

SMAD3 Antibody (Center D201)

Affinity Purified Rabbit Polyclonal Antibody (Pab)

Catalog # AP6267a

Product Information

Application	WB, IF, FC, E
Primary Accession	P84022
Other Accession	P84025 , P84024 , Q8BUN5 , P84023
Reactivity	Mouse, Rat, Human
Predicted	Chicken, Pig, Rat
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Clone Names	RB07301
Calculated MW	48081
Antigen Region	186-215

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	This SMAD3 antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 186-215 amino acids from the Central region of human SMAD3.
Dilution	WB~~1:1000 IF~~1:10~50 FC~~1:10~50 E~~Use at an assay dependent concentration.
Format	Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is purified through a protein A column, followed by peptide affinity purification.
Storage	Maintain refrigerated at 2-8°C for up to 2 weeks. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.
Precautions	SMAD3 Antibody (Center D201) is for research use only and not for use in diagnostic or therapeutic procedures.

Protein Information

Name	SMAD3 (HGNC:6769)
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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:Q8BUNS, 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}

Background

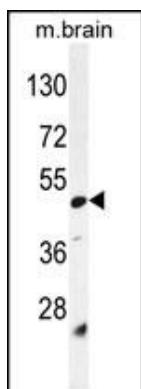
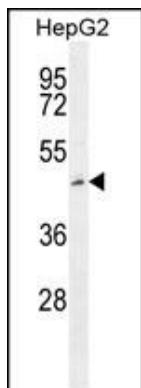
SMAD3, a receptor regulated SMAD (R-SMAD) is a transcriptional modulator activated by TGF-beta (transforming growth factor) and activin type 1 receptor kinase. SMAD3 is estimated to account for at least 80% of all TGF-beta-mediated response. Activated type I receptor phosphorylates receptor-activated SMADS (RSMADS) at their c-terminal two extreme serines in the SSXS motif. The phosphorylated R-SMADS translocate into the nucleus, where they regulate transcription of target genes. The SMAD3 signal transduction appears to be important in the regulation of muscle-specific genes. Loss of SMAD3 is a feature of pediatric T-cell lymphoblastic leukemia, while upregulation of SMAD3 may be responsible for TGF β hyperresponsiveness observed in scleroderma.

References

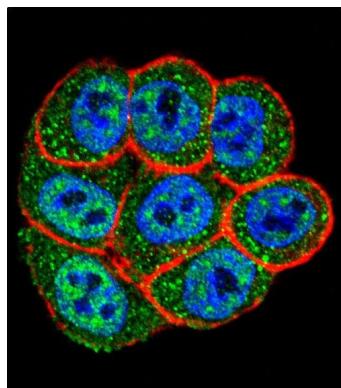
Imoto, S., et al., FEBS Lett. 579(13):2853-2862 (2005). Dubrovska, A., et al., Oncogene 24(14):2289-2297 (2005). Furumatsu, T., et al., J. Biol. Chem. 280(9):8343-8350 (2005). Kobayashi, T., et al., Biochem. Biophys. Res. Commun. 327(2):393-398 (2005). Kamaraju, A.K., et al., J. Biol. Chem. 280(2):1024-1036 (2005).

Images

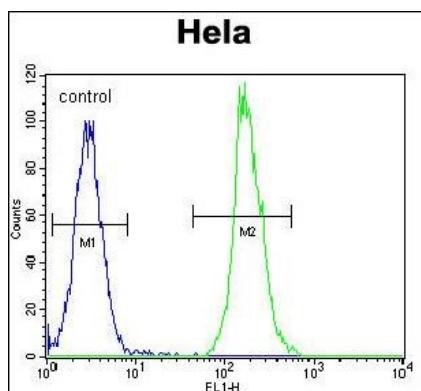
SMAD3 antibody (Center D201)(Cat.#AP6267a) western blot analysis in HepG2 cell line lysates (35ug/lane). This demonstrates the SMAD3 antibody detected the SMAD3 protein (arrow).



SMAD3 antibody (Center D201)(Cat.#AP6267a) western blot analysis in mouse brain tissue lysates (35ug/lane).This demonstrates the SMAD3 antibody detected the SMAD3 protein (arrow).



Confocal immunofluorescent analysis of SMAD3 Antibody (Center D201)(Cat#AP6267a) with HeLa cell followed by Alexa Fluor 488-conjugated goat anti-rabbit IgG (green). Actin filaments have been labeled with Alexa Fluor 555 phalloidin (red).DAPI was used to stain the cell nuclear (blue).



SMAD3 Antibody (Center D201) (Cat. #AP6267a) flow cytometric analysis of HeLa cells (right histogram) compared to a negative control cell (left histogram).FITC-conjugated goat-anti-rabbit secondary antibodies were used for the analysis.

Please note: All products are 'FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC OR THERAPEUTIC PROCEDURES'.