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SMAD3 Antibody (Center) Blocking peptide
Synthetic peptide
Catalog # BP13540c

Specification

SMAD3 Antibody (Center) Blocking peptide - Product Information

Primary Accession P84022

SMAD3 Antibody (Center) Blocking peptide - Additional Information

Gene ID 4088

Other Names
Mothers against decapentaplegic homolog 3, MAD homolog 3, Mad3, Mothers against DPP homolog 3, hMAD-3, JV15-2, SMAD family member 3, SMAD 3, Smad3, hSMAD3, SMAD3, MADH3

Target/Specificity
The synthetic peptide sequence used to generate the antibody AP13540c was selected from the Center region of SMAD3. A 10 to 100 fold molar excess to antibody is recommended. Precise conditions should be optimized for a particular assay.

Format
Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

Storage
Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

Precautions
This product is for research use only. Not for use in diagnostic or therapeutic procedures.

SMAD3 Antibody (Center) Blocking peptide - Protein Information

Name SMAD3

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}

SMAD3 Antibody (Center) Blocking peptide - Protocols

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

- Blocking Peptides

SMAD3 Antibody (Center) Blocking peptide - Images

SMAD3 Antibody (Center) Blocking peptide - Background

The protein encoded by this gene belongs to the SMAD, afamily of proteins similar to the gene products of the Drosophilagene 'mothers against decapentaplegic' (Mad) and the C. elegansgene Sma. SMAD proteins are signal transducers and transcriptionalmodulators that mediate multiple signaling pathways. This proteinfunctions as a transcriptional modulator activated by transforminggrowth factor-beta and is thought to play a role in the regulationof carcinogenesis.

SMAD3 Antibody (Center) Blocking peptide - References

Ge, Q., et al. J. Cell. Physiol. 225(3):846-854(2010)Lee, J., et al. J. Biol. Chem. 285(34):26618-26627(2010)Roder, C., et al. Childs Nerv Syst (2010) In press :Valdes, A.M., et al. Arthritis Rheum. 62(8):2347-2352(2010)Jugessur, A., et al. PLoS ONE 5 (7), E11493 (2010) :