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SMAD4 Antibody
Purified Mouse Monoclonal Antibody
Catalog # AO1444a

Specification

SMAD4 Antibody - Product Information

Application	**WB, IF, IHC, FC**
Primary Accession	Q13485
Reactivity	**Human**
Host	**Mouse**
Clonality	**Monoclonal**
Isotype	**IgG1**
Calculated MW	**65kDa KDa**

Description
Common mediator of signal transduction by TGF-beta (transforming growth factor) superfamily; SMAD4 is the common SMAD (co-SMAD). Promotes binding of the SMAD2/SMAD4/FAST-1 complex to DNA and provides an activation function required for SMAD1 or SMAD2 to stimulate transcription. May act as a tumor suppressor Mutations or deletions in this gene have been shown to result in pancreatic cancer, juvenile polyposis syndrome, and hereditary hemorrhagic telangiectasia syndrome.

Immunogen
Purified recombinant fragment of human SMAD4 expressed in E. Coli.

Formulation
Ascitic fluid containing 0.03% sodium azide.

SMAD4 Antibody - 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

Dilution
WB~~1/500 - 1/2000
IF~~1/200 - 1/1000
IHC~~1/500 - 1/2000
FC~~1/200 - 1/400

Storage
Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.

Precautions
SMAD4 Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

SMAD4 Antibody - Protein Information

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:25514493). 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 - Protocols

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

- Western Blot
- Blocking Peptides
- Dot Blot
- Immunohistochemistry
- Immunofluorescence
- Immunoprecipitation
- Flow Cytomety
- Cell Culture

SMAD4 Antibody - Images

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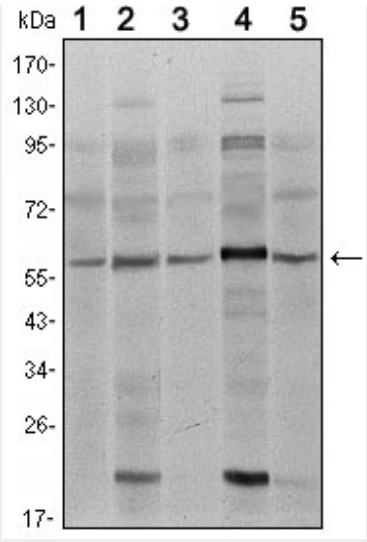


Figure 1: Western blot analysis using SMAD4 mouse mAb against A431 (1), SK-N-SH (2), K562 (3), HepG2 (4) and HUVE12 (5) cell lysate.

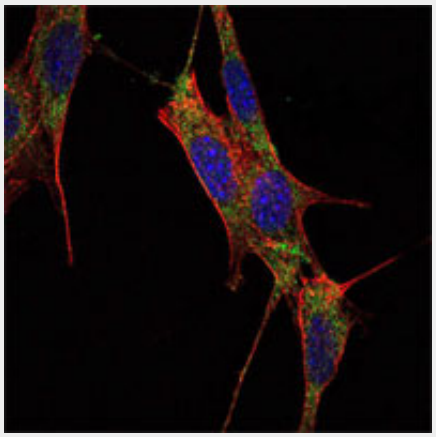


Figure 2: Immunofluorescence analysis of NIH/3T3 cells using SMAD4 mouse mAb (green). Blue: DRAQ5 fluorescent DNA dye. Red: Actin filaments have been labeled with Alexa Fluor-555 phalloidin.

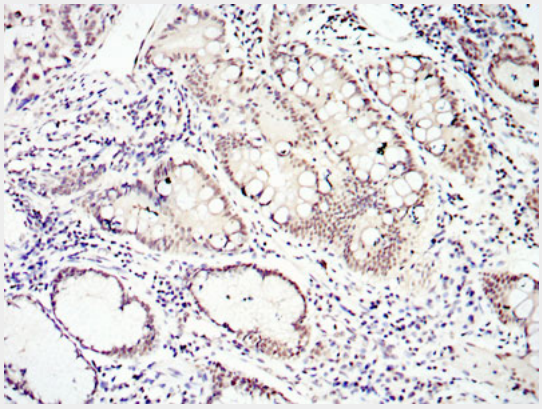


Figure 3: Immunohistochemical analysis of paraffin-embedded lung cancer tissues using SMAD4 mouse mAb with DAB staining.

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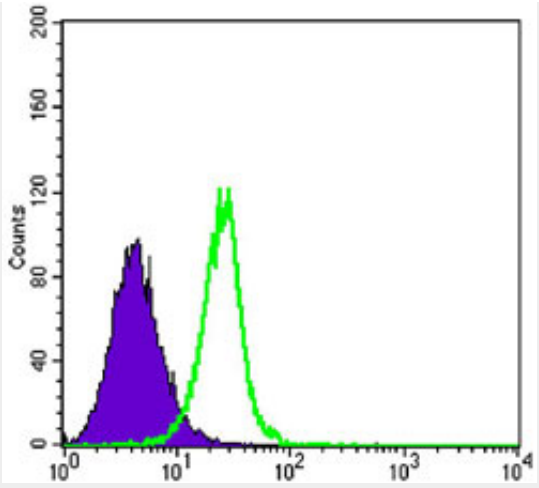


Figure 4: Flow cytometric analysis of K562 cells using SMAD4 mouse mAb (green) and negative control (purple).

SMAD4 Antibody - References

1. Am J Hum Genet. 2009 Nov;85(5):628-42. 2. Mol Endocrinol. 2010 Mar;24(3):540-51.