CELL APPLICATIONS, INC.

Cdk2 Antibody

Cat. No. CG1081

Applications:	**WB, IF**
Detected MW:	**33 kDa**
Species & Reactivity:	**Human, Mouse, Rat**
Isotype:	**Rabbit IgG**

BACKGROUND

Cdks (cyclin-dependent kinases) are heteromeric serine/threonine kinases that control progression through the cell cycle in concert with their regulatory subunits, the cyclins. Although there are 12 different cdk genes, only 5 have been shown to directly drive the cell cycle (Cdk1, -2, -3, -4, and -6).[1] Following extracellular mitogenic stimuli, cyclin D gene expression is upregulated. Cdk4 forms a complex with cyclin D and phosphorylates Rb protein, leading to liberation of the transcription factor E2F. E2F induces transcription of genes including cyclins A and E, DNA polymerase and thymidine kinase. Cdk4-cyclin E complexes form and initiate G1/S transition. Subsequently, Cdk1-cyclin B complexes form and induce G2/M phase transition. Cdk1-cyclin B activation induces the breakdown of the nuclear envelope and the initiation of mitosis.[2] Cdks are constitutively expressed and are regulated by several kinases and phosphastases, including Wee1, CDK-activating kinase and Cdc25 phosphatase. In addition, cyclin expression is induced by molecular signals at specific points of the cell cycle, leading to activation of Cdks.[3] Tight control of Cdks is essential as misregulation can induce unscheduled proliferation, and genomic and chromosomal instability. Cdk4 has been shown to be mutated in some types of cancer, whilst a chromosomal rearrangement can lead to Cdk6 overexpression in lymphoma, leukemia and melanoma. Cdks are currently under investigation as potential targets for antineoplastic therapy, but as Cdks are essential for driving each cell cycle phase, therapeutic strategies that block Cdk activity are unlikely to selectively target tumor cells.[4]

Cdk2 is a member of the Ser/Thr protein kinase family. It is a catalytic subunit of the cyclin-dependent protein kinase complex, whose activity is restricted to the G1-S phase, and essential for cell cycle G1/S phase transition. This protein associates with and regulated by the regulatory subunits of the complex including cyclin A or E, CDK inhibitor p21Cip1 (CDKN1A) and p27Kip1 (CDKN1B). Its activity is also regulated by its protein phosphorylation.[5] Two alternatively spliced variants and multiple transcription initiation sites of this gene have been reported.

References:

1. Pestell, R.G. et al: Endocrine Rev. 20:501-34, 1999
2. Nurse, P.: Cell 100:71-8, 2000
3. Morgan, D. O. : Ann. Rev. Cell Develop. Biol. 13:261-91, 1997
4. McDonald, E.R. 3rd. & El-Deiry, W.S.: Int. J. Oncol. 16:871-86, 2000
5. Poon, R.Y.C. & Hunter, T.: Science 270:90-3, 1995

TECHNICAL INFORMATION

Source:
Cdk2 antibody is a rabbit antibody raised against a short peptide from carboxyl-terminal sequence of human Cdk2.

Specificity and Sensitivity:
This antibody detects endogenous Cdk2 proteins without cross-reactivity with other family members.

Storage Buffer: Rabbit IgG in phosphate buffered saline (without Mg2+ and Ca2+), pH 7.4, 150mM NaCl, 0.02% sodium azide and 50% glycerol.

Storage:
Store at -20°C for at least one year. Store at 4°C for frequent use. Avoid repeated freeze-thaw cycles.

APPLICATIONS

Application:	*Dilution:
WB	1:500-1:1,000
IP	n/d
IHC	n/d
ICC	n/d
FACS	n/d
IF	1:100-1:500

*****Optimal dilutions must be determined by end user.*

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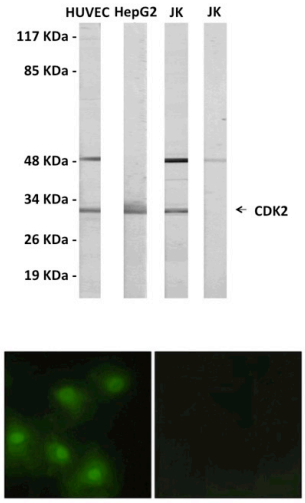
QUALITY CONTROL DATA



Top: Immunoblotting analysis of extracts from HuvEc/HepG2/Jurkat cells, using Anti-CDK2, C-Terminal antibody. The lane on the left was treated with the Anti-CDK2, C-Terminal antibody. The lane on the right (negative control) was treated with both Anti-CDK2, C-Terminal antibody and the synthesized immunogen peptide.
Bottom: Immunofluorescence of HeLa cells using Anti-CDK2, C-Terminal antibody. Cells on the left were treated with the Anti-CDK2, C-Terminal antibody. Cells on the right (negative control) were treated with both Anti-CDK2, C-Terminal antibody and the synthesized immunogen peptide.