

CCND1 Blocking Peptide (C-term T288)

Synthetic peptide

Catalog # BP20024b

Specification**CCND1 Blocking Peptide (C-term T288) - Product Information**

Primary Accession

[P24385](#)

Other Accession

[NP_444284.1](#)**CCND1 Blocking Peptide (C-term T288) - Additional Information**

Gene ID 595

Other Names

G1/S-specific cyclin-D1, B-cell lymphoma 1 protein, BCL-1, BCL-1 oncogene, PRAD1 oncogene, CCND1, BCL1, PRAD1

Target/Specificity

The synthetic peptide sequence is selected from aa 282-294 of HUMAN CCND1

Format

Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

Storage

Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

Precautions

This product is for research use only. Not for use in diagnostic or therapeutic procedures.

CCND1 Blocking Peptide (C-term T288) - Protein Information**Name** CCND1 {ECO:0000303|PubMed:8204893, ECO:0000312|HGNC:HGNC:1582}**Function**

Regulatory component of the cyclin D1-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:1833066, PubMed:1827756, PubMed:8114739, PubMed:8302605, PubMed:19412162, PubMed:33854235). 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:1833066, PubMed:1827756, PubMed:8114739, PubMed:8302605, PubMed:19412162, PubMed:33854235).

[8114739](http://www.uniprot.org/citations/8114739), PubMed:8302605, PubMed:19412162). Hypophosphorylates RB1 in early G(1) phase (PubMed:1833066, PubMed:1827756, PubMed:8114739, PubMed:8302605, PubMed:19412162). Cyclin D-CDK4 complexes are major integrators of various mitogenic and antimitogenic signals (PubMed:1833066, PubMed:1827756, PubMed:8302605, PubMed:19412162). Also a substrate for SMAD3, phosphorylating SMAD3 in a cell-cycle-dependent manner and repressing its transcriptional activity (PubMed:15241418). Component of the ternary complex, cyclin D1/CDK4/CDKN1B, required for nuclear translocation and activity of the cyclin D-CDK4 complex (PubMed:9106657). Exhibits transcriptional corepressor activity with INSM1 on the NEUROD1 and INS promoters in a cell cycle-independent manner (PubMed:16569215, PubMed:18417529).

Cellular Location

Nucleus. Cytoplasm Nucleus membrane. Note=Cyclin D-CDK4 complexes accumulate at the nuclear membrane and are then translocated to the nucleus through interaction with KIP/CIP family members

CCND1 Blocking Peptide (C-term T288) - Protocols

Provided below are standard protocols that you may find useful for product applications.

- [Blocking Peptides](#)

CCND1 Blocking Peptide (C-term T288) - Images

CCND1 Blocking Peptide (C-term T288) - 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 throughout 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 tumor suppressor protein Rb and the expression of this gene is regulated positively by Rb. Mutations, amplification and overexpression of this gene, which alters cell cycle progression, are observed frequently in a variety of tumors and may contribute to tumorigenesis.

CCND1 Blocking Peptide (C-term T288) - References

Aggarwal, P., et al. Cancer Cell 18(4):329-340(2010) Iwatani, K., et al. Biochem. Biophys. Res. Commun. 400(3):426-431(2010) Halilovic, E., et al. Cancer Res. 70(17):6804-6814(2010) Zheng, W., et al. Anal. Quant. Cytol. Histol. 32(3):155-160(2010) Satiroglu-Tufan, N.L., et al. Genet. Mol. Res. 9(3):1557-1567(2010)