

<b>Product name:</b>	p27 (phospho Thr198) Rabbit Polyclonal Antibody
<b>Cat number:</b>	ABN05151
<b>Conjugate:</b>	Unconjugated
<b>Size:</b>	100µL
<b>Clone:</b>	Polyclonal
<b>Concentration:</b>	1mg/ml
<b>Host:</b>	Rabbit
<b>Isotype:</b>	IgG
<b>Immunogen:</b>	The antiserum was produced against synthesized peptide derived from human p27 Kip1 around the phosphorylation site of Thr198. AA range:149-198
<b>Reactivity:</b>	Human,Rat,Mouse
<b>Applications:</b>	WB 1:500-1:2000,ELISA 1:10000-1:20000
<b>Molecular Weight:</b>	27kDa
<b>Purification:</b>	Affinity purification
<b>Form:</b>	Liquid
<b>Buffer:</b>	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% New type preservative N.
<b>Storage:</b>	Store at 4°C short term. Aliquot and store at -20°C for 12 months. Avoid freeze/thaw cycles.

**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],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.,