

## Smad4 (Phospho Thr276) rabbit pAb

Catalog No: YP1667

**Reactivity:** Human; Mouse; Rat

**Applications:** WB

Target: Smad4

Fields: >>FoxO signaling pathway;>>Cell cycle;>>Wnt signaling pathway;>>TGF-beta

signaling pathway;>>Apelin signaling pathway;>>Hippo signaling

pathway;>>Adherens junction;>>Signaling pathways regulating pluripotency of stem cells;>>Th17 cell differentiation;>>AGE-RAGE signaling pathway in diabetic

complications;>>Hepatitis B;>>Human T-cell leukemia virus 1 infection;>>Pathways in cancer;>>Colorectal cancer;>>Pancreatic

cancer;>>Chronic myeloid leukemia;>>Hepatocellular carcinoma;>>Gastric

cancer

Gene Name: SMAD4 DPC4 MADH4

**Protein Name:** Smad4 (Phospho-Thr276)

Q13485

P97471

Human Gene Id: 4089

**Human Swiss Prot** 

No:

Mouse Gene Id: 17128

**Mouse Swiss Prot** 

No:

Rat Gene Id: 50554

Rat Swiss Prot No: 070437

Immunogen: Synthesized peptide derived from human Smad4 (Phospho-Thr276)

**Specificity:** This antibody detects endogenous levels of Smad4 (Phospho-Thr276) at

Human, Mouse, Rat

Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.

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**Soumdation:** Polyclonal, Rabbit, IgG

**Dilution:** WB 1:500-2000

**Purification:** The antibody was affinity-purified from rabbit serum by affinity-chromatography

using specific immunogen.

Concentration: 1 mg/ml

-15°C to -25°C/1 year(Do not lower than -25°C) Storage Stability:

Molecularweight: 61kD

This gene encodes a member of the Smad family of signal transduction proteins. **Background:** 

> Smad proteins are phosphorylated and activated by transmembrane serinethreonine 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],

**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

Subcellular

Cytoplasm . Nucleus . Cytoplasmic in the absence of ligand. Migrates to the nucleus when complexed with R-SMAD (PubMed:15799969). PDPK1 prevents Location:

its nuclear translocation in response to TGF-beta (PubMed:17327236). .

Fetal brain, Muscle, Placenta, **Expression:** 

Sort: 25151

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**No4**: \_\_1

## **Products Images**

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