

## **CHK2 Antibody**

Purified Mouse Monoclonal Antibody Catalog # A01185a

## **Specification**

## **CHK2 Antibody - Product Information**

Application
Primary Accession
Reactivity
Host
Clonality
Isotype

Calculated MW **Description** 

CHK2: CHK2 checkpoint homolog (S. pombe). In response to DNA damage and replication blocks, cell cycle progression is halted through the control of critical cell cycle regulators. The protein encoded by this gene is a cell cycle checkpoint regulator and putative tumor suppressor. It contains a forkhead-associated protein interaction domain essential for activation in response to DNA damage and is rapidly phosphorylated in response to replication blocks and DNA damage. When activated, the encoded protein is known to inhibit CDC25C phosphatase, preventing entry into mitosis, and has been shown to stabilize the tumor suppressor protein p53, leading to cell cycle arrest in G1. In addition, this protein interacts with and phosphorylates BRCA1, allowing BRCA1 to restore survival after DNA damage. Mutations in this gene have been linked with Li-Fraumeni syndrome, a highly penetrant familial cancer phenotype usually associated with inherited mutations in TP53. Also, mutations in this gene are thought to confer a predisposition to sarcomas, breast cancer, and brain tumors. This nuclear protein is a member of the CDS1 subfamily of serine/threonine protein kinases. Three transcript variants encoding different isoforms have been found for this gene.

WB, IHC, ICC, E

096017

Human

Mouse

laG2b

**Monoclonal** 

61kDa KDa

## **Immunogen**

Purified recombinant fragment of human CHK2 (aa481-531) expressed in E. Coli. <br/> <br/>

## **Formulation**

Ascitic fluid containing 0.03% sodium azide.

# **CHK2 Antibody - Additional Information**

**Gene ID 11200** 

## **Other Names**

Serine/threonine-protein kinase Chk2, 2.7.11.1, CHK2 checkpoint homolog, Cds1 homolog, Hucds1, hCds1, Checkpoint kinase 2, CHEK2, CDS1, CHK2, RAD53

## Dilution

WB~~1/500 - 1/2000 IHC~~1/200 - 1/1000 ICC~~1:200~~1000 E~~N/A



### Storage

Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.

#### **Precautions**

CHK2 Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

## **CHK2 Antibody - Protein Information**

Name CHEK2 (HGNC:16627)

Synonyms CDS1, CHK2, RAD53

#### **Function**

Serine/threonine-protein kinase which is required for checkpoint-mediated cell cycle arrest, activation of DNA repair and apoptosis in response to the presence of DNA double-strand breaks. May also negatively regulate cell cycle progression during unperturbed cell cycles. Following activation, phosphorylates numerous effectors preferentially at the consensus sequence [L-X-R-X-X-S/T] (PubMed:<a href="http://www.uniprot.org/citations/37943659" target=" blank">37943659</a>). Regulates cell cycle checkpoint arrest through phosphorylation of CDC25A, CDC25B and CDC25C, inhibiting their activity. Inhibition of CDC25 phosphatase activity leads to increased inhibitory tyrosine phosphorylation of CDK-cyclin complexes and blocks cell cycle progression. May also phosphorylate NEK6 which is involved in G2/M cell cycle arrest. Regulates DNA repair through phosphorylation of BRCA2, enhancing the association of RAD51 with chromatin which promotes DNA repair by homologous recombination. Also stimulates the transcription of genes involved in DNA repair (including BRCA2) through the phosphorylation and activation of the transcription factor FOXM1. Regulates apoptosis through the phosphorylation of p53/TP53, MDM4 and PML. Phosphorylation of p53/TP53 at 'Ser-20' by CHEK2 may alleviate inhibition by MDM2, leading to accumulation of active p53/TP53. Phosphorylation of MDM4 may also reduce degradation of p53/TP53. Also controls the transcription of pro-apoptotic genes through phosphorylation of the transcription factor E2F1. Tumor suppressor, it may also have a DNA damage-independent function in mitotic spindle assembly by phosphorylating BRCA1. Its absence may be a cause of the chromosomal instability observed in some cancer cells. Promotes the CCAR2-SIRT1 association and is required for CCAR2-mediated SIRT1 inhibition (PubMed:<a href="http://www.uniprot.org/citations/25361978" target="\_blank">25361978</a>). Under oxidative stress, promotes ATG7 ubiquitination by phosphorylating the E3 ubiquitin ligase TRIM32 at 'Ser-55' leading to positive regulation of the autophagosme assembly (PubMed: <a href="http://www.uniprot.org/citations/37943659" target=" blank">37943659</a>).

#### **Cellular Location**

[Isoform 2]: Nucleus. Note=Isoform 10 is present throughout the cell [Isoform 7]: Nucleus. [Isoform 12]: Nucleus.

# **Tissue Location**

High expression is found in testis, spleen, colon and peripheral blood leukocytes. Low expression is found in other tissues

## **CHK2 Antibody - Protocols**

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

- Western Blot
- Blocking Peptides



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

## **CHK2 Antibody - Images**

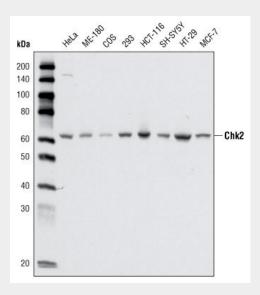


Figure 1: Western blot analysis using CHK2 mouse mAb against cell lysate from various cell types.

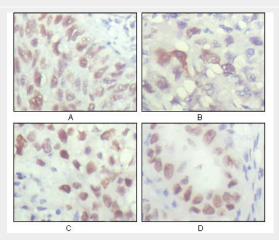


Figure 2: Immunohistochemical analysis of paraffin-embedded human lung carcinoma (A), liver carcinoma (B), breast carcinoma (C) and kiney carcinoma (D), showing nuclear localization with DAB staining using CHK2 mouse mAb.



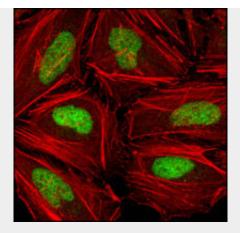


Figure 3: Confocal immunofluorescence analysis of Hela cells using CHK2 mouse mAb (green), showing nuclear localization. Red: Actin filaments have been labeled with DY-554 phalloidin.

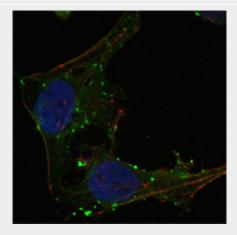


Figure 3: Confocal immunofluorescence analysis of Hela cells using GABPA mouse mAb (green). Red: Actin filaments have been labeled using DY-554 phalloidin. Blue: DRAQ5 fluorescent DNA dye.

# **CHK2 Antibody - References**

1. Int J Cancer. 2007 Dec 15;121(12):2661-7. 2. Nat Rev Cancer. 2007 Dec;7(12):925-36. 3. Carcinogenesis. 2008 Apr;29(4):762-5.