

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

Specification

Phospho-p21Cip1(S130) Antibody - Product Information

Application	DB,E
Primary Accession	P38936
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

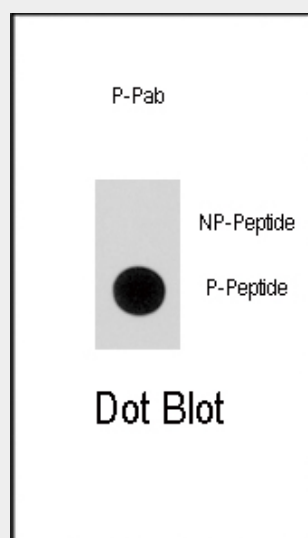
Expressed in all adult tissues, with 5-fold lower levels observed in the brain

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).