

Recombinant Human Smad4 protein ab81764

3 References 2 Images

Description

Product name	Recombinant Human Smad4 protein
Purity	> 90 % SDS-PAGE.
Expression system	Escherichia coli
Protein length	Full length protein
Animal free	No
Nature	Recombinant
Species	Human

Sequence

MGSSHHHHHH SSGLVPRGSH MDNMSITNTP
TSNDACLSIV HSLMCHRQGG ESETFAKRAI
ESLVKKLKEK KDELDSLITA ITNGAHP SK CVTIQRTLDG
RLQVAGRKGF PHVIYARLWR WPD LHKNELK
HVKYCQYAFD LKCD SVCVNP YHYERVVSPG
IDLSGLTLQS NAPSSMMVKD EYVHDFEGQP
SLSTEGHSIQ TIQHPPSNRA STETYSTPAL LAPSESNATS
TANFPNIPVA STSQPASILG GSHSEGLLQI
ASGPQPGQQQ NGFTGQPATY HHNSTTTWTG
SRTAPYTPNL PHHQNGHLQH HPPMPPHPGH
YWPVHNELAF QPPISNHPAP EYWCSIAYPE
MDVQVGETFK VPSSCPITV DGYVDPSGGD
RFCLGQLSNV HRTEAIERAR LHIGKGVQLE
CKGEGDVWVR CLSDHAVFVQ SYLDREAGR
APGDAVHKIY PSAYIKVFDL RQCHRQMQQQ
AATAQAAAAA QAAAVAGNIP GPGSVGGIAP AISLSAAAGI
GVDDLRRLCI LRMSFVKGWG PDYPRQSIKE
TPCWIEIHLH RALQLLDEV LHTMPIADPQP LD

Specifications

Our **Abpromise guarantee** covers the use of **ab81764** in the following tested applications.

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

Applications	SDS-PAGE
	Western blot

Form Liquid

Preparation and Storage

Stability and Storage Shipped at 4°C. Upon delivery aliquot and store at -20°C or -80°C. Avoid repeated freeze / thaw cycles.

pH: 8.00

Constituents: 0.316% Tris HCl, 20% Glycerol (glycerin, glycerine)

General Info

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.

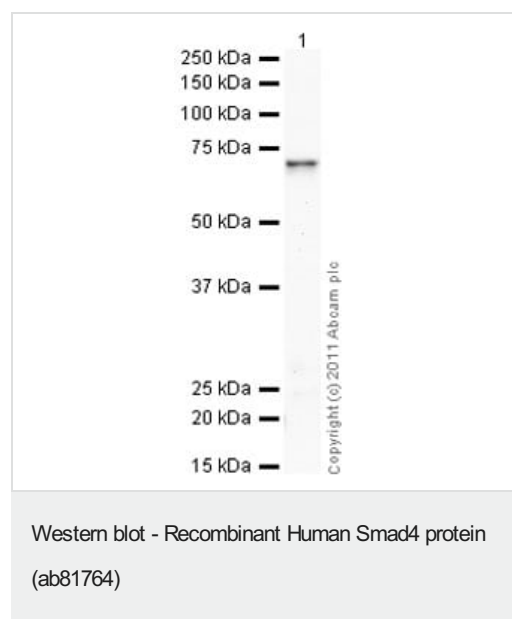
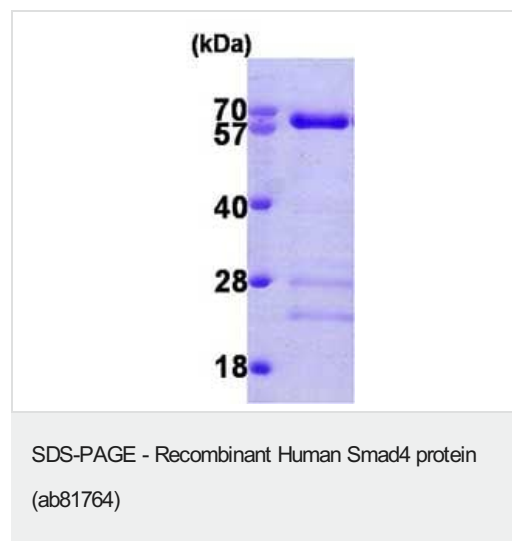
The MH2 domain is required for both homomeric and heteromeric interactions and for transcriptional regulation. Sufficient for nuclear import.

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



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