

**SMAD4 Antibody (C-term) Blocking Peptide**  
**Synthetic peptide**  
**Catalog # BP7753b****Specification****SMAD4 Antibody (C-term) Blocking Peptide - Product Information**Primary Accession [Q13485](#)**SMAD4 Antibody (C-term) Blocking Peptide - Additional Information****Gene ID** 4089**Other Names**

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, SMAD4, DPC4, MADH4

**Target/Specificity**

The synthetic peptide sequence used to generate the antibody [AP7753b](/products/AP7753b) was selected from the C-term region of human SMAD4. A 10 to 100 fold molar excess to antibody is recommended. Precise conditions should be optimized for a particular assay.

**Format**

Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

**Storage**

Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

**Precautions**

This product is for research use only. Not for use in diagnostic or therapeutic procedures.

**SMAD4 Antibody (C-term) Blocking Peptide - Protein Information****SMAD4 Antibody (C-term) Blocking Peptide - Background**

SMAD4 is the common SMAD (co-SMAD) mediator of signal transduction by TGF-beta (transforming growth factor). It promotes binding of the SMAD2/SMAD4/FAST-1 complex to DNA and provides an activation function required for SMAD1 or SMAD2 to stimulate transcription. It may act as a tumor suppressor.

**SMAD4 Antibody (C-term) Blocking Peptide - References**

Sekiya, T., et al., Biochem. Biophys. Res. Commun. 320(3):680-684 (2004). Horvath, L.G., et al., Prostate 59(3):234-242 (2004). Li, L., et al., Mol. Cell. Biol. 24(2):856-864 (2004). Wan, M., et al., J. Biol. Chem. 279(15):14484-14487 (2004). Maru, D., et al., Oncogene 23(3):859-864 (2004).

**Name** SMAD4**Synonyms** DPC4, MADH4**Function**

In muscle physiology, plays a central role in the balance between atrophy and hypertrophy. When recruited by MSTN, promotes atrophy response via phosphorylated SMAD2/4. MSTN decrease causes SMAD4 release and subsequent recruitment by the BMP pathway to promote hypertrophy via phosphorylated SMAD1/5/8. Acts synergistically with SMAD1 and YY1 in bone morphogenetic protein (BMP)-mediated cardiac- specific gene expression. Binds to SMAD binding elements (SBEs) (5'- GTCT/AGAC-3') within BMP response element (BMPRE) of cardiac activating regions (By similarity). 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 (PubMed:<a href="http://www.uniprot.org/citations/25514493" target="\_blank">25514493</a>). 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. Positively regulates PDPK1 kinase activity by stimulating its dissociation from the 14-3-3 protein YWHAQ which acts as a negative regulator.

**Cellular Location**

Cytoplasm. Nucleus Note=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)

**SMAD4 Antibody (C-term) Blocking Peptide - Protocols**

Provided below are standard protocols that you may find useful for product applications.

- [Blocking Peptides](#)