

## Chk2 Monoclonal Antibody

<b>Catalog No :</b>	YM0154
<b>Reactivity :</b>	Human
<b>Applications :</b>	WB;IHC;IF;ELISA
<b>Target :</b>	Chk2
<b>Fields :</b>	>>Cell cycle;>>p53 signaling pathway;>>Cellular senescence;>>Human T-cell leukemia virus 1 infection
<b>Gene Name :</b>	CHEK2
<b>Protein Name :</b>	Serine/threonine-protein kinase Chk2
<b>Human Gene Id :</b>	11200
<b>Human Swiss Prot No :</b>	O96017
<b>Mouse Swiss Prot No :</b>	Q9Z265
<b>Immunogen :</b>	Purified recombinant fragment of human Chk2 (aa481-531) expressed in E. Coli.
<b>Specificity :</b>	Chk2 Monoclonal Antibody detects endogenous levels of Chk2 protein.
<b>Formulation :</b>	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.
<b>Source :</b>	Monoclonal, Mouse
<b>Dilution :</b>	WB 1:500 - 1:2000. IHC 1:200 - 1:1000. IF 1:200 - 1:1000. ELISA: 1:10000. Not yet tested in other applications.
<b>Purification :</b>	Affinity purification
<b>Storage Stability :</b>	-15°C to -25°C/1 year(Do not lower than -25°C)
<b>Molecularweight :</b>	61kD

**Cell Pathway :** Cell\_Cycle\_G1S;Cell\_Cycle\_G2M\_DNA;p53;

**P 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.

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**Background :** 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 mutati

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**Function :** catalytic activity:ATP + a protein = ADP + a phosphoprotein.,cofactor:Magnesium.,disease:Defects in CHEK2 are associated with Li-Fraumeni syndrome 2 (LFS2) [MIM:609265]; a highly penetrant familial cancer phenotype usually associated with inherited mutations in p53/TP53.,disease:Defects in CHEK2 are found in some patients with osteosarcoma (OSRC) [MIM:259500].,disease:Defects in CHEK2 are found in some patients with prostate cancer (CaP) [MIM:176807].,enzyme regulation:Rapidly phosphorylated on Thr-68 by MLTK in response to DNA damage and to replication block. Kinase activity is also up-regulated by autophosphorylation.,function:Regulates cell cycle checkpoints and apoptosis in response to DNA damage, particularly to DNA double-strand breaks. Inhibits CDC25C phosphatase by phosphorylation on 'Ser-216', preventing the entry into mitosis. May also play a role in meiosis. Regulates the TP53

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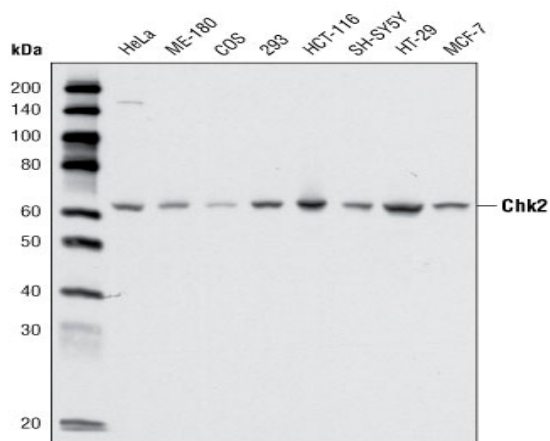
**Subcellular Location :** [Isoform 2]: Nucleus. Isoform 10 is present throughout the cell.; [Isoform 4]: Nucleus.; [Isoform 7]: Nucleus.; [Isoform 9]: Nucleus.; [Isoform 12]: Nucleus.; Nucleus, PML body. Nucleus, nucleoplasm. Recruited into PML bodies together with TP53.

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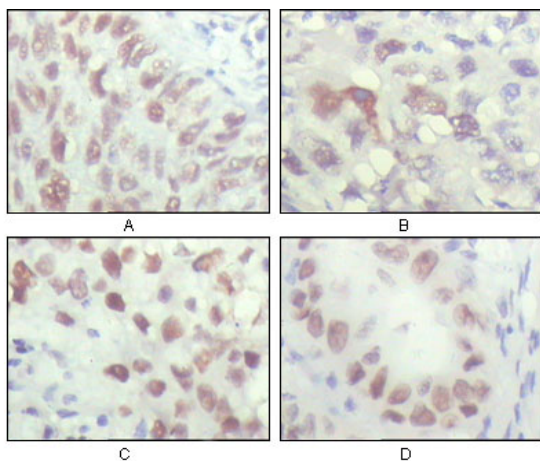
**Expression :** High expression is found in testis, spleen, colon and peripheral blood leukocytes. Low expression is found in other tissues.

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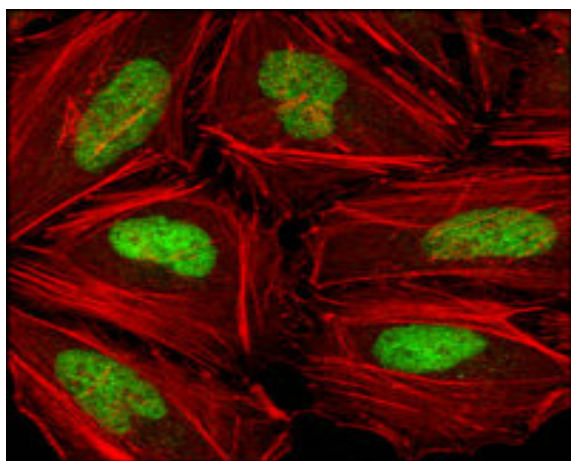
## Products Images



Western Blot analysis using Chk2 Monoclonal Antibody against cell lysate from various cell types.



Immunohistochemistry analysis of paraffin-embedded human lung carcinoma (A), liver carcinoma (B), breast carcinoma (C) and kidney carcinoma (D), showing nuclear localization with DAB staining using Chk2 Monoclonal Antibody.



Confocal immunofluorescence analysis of HeLa cells using Chk2 Monoclonal Antibody (green), showing nuclear localization. Red: Actin filaments have been labeled with DY-554 phalloidin.