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Anti-Smad4(DPC4) Antibody (Monoclonal, DCS-46)

Catalog # ABO10473

Product Information

Application	WB, ICC
Primary Accession	O70437
Host	Mouse
Isotype	Mouse IgG1
Reactivity	Human
Clonality	Monoclonal
Format	Lyophilized
Description	Mouse IgG monoclonal antibody for Smad4 (DPC4), SMAD family member 4 (SMAD4) detection. Tested with WB, ICC in Human. No cross reactivity with other proteins.
Reconstitution	Add 1ml of PBS buffer will yield a concentration of 100ug/ml.

Additional Information

Gene ID	50554
Other Names	Mothers against decapentaplegic homolog 4, MAD homolog 4, Mothers against DPP homolog 4, SMAD family member 4, SMAD 4, Smad4, Smad4, Madh4
Calculated MW	60469
Application Details	Immunocytochemistry , 1 □g/ml, Human, - Western blot, 2-4 □g/ml, Human
Subcellular Localization	Cytoplasm . Nucleus . In the cytoplasm in the absence of ligand. Migration to the nucleus when complexed with R-SMAD. PDPK1 prevents its nuclear translocation. .
Source	Eukaryota
Protein Name	Mothers against decapentaplegic homolog 4
Contents	Mouse ascites fluid, 1.2% sodium acetate, 2mg BSA, with 0.01mg NaN3 as preservative.
Clone Names	DCS-46
Immunogen	Recombinant human Smad4(DPC4).
Purification	Ascites
Cross Reactivity	No cross reactivity with other proteins

Storage	At -20˚C for one year. After r˚Constitution, at 4˚C for one month. It˚Can also be aliquotted and stored frozen at -20˚C for a longer time.Avoid repeated freezing and thawing.
Sequence Similarities	Belongs to the dwarfin/SMAD family.

Protein Information

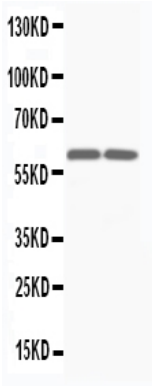
Name	Smad4 {ECO:0000312|RGD:3033}
Synonyms	Madh4
Function	Common SMAD (co-SMAD) is the coactivator and mediator of signal transduction by TGF-beta (transforming growth factor). Component of the heterotrimeric SMAD2/SMAD3-SMAD4 complex that forms in the nucleus and is required for the TGF-mediated signaling. 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. 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. 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. 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 (By similarity).
Cellular Location	Cytoplasm {ECO:0000250|UniProtKB:Q13485}. Nucleus {ECO:0000250|UniProtKB:Q13485}. Note=In the cytoplasm in the absence of ligand. Migration to the nucleus when complexed with R-SMAD. PDPK1 prevents its nuclear translocation. {ECO:0000250|UniProtKB:Q13485}

Background

SMAD4 plays a pivotal role in signal transduction of the transforming growth factor beta superfamily cytokines by mediating transcriptional activation of target genes. Smad4 signalling in T cells is required for suppression of gastrointestinal cancer. Mutational inactivation of SMAD4 causes TGF-beta unresponsiveness and gave a basis for understanding the physiologic role of this gene in tumorigenesis. Mutations in DPC4(SMAD4) cause juvenile polyposis syndrome, but only account for a minority of cases.

Images

Anti-Smad4(DPC4) antibody (monoclonal), ABO10473, Western blottingLane 1: Rat Brain Tissue LysateLane 2: Rat Brain Tissue Lysate



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