

Anti-CHK2 (pT68) Antibody

Rabbit polyclonal antibody to CHK2 (pT68) Catalog # AP60553

Product Information

Application WB, IHC **Primary Accession** 096017

Reactivity Human, Mouse, Rat, Pig

HostRabbitClonalityPolyclonalCalculated MW60915

Additional Information

Gene ID 11200

Other Names CDS1; CHK2; RAD53; Serine/threonine-protein kinase Chk2; CHK2 checkpoint

homolog; Cds1 homolog; Hucds1; hCds1; Checkpoint kinase 2

Target/Specificity KLH-conjugated synthetic peptide encompassing a sequence within the

N-term region of human CHK2. The exact sequence is proprietary.

Dilution WB~~WB (1/500 - 1/1000), IHC (1/100 - 1/200) IHC~~WB (1/500 - 1/1000), IHC

(1/100 - 1/200)

Format Liquid in 0.42% Potassium phosphate, 0.87% Sodium chloride, pH 7.3, 30%

glycerol, and 0.09% (W/V) sodium azide.

Storage Store at -20 °C.Stable for 12 months from date of receipt

Protein Information

Name CHEK2 (<u>HGNC:16627</u>)

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-S/T] (PubMed:37943659). 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: <u>25361978</u>). 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:37943659).

Cellular Location

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

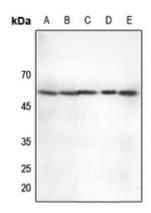
Tissue Location

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

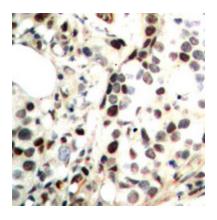
Background

KLH-conjugated synthetic peptide encompassing a sequence within the N-term region of human CHK2. The exact sequence is proprietary.

Images



Western blot analysis of CHK2 (pT68) expression in HEk293T (A), Hela (B), mouse lung (C), rat lung (D), rat kidney (E) whole cell lysates.



Immunohistochemical analysis of CHK2 (pT68) staining in human breast cancer formalin fixed paraffin embedded tissue section. The section was pre-treated using heat mediated antigen retrieval with sodium citrate buffer (pH 6.0). The section was then incubated with the antibody at room temperature and detected using an HRP conjugated compact polymer system. DAB was used as the chromogen. The section was then counterstained with haematoxylin and mounted with DPX.

Please note: All products are 'FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC OR THERAPEUTIC PROCEDURES'.