

Immunotag™ p27 (phospho Thr157) Polyclonal Antibody

Antibody Specification	
Catalog No.	ITP0990
Product Description	Immunotag™ p27 (phospho Thr157) Polyclonal Antibody
Size	50 µg, 100 µg
Conjugation	HRP, Biotin, FITC, Alexa Fluor® 350, Alexa Fluor® 405, Alexa Fluor® 488, Alexa Fluor® 555, Alexa Fluor® 594, Alexa Fluor® 647
IMPORTANT NOTE	This product is custom manufactured with a lead time of 3-4 weeks. Once in production, this item cannot be cancelled from an order and is not eligible for return.
Target Protein	p27 (Thr157)
Clonality	Polyclonal
Storage/Stability	-20°C/1 year
Application	IHC-p,ELISA
Recommended Dilution	Immunohistochemistry: 1/100 - 1/300. ELISA: 1/5000. Not yet tested in other applications.
Concentration	1 mg/ml
Reactive Species	Human
Host Species	Rabbit
Immunogen	The antiserum was produced against synthesized peptide derived from human p27 Kip1 around the phosphorylation site of Thr157. AA range:123-172
Specificity	Phospho-p27 (T157) Polyclonal Antibody detects endogenous levels of p27 protein only when phosphorylated at T157.
Purification	The antibody was affinity-purified from rabbit antiserum by affinity-chromatography using epitope-specific immunogen
Form	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.
Gene Name	CDKN1B
Accession No.	P46527 P46414
Alternate Names	CDKN1B; KIP1; Cyclin-dependent kinase inhibitor 1B; Cyclin-dependent kinase inhibitor p27; p27Kip1

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Description	cyclin dependent kinase inhibitor 1B(CDKN1B) Homo sapiens 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],
Cell Pathway/ Category	ErbB_HER,Cell_Cycle_G1S,Cell_Cycle_G2M_DNA,Pathways in cancer,Prostate cancer,Chronic myeloid leukemia,Small cell lung cancer,
Protein Expression	Bone marrow,Cervix,Epithelium,Hepatocellular carcinoma,Hippocampus,
Subcellular Localization	nucleus,nucleoplasm,cytoplasm,endosome,cytosol,Cul4A-RING E3 ubiquitin ligase complex,protein complex,

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Protein Function	<p>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 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 YWHA E and YWHA H, but not YWHA B nor YWHA Z; the interaction with YWHA Q 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.,</p>
Usage	For Research Use Only! Not for diagnostic or therapeutic procedures.