



Smad4 Rabbit mAb

Catalog No	YP-rAb-17757
Isotype	IgG
Reactivity	Human,Mouse,Rat
Applications	WB,IHC,IF,IP,ELISA
Gene Name	SMAD4
Protein Name	Mothers against decapentaplegic homolog 4
Purification Process	Protein A
Specificity	Endogenous
Formulation	PBS, 50% glycerol, 0.05% Proclin 300, 0.05%BSA
Source	Monoclonal, Rabbit,IgG
Dilution	IHC 1:2000-1:10000; WB 1:2000-1:10000; IF 1:200-1:1000; ELISA 1:5000-1:20000; IP 1:50-1:200; Note: For IHC, we suggest antigen retrieval with TE buffer pH 9.0
Concentration	0.5 mg/ml
Purity	≥90%
Storage Stability	-15 ° C to -25 ° C/1 year(Do not lower than -25 ° C)
Synonyms	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
Observed Band	60kD
Calculated Molecular Weight	60kD
Cell Pathway	Cytoplasm
Tissue Specificity	Fetal brain,Muscle,Placenta,
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.**

Background

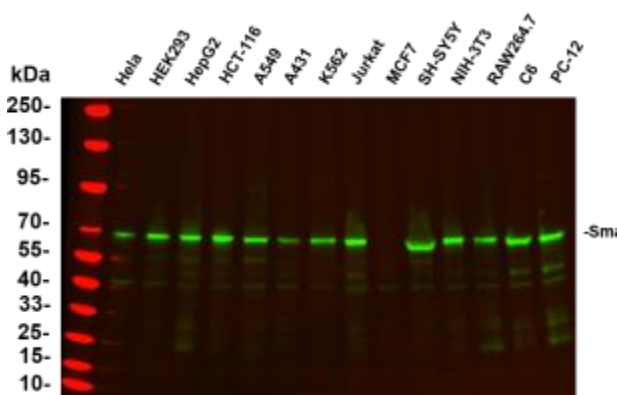
This gene encodes a member of the Smad family of signal transduction proteins. Smad proteins are phosphorylated and activated by transmembrane serine-threonine receptor kinases in response to TGF-beta signaling. The product of this gene forms homomeric complexes and heteromeric complexes with other activated 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].

matters needing attention

Avoid repeated freezing and thawing!

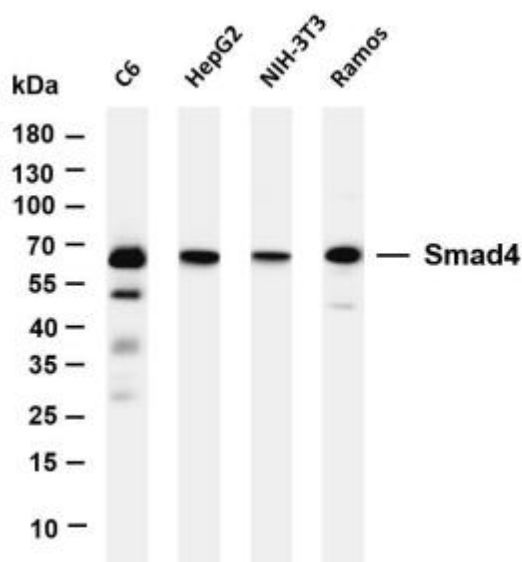
Usage suggestions

This product can be used in immunological reaction related experiments. For more information, please consult technical personnel.

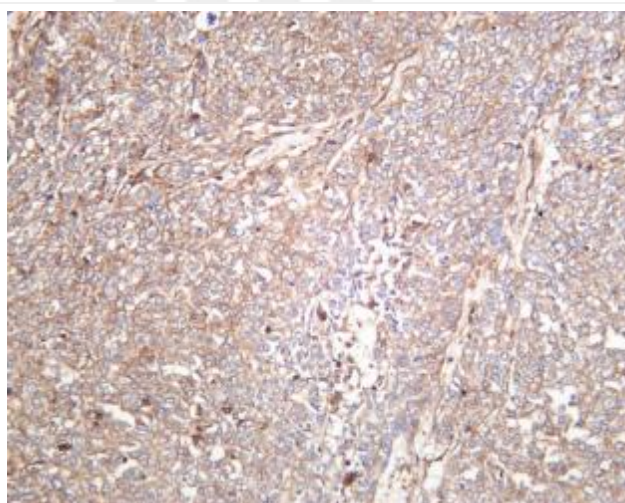


Various whole cell lysates were separated by 4-20% SDS-PAGE, and the primary antibody was used at 4°C, over night with a 1:5000 dilution. The Dylight 800-conjugated Goat anti-Rabbit antibody

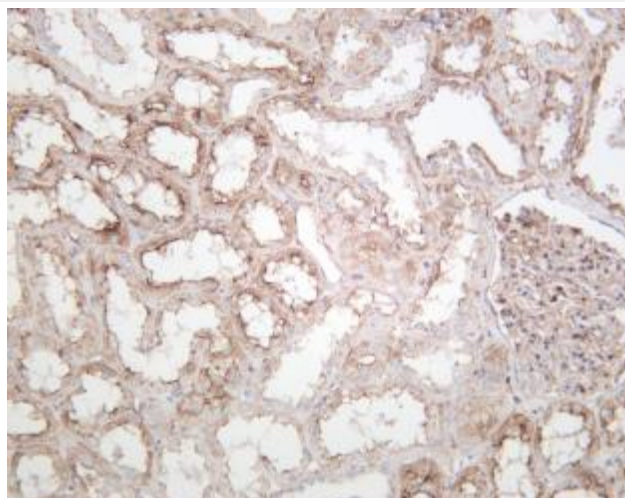




Various whole cell lysates were separated by 4-20% SDS-PAGE, and the membrane was blotted with anti-Smad4 antibody. The HRP-conjugated Goat anti-Rabbit IgG(H + L) antibody was used to detect the antibody. Lane 1: C6 Lane 2: HepG2 Lane 3: NIH-3T3 Lane 4: Ramos Predicted band size: 60kDa Observed band size: 60kDa

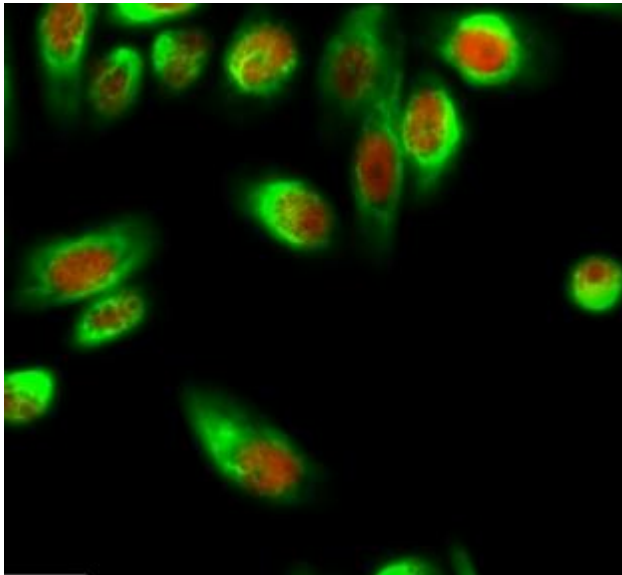


Human breast carcinoma was stained with anti-Smad4 rabbit antibody



Human kidney was stained with anti-Smad4 rabbit antibody





Immunofluorescence analysis of HeLa cell. 1, Smad4 Antibody (red) was diluted at 1:200 (4 ° overnight). GAPDH Monoclonal Antibody (2B8) (green) was diluted at 1:200 (4 ° overnight). 2, Goat Anti Rabbit Alexa Fluor 594 Catalog: RS3611 was diluted at 1:1000 (room temperature, 50min). Goat Anti Mouse Alexa Fluor 488 Catalog: RS3208 was diluted at 1:1000 (room temperature, 50min).

