

p27 KIP 1 (PT0324R) PT® Rabbit mAb

CatalogNo: YM8191 Recombinant R

Key Features

Host Species

Rabbit

MW
• 27kD (Calculated)
27kD (Observed)

Reactivity

· Human, Mouse, Rat,

Isotype

IgG,Kappa

Applications

• WB,IHC,IF,IP,ELISA

Recommended Dilution Ratios

IHC 1:200-1:1000 WB 1:1000-1:5000 IF 1:200-1:1000

ELISA 1:5000-1:20000

IP 1:50-1:200,

Storage

Storage* -15°C to -25°C/1 year(Do not lower than -25°C)

Formulation PBS, 50% glycerol, 0.05% Proclin 300, 0.05%BSA

Basic Information

Clonality Monoclonal

Clone Number PT0324R

Immunogen Information

Specificity Endogenous

| Target Information

Gene name

CDKN1B

Protein Name

Cyclin-dependent kinase inhibitor 1B

Organism	Gene ID	UniProt ID	
Human	<u>1027</u> ;	<u>P46527;</u>	
Mouse		P46414;	

Cellular Localization

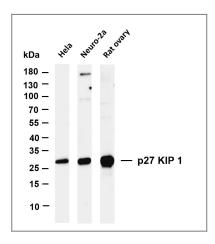
Nucleus

Tissue specificity Expressed in kidney (at protein level) (PubMed:15509543). Expressed in all tissues tested (PubMed:8033212). Highest levels in skeletal muscle, lowest in liver and kidney (PubMed:8033212).

Function

Disease: Defects in CDKN1B are the cause of multiple endocrine neoplasia type 4 (MEN4) [MIM:610755]. Multiple endocrine neoplasia (MEN) syndromes are inherited cancer syndromes of the thyroid. MEN4 is a MEN-like syndrome with a phenotypic overlap of both MEN1 and MEN2., Domain: A peptide sequence containing only AA 28-79 retains substantial Kip1 cyclin A/CDK2 inhibitory activity., Function: Important regulator of cell cycle progrssion. Involved in G1 arrest. Potent inhibitor of cyclin E- and cyclin A-CDK2 complexes. Positive regulator of cyclin D-dependent kinases such as CDK4. Regulated by phosphorylation and degradation events.,induction:Maximal levels in quiescence cells and early G(1). Levels decrease after mitogen stimulation as cells progress toward Sphase., miscellaneous: Decreased levels of p27Kip1, mainly due to proteosomal degradation, are found in various epithelial tumors originating from lung, breast, colon, ovary, esophagus, thyroid and prostate., PTM: Phosphorylated; phosphorylation occurs on serine, threonine and tyrosine residues. Phosphorylation on Ser-10 is the major site of phosphorylation in resting cells, takes place at the G(0)-G(1) phase and leads to protein stability. Phosphorylation on other sites is greatly enhanced by mitogens, growth factors, cMYC and in certain cancer cell lines. The phosphorylated form found in the cytoplasm is inactivate. Phosphorylation on Thr-198 is required for interaction with 14-3-3 proteins. Phosphorylation on Thr-187, by CDK2 leads to protein ubiquitination and proteasomal degradation. Tyrosine phosphorylation promotes this process. Phosphorylation by PKB/AKT1 can be suppressed by LY294002, an inhibitor of the catalytic subunit of PI3K. Phosphorylation on Tyr-88 and Tyr-89 has no effect on binding CDK2, but is required for binding CDK4. Dephosphorylated on tyrosine residues by G-CSF.,PTM:Ubiquitinated; in the cytoplasm by the KPC1/KPC2 complex and, in the nucleus, by SCF/SKP2. The latter requires prior phosphorylation on Thr-187., similarity: Belongs to the CDI family., subcellular location: Nuclear and cytoplasmic in quiescent cells. AKT- or RSK-mediated phosphorylation on Thr-198, binds 14-3-3, translocates to the cytoplasm and promotes cell cycle progression. Mitogen-activated UHMK1 phosphorylation on Ser-10 also results in translocation to the cytoplasm and cell cycle progression. Phosphorylation on Ser-10 facilitates nuclear export. Translocates to the nucleus on phosphorylation of Tyr-88 and Tyr-89., subunit: Interacts with NUP50; the interaction leads to nuclear import and degradation of phosphorylated p27kip1. Interacts with COPS5, subunit of the COP9 signalosome complex; the interaction leads to p27KIP degradation. Interacts with SPDYA in the SPDYA/CDK2/p27kip1 complex. Interacts (Thr-198 phosphorylated-form) with 14-3-3 proteins, binds strongly YWHAQ, weakly YWHAE and YWHAH, but not YWHAB nor YWHAZ; the interaction with YWHAQ results in translocation to the cytoplasm. Interacts with AKT1, LYN and UHMK1; the interactions lead to cytoplasmic mislocation, phosphorylation of p27kip1 and inhibition of cell cycle arrest. Interacts (unphosphorylated form) with CDK2. Interacts (phosphorylated on Tyr-88 and Tyr-89) with CDK4; the interaction induces nuclear translocation. Interacts with GRB2., tissue specificity: Expressed in all tissues tested. Highest levels in skeletal muscle, lowest in liver and kidney.,

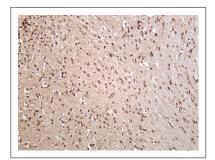
Validation Data



Various whole cell lysates were separated by 4-20% SDS-PAGE, and the membrane was blotted with anti-p27 KIP 1 antibody. The HRP-conjugated Goat anti-Rabbit IgG(H + L) antibody was used to detect the antibody. Lane 1: Hela Lane 2: Rat ovary Predicted band size: 27kDa Observed band size: 27kDa



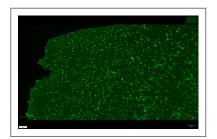
Mouse brain was stained with anti-p27 KIP 1 rabbit antibody



Rat brain was stained with anti-p27 KIP 1 rabbit antibody



Human brain was stained with anti-p27 KIP 1 rabbit antibody



mouse brain was stained with anti-p27 KIP 1 rabbit antibody



Immunofluorescence analysis of HEK293. Picture A: p27 KIP 1 antibody (red). Picture B: DAPI (blue). Picture C: Merge of A+B

| Contact information

Orders: order.cn@immunoway.com Support: support.cn@immunoway.com

Telephone: 400-8787-807(China)

Website: http://www.immunoway.com.cn

Address: 2200 Ringwood Ave San Jose, CA 95131 USA



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