

Phospho-Smad3 (S423 + S425) Antibody

Rabbit mAb

Catalog # AP90197

Product Information

Application	WB, IHC, IF, ICC, IHF
Primary Accession	P84022
Reactivity	Human, Mouse
Clonality	Monoclonal
Other Names	JV15-2, MAD-3, MADH3, Mad3, Mothers against DPP homolog 3, Mothers against decapentaplegic homolog 3, SMAD 3, Smad 3
Isotype	Rabbit IgG
Host	Rabbit
Calculated MW	48081

Additional Information

Dilution	WB 1:500~1:2000 IHC 1:50~1:200 ICC/IF 1:50~1:200
Purification	Affinity-chromatography
Immunogen	A synthesized peptide derived from human Phospho-Smad3 (S423 + S425)
Description	Receptor-regulated SMAD (R-SMAD) that is an intracellular signal transducer and transcriptional modulator activated by TGF-beta (transforming growth factor) and activin type 1 receptor kinases. Binds the TRE element in the promoter region of many genes that are regulated by TGF-beta and, on formation of the SMAD3/SMAD4 complex, activates transcription.
Storage Condition and Buffer	Rabbit IgG in phosphate buffered saline , pH 7.4, 150mM NaCl, 0.02% sodium azide and 50% glycerol. Store at +4°C short term. Store at -20°C long term. Avoid freeze / thaw cycle.

Protein Information

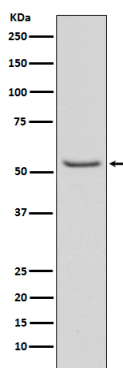
Name	SMAD3 (HGNC:6769)
Synonyms	MADH3
Function	Receptor-regulated SMAD (R-SMAD) that is an intracellular signal transducer and transcriptional modulator activated by TGF-beta (transforming growth factor) and activin type 1 receptor kinases. Binds the TRE element in the promoter region of many genes that are regulated by TGF-beta and, on formation of the SMAD3/SMAD4 complex, activates transcription. Also can form a SMAD3/SMAD4/JUN/FOS complex at the AP- 1/SMAD site to regulate TGF-beta-mediated transcription. Has an inhibitory effect on wound healing probably by modulating both growth and migration of primary keratinocytes and by altering the TGF-mediated chemotaxis of monocytes. This effect on wound healing appears to be hormone-sensitive. Regulator of chondrogenesis and osteogenesis and inhibits early healing of bone fractures.

Positively regulates PDPK1 kinase activity by stimulating its dissociation from the 14-3-3 protein YWHAQ which acts as a negative regulator.

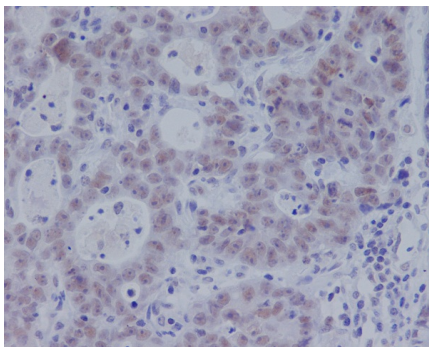
Cellular Location

Cytoplasm. Nucleus. Note=Cytoplasmic and nuclear in the absence of TGF-beta. On TGF-beta stimulation, migrates to the nucleus when complexed with SMAD4 (PubMed:15799969, PubMed:21145499). Through the action of the phosphatase PPM1A, released from the SMAD2/SMAD4 complex, and exported out of the nucleus by interaction with RANBP1 (PubMed:16751101, PubMed:19289081). Co-localizes with LEMD3 at the nucleus inner membrane (PubMed:15601644). MAPK-mediated phosphorylation appears to have no effect on nuclear import (PubMed:19218245). PDPK1 prevents its nuclear translocation in response to TGF-beta (PubMed:17327236). Localized mainly to the nucleus in the early stages of embryo development with expression becoming evident in the cytoplasm of the inner cell mass at the blastocyst stage (By similarity) {ECO:0000250 | UniProtKB:Q8BUN5, ECO:0000269 | PubMed:15601644, ECO:0000269 | PubMed:15799969, ECO:0000269 | PubMed:16751101, ECO:0000269 | PubMed:17327236, ECO:0000269 | PubMed:19218245, ECO:0000269 | PubMed:19289081, ECO:0000269 | PubMed:21145499}

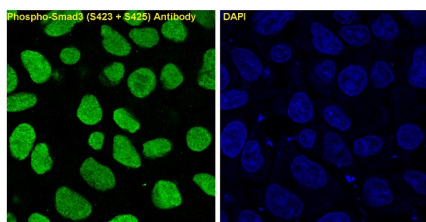
Images



Western blot analysis of Phospho-Smad3 (S423/S425) expression in A549 cell lysate treated with TGF- β 1.



Immunohistochemical analysis of paraffin-embedded human gastric adenocarcinoma, using Phospho-Smad3 (S423 + S425) Antibody.



Immunofluorescent analysis of A549 cells treated with TGF β , using Phospho-Smad3 (S423 + S425) Antibody.