



p27 KIP 1 Rabbit mAb

Catalog No	YP-rAb-18016
Isotype	IgG
Reactivity	Human,Mouse,Rat,Bovine
Applications	WB,IHC,IF,IP,ELISA
Gene Name	CDKN1B KIP1
Protein Name	Cyclin-dependent kinase inhibitor 1B
Purification Process	Protein A
Specificity	Endogenous
Formulation	PBS, 50% glycerol, 0.05% Proclin 300, 0.05%BSA
Source	Monoclonal, Rabbit,IgG
Dilution	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, Note: For IHC, we suggest antigen retrieval with TE buffer pH 9.0
Concentration	0.5 mg/ml
Purity	≥90%
Storage Stability	-15° C to -25° C/1 year(Do not lower than -25° C)
Synonyms	CDKN1B ; KIP1 ; Cyclin-dependent kinase inhibitor 1B ; Cyclin-dependent kinase inhibitor p27 ; p27Kip1
Observed Band	27kD
Calculated Molecular Weight	27kD
Cell Pathway	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 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





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.,

Background

This gene encodes a cyclin-dependent kinase inhibitor, which shares a limited similarity with CDK inhibitor CDKN1A/p21. The encoded protein binds to and prevents the activation of cyclin E-CDK2 or cyclin D-CDK4 complexes, and thus controls the cell cycle progression at G1. The degradation of this protein, which is triggered by its CDK dependent phosphorylation and subsequent ubiquitination by SCF complexes, is required for the cellular transition from quiescence to the proliferative state. Mutations in this gene are associated with multiple endocrine neoplasia type IV (MEN4). [provided by RefSeq, Apr 2014],

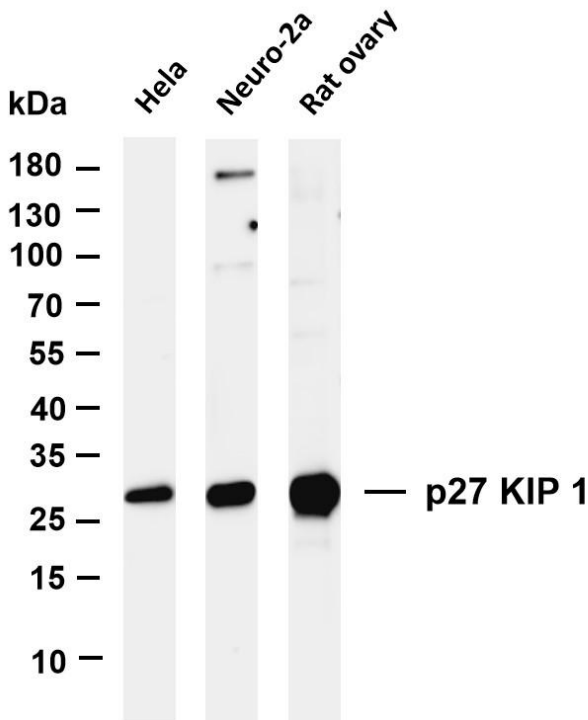
matters needing attention

Avoid repeated freezing and thawing!

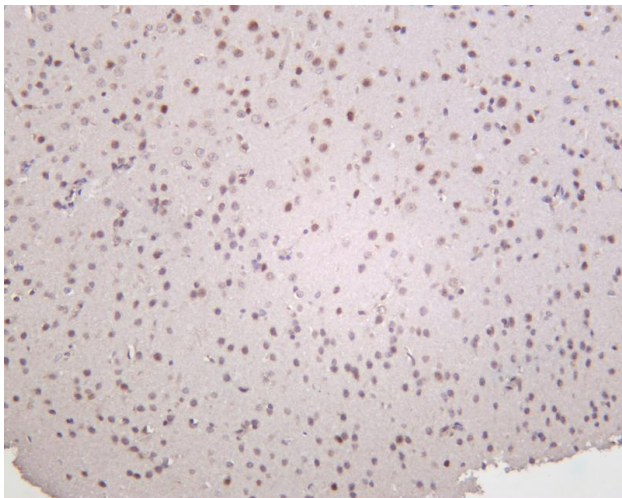
Usage suggestions

This product can be used in immunological reaction related experiments. For more information, please consult technical personnel.

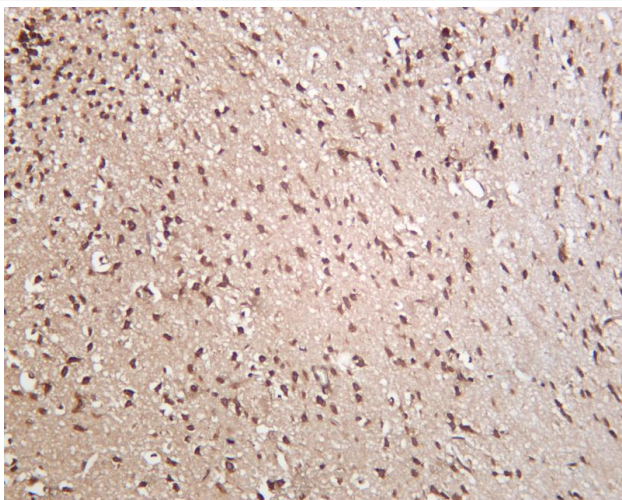




Various whole cell lysates were separated by 4-20% SDS-PAGE, and the membrane was blotted with anti-p27 KIP 1 antibody. The HRP-conjugated Goat anti-Rabbit IgG(H + L) antibody was used to detect the antibody. Lane 1: HeLa Lane 2: Rat ovary Predicted band size: 27kDa Observed band size: 27kDa

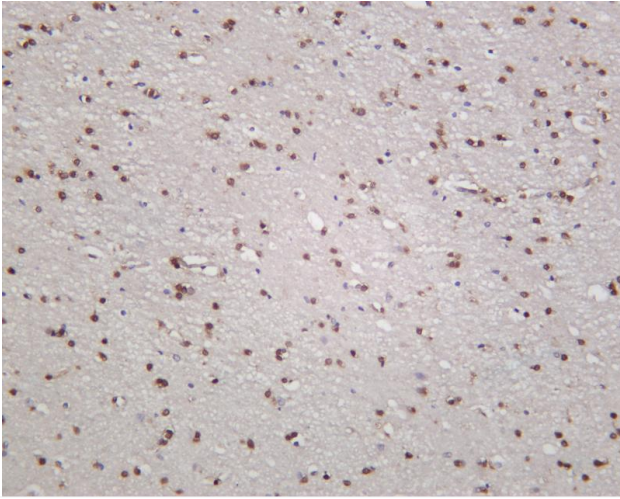


Mouse brain was stained with anti-p27 KIP 1 rabbit antibody

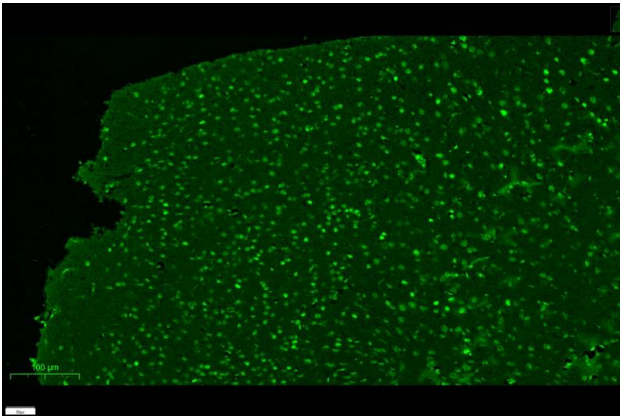


Rat brain was stained with anti-p27 KIP 1 rabbit antibody





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



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

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