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SMAD4 Antibody (T277)

Affinity Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP7753a

Product Information

Application	WB, IF, E
Primary Accession	Q13485
Other Accession	O70437, P97471
Reactivity	Human, Rat, Mouse
Predicted	Mouse, Rat
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Clone Names	RB14714
Calculated MW	60439
Antigen Region	255-284

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	This SMAD4 antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 255-284 amino acids from human SMAD4.
Dilution	WB~~1:1000 IF~~1:10~50 E~~Use at an assay dependent concentration.
Format	Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is purified through a protein A column, followed by peptide affinity purification.
Storage	Maintain refrigerated at 2-8°C for up to 2 weeks. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.
Precautions	SMAD4 Antibody (T277) is for research use only and not for use in diagnostic or therapeutic procedures.

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)

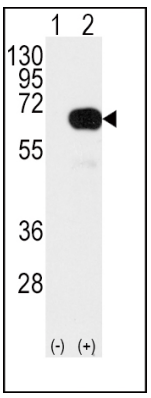
Background

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.

References

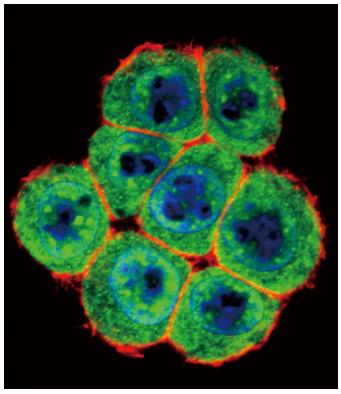
Sekiya, T., et al., Biochem. Biophys. Res. Commun. 320(3):680-684 (2004).
Horvath, L.G., et al., Prostate 59(3):234-242 (2004).
Li, L., et al., Mol. Cell. Biol. 24(2):856-864 (2004).
Wan, M., et al., J. Biol. Chem. 279(15):14484-14487 (2004).
Maru, D., et al., Oncogene 23(3):859-864 (2004).

Images



Western blot analysis of SMAD4 Antibody (T277)(arrow) using rabbit polyclonal SMAD4 Antibody (T277) (Cat.#AP7753a). 293 cell lysates (2 ug/lane) either nontransfected (Lane 1) or transiently transfected with the SMAD4 gene (Lane 2) (Origene Technologies).

Confocal immunofluorescent analysis of SMAD4 Antibody (T277)(Cat#AP7753a) with Hela cell followed by Alexa



Fluor 488-conjugated goat anti-rabbit lgG (green). Actin filaments have been labeled with Alexa Fluor 555 phalloidin (red).DAPI was used to stain the cell nuclear (blue).

Citations

- Resistance to aerobic exercise training causes metabolic dysfunction and reveals novel exercise-regulated signaling networks.