and modulates G2 progression; controls the timing of entry into mitosis/meiosis by controlling the subsequent activation of cyclin B/CDK1 by phosphorylation, and coordinates the activation of cyclin B/CDK1 at the centrosome and in the nucleus. Crucial role in orchestrating a fine balance between cellular proliferation, cell death, and DNA repair in human embryonic stem cells (hESCs). Activity of CDK2 is maximal during S phase and G2; activated by interaction with cyclin E during the early stages of DNA synthesis to permit G1-S transition, and subsequently activated by cyclin A2 (cyclin A1 in germ cells) during the late stages of DNA replication to drive the transition from S phase to mitosis, the G2 phase. EZH2 phosphorylation promotes H3K27me3 maintenance and epigenetic gene silencing. Phosphorylates CABLES1 (By similarity). Cyclin E/CDK2 prevents oxidative stress-mediated Ras-induced senescence by phosphorylating MYC. Involved in G1-S phase DNA damage checkpoint that prevents cells with damaged DNA from initiating mitosis; regulates homologous recombination-dependent repair by phosphorylating BRCA2, this phosphorylation is low in S phase when recombination is active, but increases as cells progress towards mitosis. In response to DNA damage, double-strand break repair by homologous recombination a reduction of CDK2-mediated BRCA2 phosphorylation. Phosphorylation of RB1 disturbs its interaction with E2F1. NPM1 phosphorylation by cyclin E/CDK2 promotes its dissociates from unduplicated centrosomes, thus initiating centrosome duplication. Cyclin E/CDK2-mediated phosphorylation of NPAT at G1-S transition and until prophase stimulates the NPAT-mediated activation of histone gene transcription during S phase. Required for



vitamin D-mediated growth inhibition by being itself inactivated. Involved in the nitric oxide- (NO) mediated signaling in a nitrosylation/activation-dependent manner. USP37 is activated by phosphorylation and thus triggers G1-S transition. CTNNB1 phosphorylation regulates insulin internalization. Phosphorylates FOXP3 and negatively regulates its transcriptional activity and protein stability (By similarity). Phosphorylates CDK2AP2 (PubMed: 12944431). Phosphorylates ERCC6 which is essential for its chromatin remodeling activity at DNA double-strand breaks (PubMed: 29203878).

Cellular Location

Cytoplasm, cytoskeleton, microtubule organizing center, centrosome. Nucleus, Cajal body. Cytoplasm. Endosome Note=Localized at the centrosomes in late G2 phase after separation of the centrosomes but before the start of prophase. Nuclear-cytoplasmic trafficking is mediated during the inhibition by 1,25-(OH)(2)D(3)

CDK2 Antibody (C-term) Blocking Peptide -Protocols

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

Blocking Peptides