

Mouse Cdk2 Antibody (C-term)
Affinity Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP16161b**Specification**

Mouse Cdk2 Antibody (C-term) - Product Information

Application	WB,E
Primary Accession	P97377
Other Accession	NP_904326.1 , NP_058036.1
Reactivity	Mouse
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Calculated MW	38978
Antigen Region	227-254

Mouse Cdk2 Antibody (C-term) - Additional Information**Gene ID** 12566**Other Names**

Cyclin-dependent kinase 2, Cell division protein kinase 2, Cdk2, Cdkn2

Target/Specificity

This Mouse Cdk2 antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 227-254 amino acids from the C-terminal region of mouse Cdk2.

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

Mouse Cdk2 Antibody (C-term) is for research use only and not for use in diagnostic or therapeutic procedures.

Mouse Cdk2 Antibody (C-term) - Protein Information**Name** Cdk2**Synonyms** Cdkn2

Function Serine/threonine-protein kinase involved in the control of the cell cycle; essential for meiosis, but dispensable for mitosis (PubMed:[11733001](#), PubMed:[12923533](#), PubMed:[14561402](#), PubMed:[17942597](#), PubMed:[23853094](#)). Phosphorylates CABLES1, CTNNB1, CDK2AP2, ERCC6, NBN, USP37, p53/TP53, NPM1, CDK7, RB1, BRCA2, MYC, NPAT, EZH2 (PubMed:[11733001](#), PubMed:[23853094](#)). Triggers duplication of centrosomes and DNA (By similarity). 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 (By similarity). Crucial role in orchestrating a fine balance between cellular proliferation, cell death, and DNA repair in embryonic stem cells (ESCs) (By similarity). 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 (By similarity). EZH2 phosphorylation promotes H3K27me3 maintenance and epigenetic gene silencing (By similarity). Cyclin E/CDK2 prevents oxidative stress-mediated Ras-induced senescence by phosphorylating MYC (By similarity). 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 (By similarity). In response to DNA damage, double-strand break repair by homologous recombination a reduction of CDK2-mediated BRCA2 phosphorylation (By similarity). Involved in regulation of telomere repair by mediating phosphorylation of NBN (By similarity). Phosphorylation of RB1 disturbs its interaction with E2F1 (By similarity). NPM1 phosphorylation by cyclin E/CDK2 promotes its dissociation from unduplicated centrosomes, thus initiating centrosome duplication (By similarity). 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 (By similarity). Required for vitamin D-mediated growth inhibition by being itself inactivated (By similarity). Involved in the nitric oxide- (NO) mediated signaling in a nitrosylation/activation-dependent manner (By similarity). USP37 is activated by phosphorylation and thus triggers G1-S transition (By similarity). CTNNB1 phosphorylation regulates insulin internalization (By similarity). Phosphorylates FOXP3 and negatively regulates its transcriptional activity and protein stability (PubMed:[23853094](#)). Phosphorylates ERCC6 which is essential for its chromatin remodeling activity at DNA double-strand breaks (By similarity). Acts as a regulator of the phosphatidylinositol 3- kinase/protein kinase B signal transduction by mediating phosphorylation of the C-terminus of protein kinase B (PKB/AKT1 and PKB/AKT2), promoting its activation (PubMed:[24670654](#)).

Cellular Location

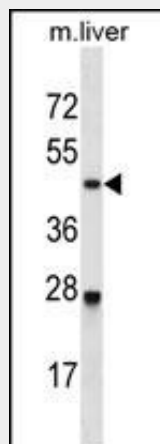
Cytoplasm, cytoskeleton, microtubule organizing center, centrosome. Nucleus, Cajal body
Cytoplasm. Endosome. Note=Localized at the centrosomes in late G2 phase after separation of the centrosomes but before the start of prophase. Nuclear-cytoplasmic trafficking is mediated during the inhibition by 1,25-(OH)(2)D(3)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

Mouse Cdk2 Antibody (C-term) - Protocols

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

- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

Mouse Cdk2 Antibody (C-term) - Images



Mouse Cdk2 Antibody (C-term) (Cat. #AP16161b) western blot analysis in mouse liver tissue lysates (35ug/lane). This demonstrates the Cdk2 antibody detected the Cdk2 protein (arrow).

Mouse Cdk2 Antibody (C-term) - Background

Cdk2 is involved in the control of the cell cycle. Interacts with cyclins A, B1, B3, D, or E. Activity of CDK2 is maximal during S phase and G2 (By similarity).

Mouse Cdk2 Antibody (C-term) - References

- Puyol, M., et al. Cancer Cell 18(1):63-73(2010)
- Hodeify, R., et al. Am. J. Physiol. Renal Physiol. 299 (1), F112-F120 (2010) :
- Risley, M.D., et al. Dev. Biol. 342(2):146-156(2010)
- Copeland, N.A., et al. J. Cell. Sci. 123 (PT 7), 1108-1115 (2010) :
- Koledova, Z., et al. Stem Cells 28(3):450-461(2010)