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CHEK2 Antibody (N-term) Blocking Peptide

Synthetic peptide
Catalog # BP4999a

Specification

CHEK2 Antibody (N-term) Blocking Peptide - Product Information

Primary Accession O96017

CHEK2 Antibody (N-term) Blocking Peptide - Additional Information

Gene ID 11200

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

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.

CHEK2 Antibody (N-term) Blocking Peptide - Protein Information

Name CHEK2

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]. 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:25361978).

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

CHEK2 Antibody (N-term) Blocking Peptide - Protocols

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

- Blocking Peptides

CHEK2 Antibody (N-term) Blocking Peptide - Images

CHEK2 Antibody (N-term) Blocking Peptide - Background

CHEK2 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.

CHEK2 Antibody (N-term) Blocking Peptide - References

Yang, X., et al. J. Biol. Chem. 285(5):3030-3034(2010)Varmark, H., et al. Cell Cycle 9(2):312-320(2010)Zhu, H., et al. Neoplasia 11(11):1226-1234(2009)