

CTNB1 Antibody (C-term) Blocking peptide Synthetic peptide

Catalog # BP11231b

Specification

CTNB1 Antibody (C-term) Blocking peptide - Product Information

Primary Accession

<u>P35222</u>

CTNB1 Antibody (C-term) Blocking peptide - Additional Information

Gene ID 1499

Other Names Catenin beta-1, Beta-catenin, CTNNB1, CTNNB

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.

CTNB1 Antibody (C-term) Blocking peptide - Protein Information

Name CTNNB1 (HGNC:2514)

Synonyms CTNNB

Function

Key downstream component of the canonical Wnt signaling pathway (PubMed:17524503, PubMed:18077326, PubMed:18086858, PubMed:18086858, PubMed:21262353, PubMed:21262353, PubMed:22155184, PubMed:22647378, PubMed:22647378, PubMed:22647378, PubMed:22699938, In the absence of Wnt, forms a complex with AXIN1, AXIN2, APC, CSNK1A1 and GSK3B that promotes phosphorylation on N- terminal Ser and Thr residues and ubiquitination of CTNNB1 via BTRC and its subsequent degradation by the proteasome (PubMed:17524503, PubMed:18077326, PubMed:18086858, PubMed:180877326, PubMed:18086858, PubMed:18086858, PubMed:18086858, PubMed:18086858, PubMed:<a href="http://www.uniprot.org/citations/180



href="http://www.uniprot.org/citations/21262353" target=" blank">21262353, PubMed:22155184, PubMed:22647378, PubMed:22699938). In the presence of Wnt ligand, CTNNB1 is not ubiquitinated and accumulates in the nucleus, where it acts as a coactivator for transcription factors of the TCF/LEF family, leading to activate Wnt responsive genes (PubMed: 17524503, PubMed:18077326, PubMed:18086858, PubMed:18957423, PubMed:21262353, PubMed:22155184, PubMed:22647378, PubMed:22699938). Involved in the regulation of cell adhesion, as component of an E-cadherin:catenin adhesion complex (By similarity). Acts as a negative regulator of centrosome cohesion (PubMed:18086858). Involved in the CDK2/PTPN6/CTNNB1/CEACAM1 pathway of insulin internalization (PubMed:21262353). Blocks anoikis of malignant kidney and intestinal epithelial cells and promotes their anchorage-independent growth by down-regulating DAPK2 (PubMed:18957423). Disrupts PML function and PML- NB formation by inhibiting RANBP2-mediated sumoylation of PML (PubMed: 22155184). Promotes neurogenesis by maintaining sympathetic neuroblasts within the cell cycle (By similarity). Involved in chondrocyte differentiation via interaction with SOX9: SOX9-binding competes with the binding sites of TCF/LEF within CTNNB1, thereby inhibiting the Wnt signaling (By similarity). Acts as a positive regulator of odontoblast differentiation during mesenchymal tooth germ formation, via promoting the transcription of differentiation factors such as LEF1, BMP2 and BMP4 (By similarity). Activity is repressed in a MSX1-mediated manner at the bell stage of mesenchymal tooth germ formation which prevents premature differentiation of odontoblasts (By similarity).

Cellular Location

Cytoplasm. Nucleus. Cytoplasm, cytoskeleton {ECO:0000250|UniProtKB:B6V8E6}. Cell junction, adherens junction. Cell junction {ECO:0000250|UniProtKB:B6V8E6}. Cell membrane. Cytoplasm, cytoskeleton, microtubule organizing center, centrosome. Cytoplasm, cytoskeleton, spindle pole. Synapse {ECO:0000250|UniProtKB:Q02248} Cytoplasm, cytoskeleton, cilium basal body {ECO:0000250|UniProtKB:Q02248}. Note=Colocalized with RAPGEF2 and TJP1 at cell-cell contacts (By similarity). Cytoplasmic when it is un-stable (highly phosphorylated) or bound to CDH1. Translocates to the nucleus when it is stabilized (low level of phosphorylation). Interaction with GLIS2 and MUC1 promotes nuclear translocation. Interaction with EMD inhibits nuclear localization. The majority of beta-catenin is localized to the cell membrane. In interphase, colocalizes with CROCC between CEP250 puncta at the proximal end of centrioles, and this localization is dependent on CROCC and CEP250. In mitosis, when NEK2 activity increases, it localizes to centrosomes at spindle poles independent of CROCC. Colocalizes with CDK5 in the cell-cell contacts and plasma membrane of undifferentiated and differentiated neuroblastoma cells. Interaction with FAM53B promotes translocation to the nucleus (PubMed:25183871). {ECO:0000250|UniProtKB:B6V8E6, ECO:0000269|PubMed:25183871}

Tissue Location

Expressed in several hair follicle cell types: basal and peripheral matrix cells, and cells of the outer and inner root sheaths. Expressed in colon. Present in cortical neurons (at protein level). Expressed in breast cancer tissues (at protein level) (PubMed:29367600).

CTNB1 Antibody (C-term) Blocking peptide - Protocols



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

<u>Blocking Peptides</u>

CTNB1 Antibody (C-term) Blocking peptide - Images

CTNB1 Antibody (C-term) Blocking peptide - Background

The protein encoded by this gene is part of a complex of proteins that constitute adherens junctions (AJs). AJs arenecessary for the creation and maintenance of epithelial celllayers by regulating cell growth and adhesion between cells. The encoded protein also anchors the actin cytoskeleton and may be responsible for transmitting the contact inhibition signal that causes cells to stop dividing once the epithelial sheet is complete. Finally, this protein binds to the product of the APCgene, which is mutated in adenomatous polyposis of the colon. Mutations in this gene are a cause of colorectal cancer (CRC), pilomatrixoma (PTR), medulloblastoma (MDB), and ovarian cancer. Three transcript variants encoding the same protein have been foundfor this gene.

CTNB1 Antibody (C-term) Blocking peptide - References

Huang, W., et al. Mol. Cell. Biol. 30(19):4575-4594(2010)Chairoungdua, A., et al. J. Cell Biol. 190(6):1079-1091(2010)Mirza, M.K., et al. J. Exp. Med. 207(8):1675-1685(2010)Guo, Q., et al. Acta Biochim. Biophys. Sin. (Shanghai) 42(7):450-456(2010)Teng, Y., et al. Zhonghua Yi Xue Za Zhi 90(14):988-992(2010)