Smad4 (Phospho Thr276) Rabbit pAb

CatalogNo: YP1667

Key Features

Host Species
- Rabbit

Reactivity
- Human,Mouse,Rat

Applications
- WB

MW
- 61kD (Calculated)

Isotype
- IgG

Storage

Storage*	-15°C to -25°C/1 year (Do not lower than -25°C)
Formulation	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.

Recommended Dilution Ratios

WB 1:500-2000

Basic Information

Clonality	Polyclonal

Immunogen Information

Immunogen	Synthesized peptide derived from human Smad4 (Phospho-Thr276)
Specificity	This antibody detects endogenous levels of Smad4 (Phospho-Thr276) at Human, Mouse,Rat.The name of modified sites may be influenced by many factors, such as species (the modified site was not originally found in human samples) and the change of protein sequence (the previous protein sequence is incomplete, and the protein sequence may be prolonged with the development of protein sequencing technology). When naming, we will use the "numbers" in historical reference to keep the sites consistent with the reports. The antibody binds to the following modification sequence (lowercase letters are modification sites):PYtPN

Target Information

Gene name SMAD4 DPC4 MADH4

Protein Name Smad4 (Phospho-Thr276)

Organism	Gene ID	UniProt ID
Human	4089;	Q13485;
Mouse	17128;	P97471;
Rat	50554;	O70437;

Cellular Localization Cytoplasm . Nucleus . Cytoplasmic in the absence of ligand. Migrates to the nucleus when complexed with R-SMAD (PubMed:15799969) . PDPK1 prevents its nuclear translocation in response to TGF-beta (PubMed:17327236) . .

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. ,

Validation Data

Contact information

Orders:	order.cn@immunoway.com
Support:	support.cn@immunoway.com
Telephone:	400-8787-807(China)
Website:	http://www.immunoway.com.cn
Address:	2200 Ringwood Ave San Jose, CA 95131 USA

Please scan the QR code to access additional product information:
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