

#### Cdk2/Cdc2 (phospho Thr160) Polyclonal Antibody Catalog # AP67524

### Specification

# Cdk2/Cdc2 (phospho Thr160) Polyclonal Antibody - Product Information

Application Primary Accession Reactivity Host Clonality WB, IHC-P <u>P24941</u> Human, Mouse, Rat Rabbit Polyclonal

### Cdk2/Cdc2 (phospho Thr160) Polyclonal Antibody - Additional Information

Gene ID 1017

Other Names CDK2; CDKN2; Cyclin-dependent kinase 2; Cell division protein kinase 2; p33 protein kinase

Dilution WB~~Western Blot: 1/500 - 1/2000. Immunohistochemistry: 1/100 - 1/300. ELISA: 1/20000. Not yet tested in other applications. IHC-P~~N/A

**Format** Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.09% (W/V) sodium azide.

**Storage Conditions** -20°C

## Cdk2/Cdc2 (phospho Thr160) Polyclonal Antibody - 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:<a href="http://www.uniprot.org/citations/10499802" target="\_blank">10499802</a>, PubMed:<a href="http://www.uniprot.org/citations/10884347" target="\_blank">10499802</a>, PubMed:<a href="http://www.uniprot.org/citations/10995386" target="\_blank">10884347</a>, PubMed:<a href="http://www.uniprot.org/citations/10995386" target="\_blank">10995386</a>, PubMed:<a href="http://www.uniprot.org/citations/10995387" target="\_blank">10995386</a>, PubMed:<a href="http://www.uniprot.org/citations/10995387" target="\_blank">10995387</a>, PubMed:<a href="http://www.uniprot.org/citations/11051553" target="\_blank">10995387</a>, PubMed:<a href="http://www.uniprot.org/citations/11051553" target="\_blank">11051553</a>, PubMed:<a href="http://www.uniprot.org/citations/11113184" target="\_blank">1113184</a>, PubMed:<a href="http://www.uniprot.org/citations/12944431" target="\_blank">12944431</a>, PubMed:<a href="http://www.uniprot.org/citations/15800615" target="\_blank">15800615</a>, PubMed:<a href="http://www.uniprot.org/citations/15800615" target="\_blank">15800615</a>, PubMed:<a href="http://www.uniprot.org/citations/17495531" target="\_blank">15800615</a>, PubMed:<a href="http://www.uniprot.org/citations/17495531" target="\_blank">15800615</a>, PubMed:<a href="http://www.uniprot.org/citations/17495531" target="\_blank">15800615</a>, PubMed:<a href="http://www.uniprot.org/citations/17495531" target="\_blank">17495531</a>, PubMed:<a href="http://www.uniprot.org/citations/19966300" target="\_blank">19966300</a>, PubMed:<a href="http://www.uniprot.org/citations/19966300" target="\_blank">19966300</a>, PubMed:<a href="http://www.uniprot.org/citations/19966300" target="\_blank">19966300</a>, PubMed:<a href="http://www.uniprot.org/citations/19966300"</a>



target=" blank">20935635</a>, PubMed:<a href="http://www.uniprot.org/citations/21262353" target="blank">21262353</a>, PubMed:<a href="http://www.uniprot.org/citations/21596315" target=" blank">21596315</a>, PubMed:<a href="http://www.uniprot.org/citations/28216226" target="\_blank">28216226</a>, PubMed:<a href="http://www.uniprot.org/citations/28666995" target=" blank">28666995</a>). Phosphorylates CABLES1, CTNNB1, CDK2AP2, ERCC6, NBN, USP37, p53/TP53, NPM1, CDK7, RB1, BRCA2, MYC, NPAT, EZH2 (PubMed:<a href="http://www.uniprot.org/citations/10499802" target=" blank">10499802</a>, PubMed:<a href="http://www.uniprot.org/citations/10995386" target=" blank">10995386</a>, PubMed:<a href="http://www.uniprot.org/citations/10995387" target=" blank">10995387</a>, PubMed:<a href="http://www.uniprot.org/citations/11051553" target="\_blank">11051553</a>, PubMed:<a href="http://www.uniprot.org/citations/11113184" target="\_blank">11113184</a>, PubMed:<a href="http://www.uniprot.org/citations/12944431" target=" blank">12944431</a>, PubMed:<a href="http://www.uniprot.org/citations/15800615" target=" blank">15800615</a>, PubMed:<a href="http://www.uniprot.org/citations/19966300" target=" blank">19966300</a>, PubMed:<a href="http://www.uniprot.org/citations/20935635" target=" blank">20935635</a>, PubMed:<a href="http://www.uniprot.org/citations/21262353" target=" blank">21262353</a>, PubMed:<a href="http://www.uniprot.org/citations/21596315" target=" blank">21596315</a>, PubMed:<a href="http://www.uniprot.org/citations/28216226" target=" blank">28216226</a>). Triggers duplication of centrosomes and DNA (PubMed:<a href="http://www.uniprot.org/citations/11051553" target=" blank">11051553</a>). 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 (PubMed:<a href="http://www.uniprot.org/citations/18372919" target="\_blank">18372919</a>, PubMed:<a href="http://www.uniprot.org/citations/19238148" target=" blank">19238148</a>, PubMed:<a href="http://www.uniprot.org/citations/19561645" target=" blank">19561645</a>). Crucial role in orchestrating a fine balance between cellular proliferation, cell death, and DNA repair in embryonic stem cells (ESCs) (PubMed:<a href="http://www.uniprot.org/citations/18372919" target=" blank">18372919</a>, PubMed:<a href="http://www.uniprot.org/citations/19238148" target=" blank">19238148</a>, PubMed:<a href="http://www.uniprot.org/citations/19561645" target=" blank">19561645</a>). 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 (PubMed:<a href="http://www.uniprot.org/citations/18372919" target=" blank">18372919</a>, PubMed:<a href="http://www.uniprot.org/citations/19238148" target=" blank">19238148</a>, PubMed:<a href="http://www.uniprot.org/citations/19561645" target=" blank">19561645</a>). EZH2 phosphorylation promotes H3K27me3 maintenance and epigenetic gene silencing (PubMed:<a href="http://www.uniprot.org/citations/20935635" target="\_blank">20935635</a>). Cyclin E/CDK2 prevents oxidative stress- mediated Ras-induced senescence by phosphorylating MYC (PubMed:<a href="http://www.uniprot.org/citations/19966300" target=" blank">19966300</a>). 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 (PubMed:<a href="http://www.uniprot.org/citations/15800615" target=" blank">15800615</a>, PubMed:<a href="http://www.uniprot.org/citations/20195506" target=" blank">20195506</a>, PubMed:<a href="http://www.uniprot.org/citations/21319273" target=" blank">21319273</a>). In response to DNA damage, double- strand break repair by homologous recombination a reduction of CDK2- mediated BRCA2 phosphorylation (PubMed:<a href="http://www.uniprot.org/citations/15800615" target=" blank">15800615</a>). Involved in regulation of telomere repair by mediating phosphorylation of NBN (PubMed:<a href="http://www.uniprot.org/citations/28216226" target=" blank">28216226</a>). Phosphorylation of RB1 disturbs its interaction with E2F1 (PubMed:<a href="http://www.uniprot.org/citations/10499802" target=" blank">10499802</a>). NPM1 phosphorylation by cyclin E/CDK2 promotes its dissociates from unduplicated centrosomes, thus initiating centrosome duplication (PubMed:<a href="http://www.uniprot.org/citations/11051553"



target=" blank">11051553</a>). 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 (PubMed:<a href="http://www.uniprot.org/citations/10995386" target=" blank">10995386</a>, PubMed:<a href="http://www.uniprot.org/citations/10995387" target=" blank">10995387</a>). Required for vitamin D-mediated growth inhibition by being itself inactivated (PubMed: <a href="http://www.uniprot.org/citations/20147522" target=" blank">20147522</a>). Involved in the nitric oxide- (NO) mediated signaling in a nitrosylation/activation-dependent manner (PubMed:<a href="http://www.uniprot.org/citations/20079829" target="\_blank">20079829</a>). USP37 is activated by phosphorylation and thus triggers G1-S transition (PubMed:<a href="http://www.uniprot.org/citations/21596315" target=" blank">21596315</a>). CTNNB1 phosphorylation regulates insulin internalization (PubMed:<a href="http://www.uniprot.org/citations/21262353" target=" blank">21262353</a>). Phosphorylates FOXP3 and negatively regulates its transcriptional activity and protein stability (By similarity). Phosphorylates ERCC6 which is essential for its chromatin remodeling activity at DNA double-strand breaks (PubMed:<a href="http://www.uniprot.org/citations/29203878" target=" blank">29203878</a>). 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: <a href="http://www.uniprot.org/citations/24670654" target=" blank">24670654</a>).

#### **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)

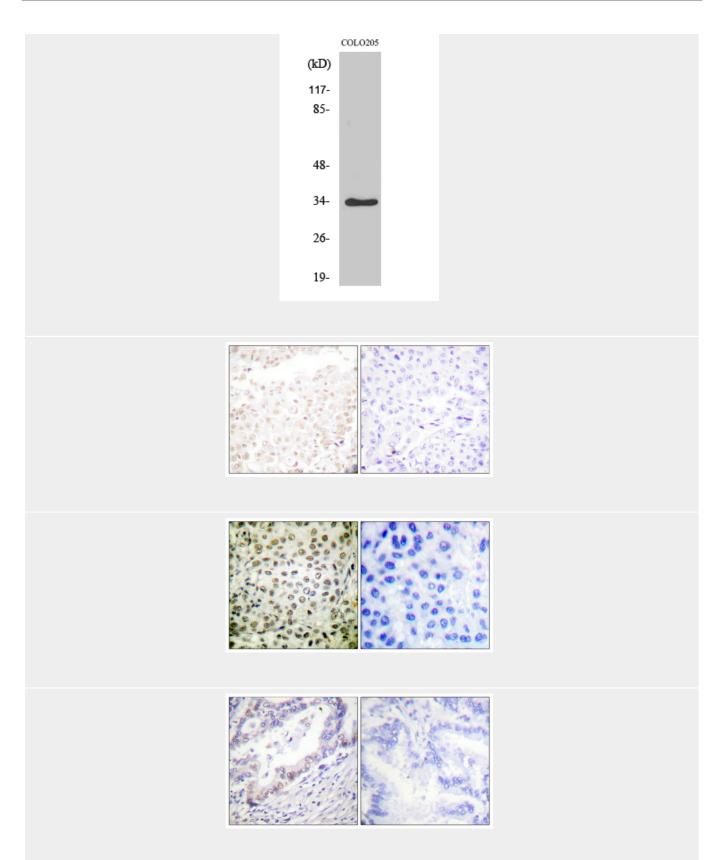
### Cdk2/Cdc2 (phospho Thr160) Polyclonal Antibody - Protocols

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>

### Cdk2/Cdc2 (phospho Thr160) Polyclonal Antibody - Images





# Cdk2/Cdc2 (phospho Thr160) Polyclonal Antibody - Background

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).