

Phospho-p27Kip1(T170) Antibody
Affinity Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP3876a**Specification**

Phospho-p27Kip1(T170) Antibody - Product Information

Application	DB,E
Primary Accession	P46527
Other Accession	P46414 , Q60439 , NP_004055.1
Reactivity	Human
Predicted	Hamster, Mouse
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Calculated MW	22073

Phospho-p27Kip1(T170) Antibody - Additional Information**Gene ID** 1027**Other Names**

Cyclin-dependent kinase inhibitor 1B, Cyclin-dependent kinase inhibitor p27, p27Kip1, CDKN1B, KIP1

Target/Specificity

This p27Kip1 Antibody is generated from rabbits immunized with a KLH conjugated synthetic phosphopeptide corresponding to amino acid residues surrounding T170 of human p27Kip1.

Dilution

DB~~1:500

E~~Use at an assay dependent concentration.

Format

Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is purified through a protein A column, followed by peptide affinity purification.

Storage

Maintain refrigerated at 2-8°C for up to 2 weeks. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.

Precautions

Phospho-p27Kip1(T170) Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

Phospho-p27Kip1(T170) Antibody - Protein Information**Name** CDKN1B {ECO:0000303|PubMed:20824794}

Function Important regulator of cell cycle progression. Inhibits the kinase activity of CDK2 bound to cyclin A, but has little inhibitory activity on CDK2 bound to SPDYA (PubMed:[28666995](#)). Involved in G1 arrest. Potent inhibitor of cyclin E- and cyclin A-CDK2 complexes. Forms a complex with cyclin type D-CDK4 complexes and is involved in the assembly, stability, and modulation of CCND1-CDK4 complex activation. Acts either as an inhibitor or an activator of cyclin type D-CDK4 complexes depending on its phosphorylation state and/or stoichiometry.

Cellular Location

Nucleus. Cytoplasm. Endosome. Note=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 Location

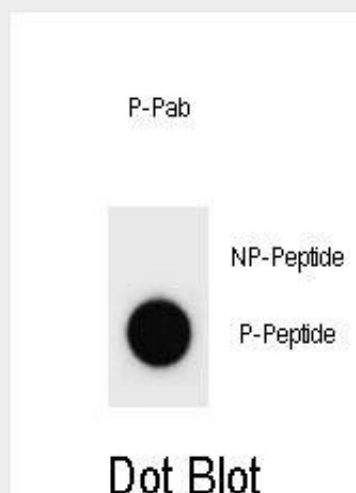
Expressed in kidney (at protein level) (PubMed:15509543). Expressed in all tissues tested (PubMed:8033212) Highest levels in skeletal muscle, lowest in liver and kidney (PubMed:8033212).

Phospho-p27Kip1(T170) Antibody - Protocols

Provided below are standard protocols that you may find useful for product applications.

- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

Phospho-p27Kip1(T170) Antibody - Images



Dot blot analysis of p27Kip1 Antibody (Phospho T170) Phospho-specific Pab (Cat. #AP3876a) on nitrocellulose membrane. 50ng of Phospho-peptide or Non Phospho-peptide per dot were adsorbed. Antibody working concentrations are 0.6ug per ml.

Phospho-p27Kip1(T170) Antibody - 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. [provided by RefSeq].

Phospho-p27Kip1(T170) Antibody - References

Kajihara, R., et al. Biochem. Biophys. Res. Commun. 401(3):350-355(2010)
Kedde, M., et al. Nat. Cell Biol. 12(10):1014-1020(2010)
Canbay, E., et al. Anticancer Res. 30(7):3093-3098(2010)
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Qin, J., et al. Hepatogastroenterology 57 (99-100), 547-553 (2010) :