

Phospho-CCND3(S264) Antibody
Affinity Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP3885a**Specification**

Phospho-CCND3(S264) Antibody - Product Information

| | |
|-------------------|--------------------------------|
| Application | DB,E |
| Primary Accession | P30281 |
| Other Accession | NP_001129489.1 |
| Reactivity | Human |
| Host | Rabbit |
| Clonality | Polyclonal |
| Isotype | Rabbit IgG |
| Calculated MW | 32520 |

Phospho-CCND3(S264) Antibody - Additional Information**Gene ID** 896**Other Names**

G1/S-specific cyclin-D3, CCND3

Target/Specificity

This CCND3 Antibody is generated from rabbits immunized with a KLH conjugated synthetic phosphopeptide corresponding to amino acid residues surrounding S264 of human CCND3.

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-CCND3(S264) Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

Phospho-CCND3(S264) Antibody - Protein Information**Name** CCND3 {ECO:0000303|PubMed:1386336, ECO:0000312|HGNC:HGNC:1585}

Function Regulatory component of the cyclin D3-CDK4 (DC) complex that phosphorylates and inhibits members of the retinoblastoma (RB) protein family including RB1 and regulates the cell-cycle during G(1)/S transition (PubMed:[8114739](#)). Phosphorylation of RB1 allows dissociation

of the transcription factor E2F from the RB/E2F complex and the subsequent transcription of E2F target genes which are responsible for the progression through the G(1) phase (PubMed:[8114739](#)). Hypophosphorylates RB1 in early G(1) phase (PubMed:[8114739](#)). Cyclin D- CDK4 complexes are major integrators of various mitogenic and antimitogenic signals (PubMed:[8114739](#)). Component of the ternary complex, cyclin D3/CDK4/CDKN1B, required for nuclear translocation and activity of the cyclin D-CDK4 complex (PubMed:[16782892](#)). Shows transcriptional coactivator activity with ATF5 independently of CDK4 (PubMed:[15358120](#)).

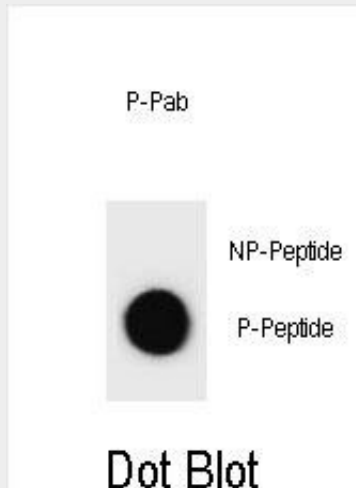
Cellular Location

Nucleus. Cytoplasm

Phospho-CCND3(S264) 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-CCND3(S264) Antibody - Images

Dot blot analysis of CCND3 Antibody (Phospho S264) Phospho-specific Pab (Cat. #AP3885a) on nitrocellulose membrane. 50ng of Phospho-peptide or Non Phospho-peptide per dot were adsorbed. Antibody working concentrations are 0.6ug per ml.

Phospho-CCND3(S264) Antibody - Background

The protein encoded by this gene belongs to the highly conserved cyclin family, whose members are characterized by a dramatic periodicity in protein abundance through the cell cycle. Cyclins function as regulators of CDK kinases. Different cyclins exhibit distinct expression and degradation patterns which contribute to the temporal coordination of each mitotic event. This cyclin forms a complex with and functions as a regulatory subunit

of CDK4 or CDK6, whose activity is required for cell cycle G1/S transition. This protein has been shown to interact with and be involved in the phosphorylation of tumor suppressor protein Rb. The CDK4 activity associated with this cyclin was reported to be necessary for cell cycle progression through G2 phase into mitosis after UV radiation. Several transcript variants encoding different isoforms have been found for this gene.

Phospho-CCND3(S264) Antibody - References

Liu, C.Y., et al. Carcinogenesis 31(7):1259-1263(2010)
Kim, J., et al. Cytokine 50(1):42-49(2010)
Kamatani, Y., et al. Nat. Genet. 42(3):210-215(2010)
Gumina, M.R., et al. Cell Cycle 9(4):820-828(2010)
Radulovich, N., et al. Mol. Cancer 9, 24 (2010) :