

**Phospho-p21Cip1(S130) Antibody**  
**Affinity Purified Rabbit Polyclonal Antibody (Pab)**  
**Catalog # AP3187a**

**Specification**

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**Phospho-p21Cip1(S130) Antibody - Product Information**

Application	DB,E
Primary Accession	<a href="#">P38936</a>
Reactivity	Human
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Calculated MW	18119

**Phospho-p21Cip1(S130) Antibody - Additional Information**

**Gene ID** 1026

**Other Names**

Cyclin-dependent kinase inhibitor 1, CDK-interacting protein 1, Melanoma differentiation-associated protein 6, MDA-6, p21, CDKN1A, CAP20, CDKN1, CIP1, MDA6, PIC1, SDI1, WAF1

**Target/Specificity**

This p21Cip1 Antibody is generated from rabbits immunized with a KLH conjugated synthetic phosphopeptide corresponding to amino acid residues surrounding S130 of human p21Cip1.

**Dilution**

DB~~1:500

**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-p21Cip1(S130) Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

**Phospho-p21Cip1(S130) Antibody - Protein Information**

**Name** CDKN1A ([HGNC:1784](#))

**Function** Plays an important role in controlling cell cycle progression and DNA damage-induced G2 arrest (PubMed:[9106657](#)). Involved in p53/TP53 mediated inhibition of cellular proliferation in

response to DNA damage. Also involved in p53-independent DNA damage-induced G2 arrest mediated by CREB3L1 in astrocytes and osteoblasts (By similarity). Binds to and inhibits cyclin-dependent kinase activity, preventing phosphorylation of critical cyclin-dependent kinase substrates and blocking cell cycle progression. Functions in the nuclear localization and assembly of cyclin D-CDK4 complex and promotes its kinase activity towards RB1. At higher stoichiometric ratios, inhibits the kinase activity of the cyclin D-CDK4 complex. Inhibits DNA synthesis by DNA polymerase delta by competing with POLD3 for PCNA binding (PubMed:[11595739](#)). Negatively regulates the CDK4- and CDK6-driven phosphorylation of RB1 in keratinocytes, thereby resulting in the release of E2F1 and subsequent transcription of E2F1-driven G1/S phase promoting genes (By similarity).

**Cellular Location**

Cytoplasm. Nucleus

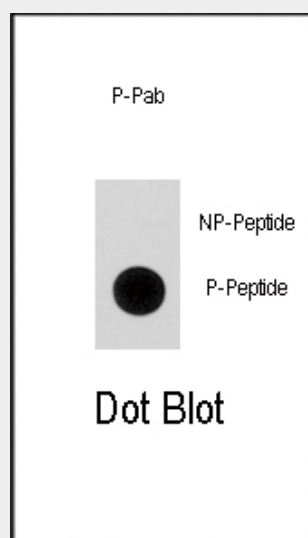
**Tissue Location**

Expressed in all adult tissues, with 5-fold lower levels observed in the brain

**Phospho-p21Cip1(S130) 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-p21Cip1(S130) Antibody - Images**

Dot blot analysis of anti-Phospho-p21Cip1-pS130 Antibody (Cat#AP3187a) on nitrocellulose membrane. 50ng of Phospho-peptide or Non Phospho-peptide per dot were adsorbed. Antibody working concentrations are 0.5ug per ml.

**Phospho-p21Cip1(S130) Antibody - Background**

p21 is a potent cyclin-dependent kinase inhibitor. It binds to and inhibits the activity of cyclin-CDK2 or -CDK4 complexes, and thus functions as a regulator of cell cycle progression at G1. The expression of this protein is tightly controlled by the tumor suppressor protein p53, through which this protein mediates the p53-dependent cell cycle G1 phase arrest in response to a variety of stress stimuli. p21 can interact with proliferating cell nuclear antigen (PCNA), a DNA polymerase accessory factor, and plays a regulatory role in S phase DNA replication and DNA damage repair. It was reported to be specifically cleaved by CASP3-like caspases, which thus leads to a dramatic activation of CDK2, and may be instrumental in the execution of apoptosis following caspase activation.

#### **Phospho-p21Cip1(S130) Antibody - References**

Scott, S.A., et al., Leuk. Res. 28(12):1293-1301 (2004).  
Amini, S., et al., J. Biol. Chem. 279(44):46046-46056 (2004).  
Chen, T., et al., Cancer Res. 64(20):7412-7419 (2004).  
Sieburg, M., et al., J. Virol. 78(19):10399-10409 (2004).  
Giraud, S., et al., Oncogene 23(44):7391-7398 (2004).