G-Biosciences Image not found or type unknown

Immunotag™ Smad4 Polyclonal Antibody

Antibody Specification

Catalog No.	ITT4337
Product Description	Immunotag™ Smad4 Polyclonal Antibody
Size	50 µg, 100 µg
Conjugation	HRP, Biotin, FITC, Alexa Fluor® 350, Alexa Fluor® 405, Alexa Fluor® 488, Alexa Fluor® 555, Alexa Fluor® 594, Alexa Fluor® 647
IMPORTANT NOTE	This product is custom manufactured with a lead time of 3-4 weeks. Once in production, this item cannot be cancelled from an order and is not eligible for return.
Target Protein	SMAD4
Clonality	Polyclonal
Storage/Stability	-20°C/1 year
Application	WB,IHC-p,IF,ELISA
Recommended Dilution	Western Blot: 1/500 - 1/2000. Immunohistochemistry: 1/100 - 1/300. Immunofluorescence: 1/200 - 1/1000. ELISA: 1/10000. Not yet tested in other applications.
Concentration	1 mg/ml
Reactive Species	Human,Mouse,Rat,Monkey
Host Species	Rabbit
Immunogen	Synthesized peptide derived from Smad4, at AA range: 40-120
Specificity	Smad4 Polyclonal Antibody detects endogenous levels of Smad4 protein.
Purification	The antibody was affinity-purified from rabbit antiserum by affinity-chromatography using epitope-specific immunogen
Form	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.
Gene Name	SMAD4
Accession No.	Q13485 P97471 O70437
Alternate Names	SMAD4; DPC4; MADH4; Mothers against decapentaplegic homolog 4; MAD homolog 4; Mothers against DPP homolog 4; Deletion target in pancreatic carcinoma 4; SMAD family member 4; SMAD 4; Smad4; hSMAD4

Antibody Specification

Description	SMAD family member 4(SMAD4) Homo sapiens This gene encodes a member of the Smad family of signal transduction proteins. Smad proteins are phosphorylated and activated by transmembrane serine-threonine receptor kinases in response to TGF-beta signaling. The product of this gene forms homomeric complexes and heteromeric complexes with other activated Smad proteins, which then accumulate in the nucleus and regulate the transcription of target genes. This protein binds to DNA and recognizes an 8-bp palindromic sequence (GTCTAGAC) called the Smad-binding element (SBE). The Smad proteins are subject to complex regulation by post-translational modifications. Mutations or deletions in this gene have been shown to result in pancreatic cancer, juvenile polyposis syndrome, and hereditary hemorrhagic telangiectasia syndrome. [provided by RefSeq, Oct 2009],
Cell Pathway/ Category	Cell_Cycle_G1S,Cell_Cycle_G2M_DNA,WNT,WNT-T CELLTGF-beta,Adherens_Junction,Pathways in cancer,Colorectal cancer,Pancreatic cancer,Chronic myeloid leukemia,
Protein Expression	Fetal brain,Muscle,Placenta,
Subcellular Localization	nuclear chromatin,intracellular,nucleus,nucleoplasm,transcription factor complex,cytoplasm,cytosol,activin responsive factor complex,SMAD protein complex,RNA polymerase II transcription factor complex,

Antibody Specification

Protein Function	disease:Defects in SMAD4 are a cause of juvenile polyposis syndrome (JPS) [MIM:174900]; also known as juvenile intestinal polyposis (JIP). JPS is an autosomal dominant gastrointestinal hamartomatous polyposis syndrome in which patients are at risk for developing gastrointestinal cancers. The lesions are typified by a smooth histological appearance, predominant stroma, cystic spaces and lack of a smooth muscle core. Multiple juvenile polyps usually occur in a number of Mendelian disorders. Sometimes, these polyps occur without associated features as in JPS; here, polyps tend to occur in the large bowel and are associated with an increased risk of colon and other gastrointestinal cancers.,disease:Defects in SMAD4 are a cause of juvenile polyposis/hereditary hemorrhagic telangiectasia syndrome (JP/HHT) [MIM:175050]. JP/HHT syndrome phenotype consists of the coexistence of juvenile polyposis (JIP) and hereditary hemorrhagic telangiectasia (HHT) [MIM:187300] in a single individual. JIP and HHT are autosomal dominant disorders with distinct and non-overlapping clinical features. The former, an inherited gastrointestinal malignancy predisposition, is caused by mutations in SMAD4 or BMPR1A, and the latter is a vascular malformation disorder caused by mutations in ENG or ACVRL1. All four genes encode proteins involved in the transforming-growth-factor-signaling pathway. Although there are reports of patients and families with phenotypes of both disorders combined, the genetic aetiology of this association is unknown.,disease:Defects in SMAD4 are a cause of pancreatic carcinoma [MIM:260350].,disease:Defects in SMAD4 may be a cause of colorectal cancer (CRC) [MIM:114500].,function:Common mediator of signal transduction by TGF-beta (transforming growth factor) superfamily; SMAD4 is the common SMAD (co-SMAD). Promotes binding of the SMAD2/SMAD4/FAST-1 complex to DNA and provides an activation function required for SMAD1 or SMAD2 to stimulate transcription. May act as a tumor suppressor.,PTM:Monoubiquitinated on Lys-519 by E3 ubiquitin-protein ligase TRIM33. Monoubiquitination hampers its ability to form a stable complex with activated SMAD2/3 resulting in inhibition of TGF-beta/BMP signaling cascade.,similarity:Belongs to the dwarfin/SMAD family.,similarity:Contains 1 MH1 (MAD homology 1) domain.,similarity:Contains 1 MH2 (MAD homology 2) domain.,subcellular location:Cytoplasmic in the absence of ligand. Migrates to the nucleus when complexed with R-SMAD.,subunit:May form trimers with receptor-regulated SMAD (R-SMAD). Found in a ternary complex composed of SMAD4, STK11 and STK11IP. Interacts with ATF2, COPS5, DACH1, MSG1, SKI, STK11, STK11IP and TRIM33. Associates with ZNF423 or ZNF521 in response to BMP2 leading to activate transcription of BMP target genes. Interacts with USP9X.,
Usage	For Research Use Only! Not for diagnostic or therapeutic procedures.