

p27 Mouse mAb

CatalogNo: YM0496 Orthogonal Validated 

Key Features

Host Species

- Mouse

Reactivity

- Human

Applications

- WB,ELISA

MW

- 22kD (Calculated)

Storage

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

Formulation Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.

Recommended Dilution Ratios

WB 1:500-1:2000

ELISA 1:10000

Not yet tested in other applications.

Basic Information

Clonality Monoclonal

Clone Number 9F1

Immunogen Information

Immunogen Purified recombinant fragment of human p27 expressed in E. Coli.

Specificity p27 Monoclonal Antibody detects endogenous levels of p27 protein.

| Target Information

Gene name CDKN1B KIP1

Protein Name Cyclin-dependent kinase inhibitor 1B

Organism	Gene ID	UniProt ID
Human	1027 ;	P46527 ;

**Cellular
Localization**

Nucleus. Cytoplasm. Endosome . 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. Colocalizes at the endosome with SNX6; this leads to lysosomal degradation (By similarity). .

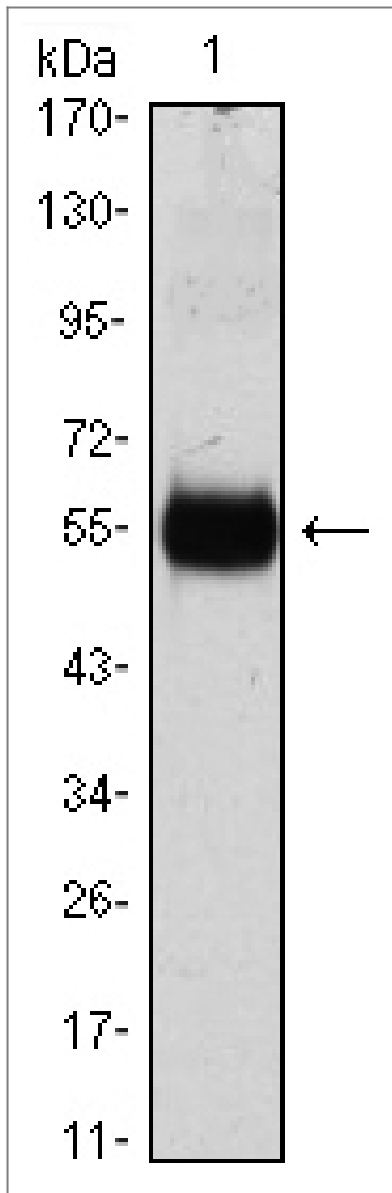
Tissue specificity

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 progression. 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 S-phase. Miscellaneous: Decreased levels of p27Kip1, mainly due to proteasomal 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 inactivated. 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



Western Blot analysis using p27 Monoclonal Antibody against CDKN1B-hlgGfc transfected HEK293 cell lysate.

Contact information

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p27 Mouse mAb

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