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SMAD3 Antibody
Purified Mouse Monoclonal Antibody
Catalog # AO1425a

Specification

SMAD3 Antibody - Product Information

Application	**WB, IF**
Primary Accession	P84022
Reactivity	**Human, Mouse**
Host	**Mouse**
Clonality	**Monoclonal**
Isotype	**IgG1**
Calculated MW	**48kDa KDa**

Description
SMAD proteins are signal transducers and transcriptional modulators that mediate multiple signaling pathways. This protein functions as a transcriptional modulator activated by transforming growth factor-beta and is thought to play a role in the regulation of carcinogenesis.

Immunogen
Purified recombinant fragment of human SMAD3 expressed in E. Coli.

Formulation
Ascitic fluid containing 0.03% sodium azide.

SMAD3 Antibody - 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

Dilution
WB~~1/500 - 1/2000
IF~~1/200 - 1/1000

Storage
Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.

Precautions
SMAD3 Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

SMAD3 Antibody - 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 - Protocols

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

- Western Blot
- Blocking Peptides
- Dot Blot
- Immunohistochemistry
- Immunofluorescence
- Immunoprecipitation
- Flow Cytomety
- Cell Culture

SMAD3 Antibody - Images

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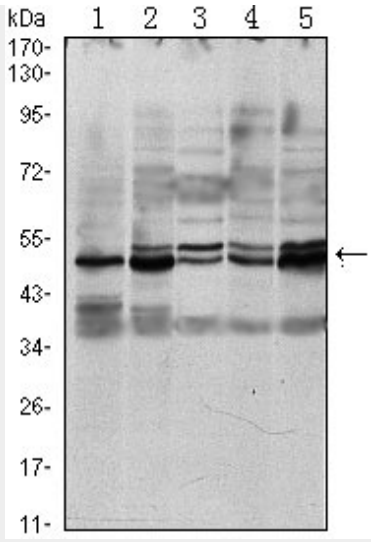

Figure 1: Western blot analysis using SMAD3 mouse mAb against A549 (1), Hela (2), Jurkat (3), PC-2 (4) and NIH/3T3 (5) cell lysate.

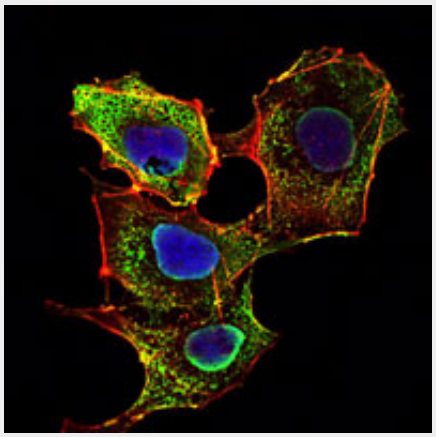

Figure 2: Immunofluorescence analysis of NIH/3T3 cells using SMAD3 mouse mAb (green). Blue: DRAQ5 fluorescent DNA dye. Red: Actin filaments have been labeled with Alexa Fluor-555 phalloidin.

SMAD3 Antibody - References

1. PLoS One. 2009 Sep 21;4(9):e7091. 2. Heart Rhythm. 2009 Dec;6(12):1745-50. 3. Am J Pathol. 2010 Mar;176(3):1139-47.