

# Phospho-p27Kip1(S10) Antibody

Affinity Purified Rabbit Polyclonal Antibody (Pab) Catalog # AP3191a

## **Product Information**

Application WB, IHC-P, E Primary Accession P46527

Other Accession <u>P46414</u>, <u>Q60439</u>

Reactivity Human

**Predicted** Mouse, Hamster

Host Rabbit
Clonality Polyclonal
Isotype Rabbit IgG
Calculated MW 22073

## **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 S10 of human p27Kip1.

**Dilution** WB~~1:1000 IHC-P~~1:100~500 E~~Use at an assay dependent concentration.

**Format** Purified polyclonal antibody supplied in PBS with 0.05% (V/V) Proclin 300. 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(S10) Antibody is for research use only and not for use in

diagnostic or therapeutic procedures.

## **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 stoichometry.

#### **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).

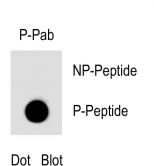
# **Background**

p27Kip1 is 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.

### References

Kawamata, N., et al., Eur. J. Haematol. 74(5):424-429 (2005). Andreu, E.J., et al., Cancer Res. 65(8):3264-3272 (2005). Wingate, H., et al., J. Biol. Chem. 280(15):15148-15157 (2005). Wang, C., et al., J. Biol. Chem. 280(13):12339-12343 (2005). Rassidakis, G.Z., et al., Blood 105(2):827-829 (2005).

## **Images**



Dot blot analysis of Phospho-p27Kip1(S10) Phospho-specific Pab on nitrocellulose membrane. 50ng of Phospho-peptide or Non Phospho-peptide per dot were adsorbed. Antobodies working concentration was 0. 5ug per ml.

## **Citations**

- Gefitinib resistance of cancer cells correlated with TM4SF5-mediated epithelial-mesenchymal transition.
- Androgen Receptor Enhances p27 Degradation in Prostate Cancer Cells through Rapid and Selective TORC2 Activation.
- The role of p27(Kip1) in dasatinib-enhanced paclitaxel cytotoxicity in human ovarian cancer cells.

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