# abcam

### **Product datasheet**

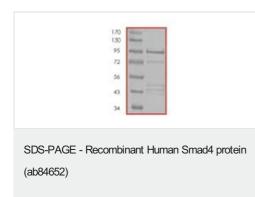
## Recombinant Human Smad4 protein ab84652

1 图**像** 

描述	
产品名称	重组人Smad4蛋白
纯 <b>度</b>	> 80 % Densitometry.
<b>表达系</b> 统	Escherichia coli
蛋白长度	Full length protein
无动 <b>物成分</b>	No
性质	Recombinant
种属	Human
技术指标	
Our Abpromise guarant	tee covers the use of ab84652 in the following tested applications.
The application notes incl	lude recommended starting dilutions; optimal dilutions/concentrations should be determined by the end user.
应用	Western blot
	SDS-PAGE
形式	Liquid
制备和贮存	
稳定性和存储	Shipped on dry ice. Upon delivery aliquot and store at -80°C. Avoid freeze / thaw cycles.
	pH: 7.50
	Constituents: 0.00174% PMSF, 0.00385% DTT, 0.79% Tris HCl, 25% Glycerol (glycerin, glycerine), 0.87% Sodium chloride
常规信息	
功能	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 syngernistic transcriptional activity in response to TGF-beta. May act as a tumor suppressor.
疾病相关	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].
序列相似性	Belongs to the dwarfin/SMAD family. Contains 1 MH1 (MAD homology 1) domain. Contains 1 MH2 (MAD homology 2) domain.
结 <b>构域</b>	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.
<b>翻译后修</b> 饰	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. Deubiqitination by USP9X restores its competence to mediate TGF-beta signaling.
细胞定位	Cytoplasm. Nucleus. Cytoplasmic in the absence of ligand. Migrates to the nucleus when complexed with R-SMAD.

#### 图片



SDS-PAGE showing ab84652 at approximately 95kDa.

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