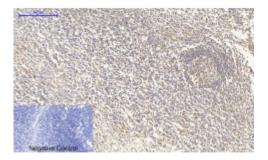


Anti-Smad4 antibody





Description Smad4 is a protein encoded by the SMAD4 gene which is approximately

60,4 kDa. Smad4 is localised to the cytoplasm and nucleus. It is involved in transcriptional activity of SMAD2/SMAD3-SMAD4 heterotrimer, the TGF-beta pathway, signalling by BMP and the cell cycle. It is phosphorylated and activated by transmembrane serine-threonine receptor kinases in response to TGF-beta signalling. It forms homomeric complexes and heteromeric complexes with other activated Smad proteins, which then accumulate in the nucleus and regulate the transcription of target genes. Smad4 is expressed in the cells of the nervous system, liver, muscle, intestine and pancreas. Mutations in the SMAD4 gene may result in pancreatic cancer and juvenile Polyposis syndrome. STJ95702 was affinity-purified from rabbit antiserum by affinity-chromatography using epitope-specific immunogen. This polyclonal antibody detects endogenous

Model STJ95702

Host Rabbit

Reactivity Human, Mouse, Rat, Simian

Applications ELISA, IF, IHC, WB

Immunogen Synthesized peptide derived from human Smad4

levels of Smad4 protein.

Immunogen Region 40-120 aa, N-terminal

Gene ID 4089

Gene Symbol SMAD4

Dilution range WB 1:500-1:2000IHC 1:100-1:300IF 1:200-1:1000ELISA 1:10000

Specificity Smad4 Polyclonal Antibody detects endogenous levels of Smad4 protein.

Purification The antibody was affinity-purified from rabbit antiserum by affinity-

chromatography using epitope-specific immunogen.

Note For Research Use Only (RUO).

Protein Name 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

Molecular Weight 60 kDa

Clonality Polyclonal

Conjugation Unconjugated

Isotype IgG

Formulation Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.

Concentration 1 mg/ml

Storage Instruction Store at -20°C, and avoid repeat freeze-thaw cycles.

Database Links <u>HGNC:6770OMIM:114500</u>

Alternative 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

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 . 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

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.

Sequence and Domain Family The MH1 domain is required for DNA binding.; The MH2 domain is required

for both homomeric and heteromeric interactions and for transcriptional

function required for SMAD1 or SMAD2 to stimulate transcription.

regulation. Sufficient for nuclear import.

Cellular Localization Cytoplasm Nucleus. Cytoplasmic in the absence of ligand. Migrates to the

nucleus when complexed with R-SMAD . PDPK1 prevents its nuclear

translocation in response to TGF-beta.

Post-translational Phosphorylated by PDPK1. Monoubiquitinated on Lys-519 by E3 ubiquitin-

Modifications

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. Deubiquitination by USP9X restores its competence to mediate TGF-beta signaling.

St John's Laboratory Ltd

 \mathbf{F} +44 (0)207 681 2580

T +44 (0)208 223 3081

W http://www.stjohnslabs.com/ E info@stjohnslabs.com