

# Phospho-p21Cip1(S130) Antibody

Affinity Purified Rabbit Polyclonal Antibody (Pab)

Catalog # AP3187a

## Product Information

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<b>Application</b>	WB, DB, E
<b>Primary Accession</b>	<a href="#">P38936</a>
<b>Reactivity</b>	Human
<b>Host</b>	Rabbit
<b>Clonality</b>	Polyclonal
<b>Isotype</b>	Rabbit IgG
<b>Clone Names</b>	RB08044
<b>Calculated MW</b>	18119

## Additional Information

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<b>Gene ID</b>	1026
<b>Other Names</b>	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
<b>Target/Specificity</b>	This p21Cip1 Antibody is generated from rabbits immunized with a KLH conjugated synthetic phosphopeptide corresponding to amino acid residues surrounding S130 of human p21Cip1.
<b>Dilution</b>	WB~~1:1000 DB~~1:500 E~~Use at an assay dependent concentration.
<b>Format</b>	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.
<b>Storage</b>	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.
<b>Precautions</b>	Phospho-p21Cip1(S130) Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

## Protein Information

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<b>Name</b>	CDKN1A ( <a href="#">HGNC:1784</a> )
<b>Function</b>	Plays an important role in controlling cell cycle progression and DNA damage-induced G2 arrest (PubMed: <a href="#">9106657</a> ). 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

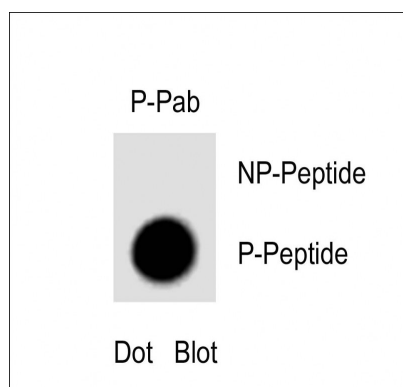
## Background

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.

## 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).

## Images



Dot blot analysis of Phospho-p21Cip1(S130) Antibody (Cat. AP3187a) on nitrocellulose membrane. 50ng of Phospho-peptide or Non Phospho-peptide per dot were adsorbed. Antibodies working concentration was 0. 5ug per ml.

Please note: All products are 'FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC OR THERAPEUTIC PROCEDURES'.