



Smad4 (Phospho-Thr276) rabbit pAb

| Catalog No | BYab-10443 |
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| Isotype | lgG |
| Reactivity | Human; Mouse;Rat |
| Applications | WB |
| Gene Name | SMAD4 DPC4 MADH4 |
| Protein Name | Smad4 (Phospho-Thr276) |
| Immunogen | Synthesized peptide derived from human Smad4 (Phospho-Thr276) |
| Specificity | This antibody detects endogenous levels of Smad4 (Phospho-Thr276) at Human, Mouse,Rat |
| Formulation | Liquid in PBS containing 50% glycerol, and 0.94% sodium azide. |
| Source | Polyclonal, Rabbit,IgG |
| Purification | The antibody was affinity-purified from rabbit serum by affinity-chromatography using specific immunogen. |
| Dilution | WB 1:500-2000 |
| Concentration | 1 mg/ml |
| Purity | ≥90% |
| Storage Stability | -20°C/1 year |
| Synonyms | 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 | |
| Cell Pathway | 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 |
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| | 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 |
| 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. |

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