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[Marker Primary Antibody]

Rabbit Anti-phospho-beta Catenin (Ser33)/FITC Conjugated antibody

Catalog Number: SL20605R-FITC

FOR RESEARCH USE ONLY

[DATASHEET]

Host:	Rabbit
Isotype:	IgG
Clonality:	Polyclonal
Target:	Anti-phospho-beta Catenin (Ser33)/FITC
Immunogen:	KLH conjugated synthetic peptide derived from human phospho-beta Catenin (Ser33)
Purification:	affinity purified by Protein A
Concentration:	1mg/ml
Storage:	Store at -20 °C for one year. Avoid repeated freeze/thaw cycles. The lyophilized antibody is stable at room temperature for at least one month and for greater than a year when kept at -20°C. When reconstituted in sterile pH 7.4 1M PBS or diluent of antibody the antibody is stable for at least two weeks at 2-4 °C.
Applications:	Flow-Cyt=1μg/Test IF=1:100-500 not yet tested in other applications. optimal dilutions/concentrations should be determined by the end user.
Reactivity:	Human,Mouse(predicted:Rat,Chicken,Pig,Rabbit)
Background:	background: The protein encoded by this gene is part of a complex of proteins that constitute adherens junctions (AJs). AJs are necessary for the creation and maintenance of epithelial cell layers 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 APC gene, 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 found for this gene.[provided by RefSeq, Oct 2009]. **Function:** Key downstream component of the canonical Wnt signaling pathway. In the absence of Wnt, forms a complex with AXIN1, AXIN2, APC,

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	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. 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. Involved in the regulation of cell adhesion. Acts as a negative regulator of centrosome cohesion. Involved in the CDK2/PTPN6/CTNNB1/CEACAM1 pathway of insulin internalization. Blocks anoikis of malignant kidney and intestinal epithelial cells and promotes their anchorage-independent growth by down-regulating DAPK2. Disrupts PML function and PML-NB formation by inhibiting RANBP2-mediated sumoylation of PML. **Subunit:** Two separate complex-associated pools are found in the cytoplasm. The majority is present as component of an E-cadherin/ catenin adhesion complex composed of at least E-cadherin/CDH1 and beta-catenin/CTNNB1, and possibly alpha-catenin/CTNNA1; the complex is located to adherens junctions. The stable association of CTNNA1 is controversial as CTNNA1 was shown not to bind to F-actin when assembled in the complex. Alternatively, the CTNNA1-containing complex may be linked to F-actin by other proteins such as LIMA1. Another cytoplasmic pool is part of a large complex containing 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. Wnt-dependent activation of DVL antagonizes the action of GSK3B. When GSK3B activity is inhibited the complex dissociates, CTNNB1 is dephosphorylated and is no longer targeted for destruction. The stabilized protein translocates to the nucleus, where it binds TCF/LEF-1 family members, TBP, BCL9 and possibly also RUVBL1 and CHD8. Binds CTNNBIP and EP300. CTNNB1 forms a ternary complex with LEF1 and EP300 that is disrupted by CTNNBIP1 binding (By similarity). Interacts with TAX1BP3 (via the PDZ domain); this interaction inhibits the transcriptional activity of CTNNB1 (By similarity). Interacts with AJAP1, BAIAP1, CARM1, CTNNA3, CXADR and PCDH11Y. Binds SLC9A3R1. Interacts with GLIS2 and MUC1. Interacts with SLC30A9. Interacts with XIRP1 (By similarity). Interacts directly with AXIN1; the interaction is regulated by CDK2 phosphorylation of AXIN1 (By similarity). Interacts with SCRIB (By similarity).

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	Interacts with PTPRU (via the cytoplasmic juxtamembrane domain). Interacts with EMD. Interacts with TNIK and TCF7L2. Interacts with SESTD1 and TRPC4. Interacts with CAV1. Interacts with TRPV4. The TRPV4 and CTNNB1 complex can interact with CDH1. Interacts with VCL (By similarity). Interacts with PTPRJ. Interacts with PKT7 and CDK2. Interacts with FAT1 (via the cytoplasmic domain) (By similarity). Interacts with NANOS1 and NDRG2. Interacts with isoform 1 of NEK2. Interacts with both isoform 1 and isoform 2 of CDK5. Interacts with PTK6. Interacts with SOX7; this interaction may lead to proteasomal degradation of active CTNNB1 and thus inhibition of Wnt/beta-catenin-stimulated transcription. Identified in a complex with HINT1 and MITF. Interacts with FHIT. The CTNNB1 and TCF4 complex interacts with PML (isoform PML-4). Interacts with FERMT2. Identified in a complex with TCF4 and FERMT2. **Subcellular Location:** Cytoplasm. Nucleus. Cytoplasm, cytoskeleton. Cell junction, adherens junction. Cell junction. Cell membrane. Cytoplasm, cytoskeleton, centrosome. Cytoplasm, cytoskeleton, spindle pole. Note=Cytoplasmic when it is unstabilized (high level of phosphorylation) 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. Co-localizes with CDK5 in the cell-cell contacts and plasma membrane of undifferentiated and differentiated neuroblastoma cells. **Tissue Specificity:** 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). **Post-translational modifications:** Phosphorylation at Ser-552 by AMPK promotes stabilizion of the protein, enhancing TCF/LEF-mediated transcription. Phosphorylation by GSK3B requires prior phosphorylation of Ser-45 by another kinase. Phosphorylation proceeds then from Thr-41 to Ser-37 and Ser-33. Phosphorylated by NEK2. EGF stimulates tyrosine phosphorylation. Phosphorylation on Tyr-654 decreases CDH1 binding and

	enhances TBP binding. Phosphorylated on Ser-33 and Ser-37 by HIPK2. This phosphorylation triggers proteasomal degradation. Phosphorylation on Ser-191 and Ser-246 by CDK5. Phosphorylation by CDK2 regulates insulin internalization. Phosphorylation by PTK6 at Tyr-64, Tyr-142, Tyr-331 and/or Tyr-333 with the predominant site at Tyr-64 is not essential for inhibition of transcriptional activity. Ubiquitinated by the SCF(BTRC) E3 ligase complex when phosphorylated by GSK3B, leading to its degradation. Ubiquitinated by a E3 ubiquitin ligase complex containing UBE2D1, SIAH1, CACYBP/SIP, SKP1, APC and TBL1X, leading to its subsequent proteasomal degradation (By similarity). S-nitrosylation at Cys-619 within adherens junctions promotes VEGF-induced, NO-dependent endothelial cell permeability by disrupting interaction with E-cadherin, thus mediating disassembly adherens junctions. DISEASE: Defects in CTNNB1 are associated with colorectal cancer (CRC) [MIM:114500]. Note=Activating mutations in CTNNB1 have oncogenic activity resulting in tumor development. Somatic mutations are found in various tumor types, including colon cancers, ovarian and prostate carcinomas, hepatoblastoma (HB), hepatocellular carcinoma (HCC). HBs are malignant embryonal tumors mainly affecting young children in the first three years of life. Defects in CTNNB1 are a cause of pilomatrixoma (PTR) [MIM:132600]; a common benign skin tumor. Defects in CTNNB1 are a cause of medulloblastoma (MDB) [MIM:155255]. MDB is a malignant, invasive embryonal tumor of the cerebellum with a preferential manifestation in children. Defects in CTNNB1 are a cause of susceptibility to ovarian cancer (OC) [MIM:167000]. Ovarian cancer common malignancy originating from ovarian tissue. Although many histologic types of ovarian neoplasms have been described, epithelial ovarian carcinoma is the most common form. Ovarian cancers are often asymptomatic and the recognized signs and symptoms, even of late-stage disease, are vague. Consequently, most patients are diagnosed with advanced disease. **Similarity:** Belongs to the beta-catenin family. Contains 12 ARM repeats. **Database links:** **Entrez Gene:** 1499 Human **Entrez Gene:** 12387 Mouse **Entrez Gene:** 84353 Rat Omim: 116806 Human **SwissProt:** P35222 Human

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	SwissProt: Q02248 Mouse **SwissProt:** Q9WU82 Rat **Unigene:** 476018 Human **Unigene:** 291928 Mouse **Unigene:** 112601 Rat Important **Note:** This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.