

Product datasheet

Anti-Smad4 antibody - N-terminal ab191026

[2 References](#) [5 Images](#)

Overview

Product name	Anti-Smad4 antibody - N-terminal
Description	Rabbit polyclonal to Smad4 - N-terminal
Host species	Rabbit
Tested applications	Suitable for: WB, IHC-P
Species reactivity	Reacts with: Rat, Human Predicted to work with: Mouse, Cow, Pig
Immunogen	Synthetic peptide corresponding to Human Smad4 aa 97-113 (N terminal). Sequence: RLWRWPD L HKNELKHVK Database link: Q13485 Run BLAST with Run BLAST with
Positive control	Rat Skeletal Muscle Tissue Lysate, A549 and U87 Cell Lysates; Human Lung Cancer Tissue; Rat Intestine and Brain Tissues.

Properties

Form	Liquid
Storage instructions	Shipped at 4°C. Store at +4°C short term (1-2 weeks). Upon delivery aliquot. Store at -20°C long term. Avoid freeze / thaw cycle.
Storage buffer	Preservatives: 0.025% Thimerosal (merthiolate), 0.025% Sodium azide Constituents: 2.5% BSA, 0.1% Dibasic monohydrogen sodium phosphate, 0.45% Sodium chloride
Purity	Immunogen affinity purified
Clonality	Polyclonal
Isotype	IgG

Applications

Our [Abpromise guarantee](#) covers the use of **ab191026** in the following tested applications.

The application notes include recommended starting dilutions; optimal dilutions/concentrations should be determined by the end user.

Application	Abreviews	Notes
WB		Use a concentration of 0.1 - 0.5 µg/ml. Predicted molecular weight: 60 kDa. The detection limit for Smad4 is approximately 2.5ng/lane under reducing conditions
IHC-P		Use a concentration of 0.5 - 1 µg/ml. Perform heat mediated antigen retrieval with citrate buffer pH 6 before commencing with IHC staining protocol.

Target

Function

Common SMAD (co-SMAD) is the coactivator and mediator of signal transduction by TGF-beta (transforming growth factor). Component of the heterotrimeric SMAD2/SMAD3-SMAD4 complex that forms in the nucleus and is required for the TGF-mediated signaling. Promotes binding of the SMAD2/SMAD4/FAST-1 complex to DNA and provides an activation function required for SMAD1 or SMAD2 to stimulate transcription. Component of the multimeric SMAD3/SMAD4/JUN/FOS complex which forms at the AP1 promoter site; required for synergistic transcriptional activity in response to TGF-beta. May act as a tumor suppressor.

Involvement in disease

Defects in SMAD4 are a cause of pancreatic cancer (PNCA) [MIM:260350].
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.
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 etiology of this association is unknown.
Defects in SMAD4 may be a cause of colorectal cancer (CRC) [MIM:114500].

Sequence similarities

Belongs to the dwarfin/SMAD family.
Contains 1 MH1 (MAD homology 1) domain.
Contains 1 MH2 (MAD homology 2) domain.

Domain

The MH1 domain is required for DNA binding.
The MH2 domain is required for both homomeric and heteromeric interactions and for transcriptional regulation. Sufficient for nuclear import.

Post-translational modifications

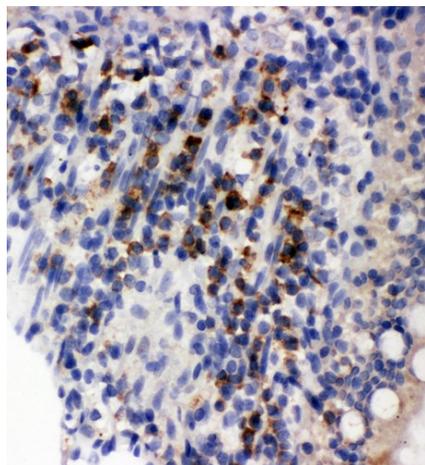
Monoubiquitinated on Lys-519 by E3 ubiquitin-protein ligase TRIM33. Monoubiquitination hampers its ability to form a stable complex with activated SMAD2/3 resulting in inhibition of TGF-beta/BMP signaling cascade. Deubiquitination by USP9X restores its competence to mediate TGF-beta signaling.

Cellular localization

Cytoplasm. Nucleus. Cytoplasmic in the absence of ligand. Migrates to the nucleus when

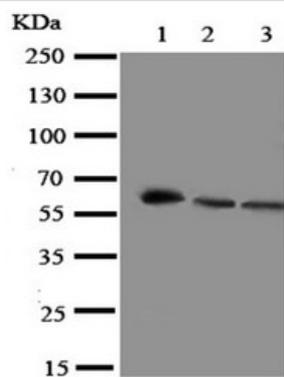
complexed with R-SMAD.

Images



Immunohistochemical analysis of paraffin-embedded Rat Intestine Tissue labeling Smad4 with ab191026 at 1 $\mu\text{g/ml}$.

Immunohistochemistry (Formalin/PFA-fixed paraffin-embedded sections) - Anti-Smad4 antibody - N-terminal (ab191026)



All lanes : Anti-Smad4 antibody - N-terminal (ab191026) at 0.5 $\mu\text{g/ml}$

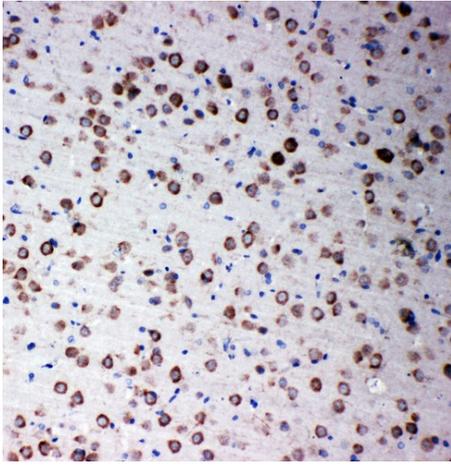
Lane 1 : Rat Skeletal Muscle Tissue Lysate

Lane 2 : A549 Cell Lysate

Lane 3 : U87 Cell Lysate

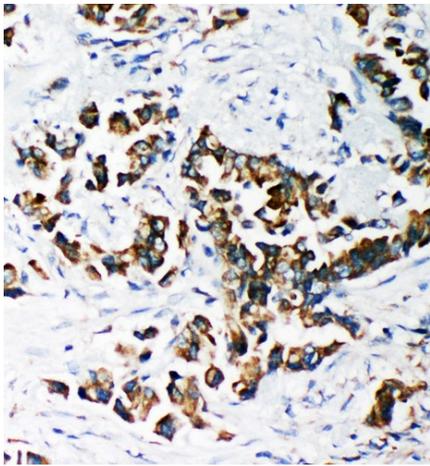
Predicted band size: 60 kDa

Western blot - Anti-Smad4 antibody - N-terminal (ab191026)



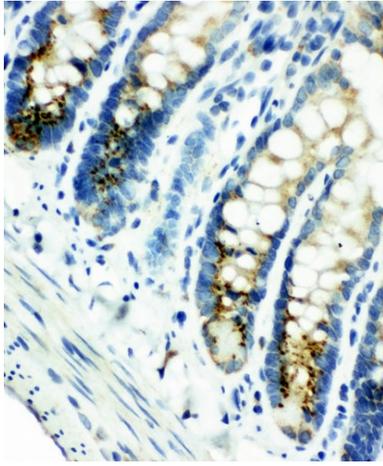
Immunohistochemical analysis of paraffin-embedded Rat Brain
Tissue labeling Smad4 with ab191026 at 1 $\mu\text{g/ml}$.

Immunohistochemistry (Formalin/PFA-fixed paraffin-
embedded sections) - Anti-Smad4 antibody - N-
terminal (ab191026)



Immunohistochemical analysis of paraffin-embedded Human Lung
Cancer Tissue labeling Smad4 with ab191026 at 1 $\mu\text{g/ml}$.

Immunohistochemistry (Formalin/PFA-fixed paraffin-
embedded sections) - Anti-Smad4 antibody - N-
terminal (ab191026)



Immunohistochemical analysis of paraffin-embedded Rat Intestine
Tissue labeling Smad4 with ab191026 at 1 µg/ml.

Immunohistochemistry (Formalin/PFA-fixed paraffin-embedded sections) - Anti-Smad4 antibody - N-terminal (ab191026)

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