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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)

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}

Background

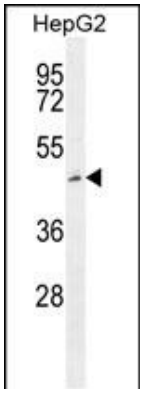
SMAD3, a receptor regulated SMAD (R-SMAD) is a transcriptional modulator activated by TGF-beta (transforming growth factor) and activin type 1 receptor kinase. SMAD3 is estimated to account for at least 80% of all TGF-beta-mediated response. Activated type I receptor phosphorylates receptor-activated 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 rgulation of muscle-specific genes. Loss of SMAD3 is a feature of pediatric T-cell lymphoblastic leukemia, while upregulation of SMAD3 may be responsible for TGFB hyperresponsiveness observed in scleroderma.

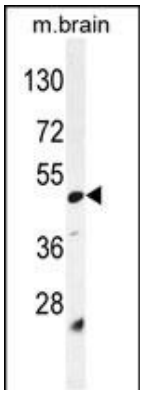
References

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).

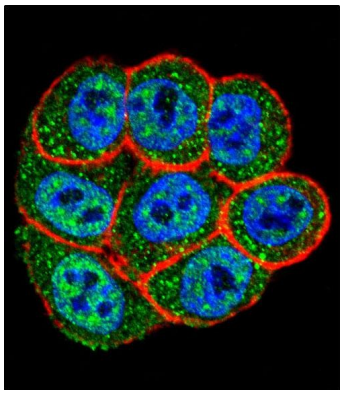
Images

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).

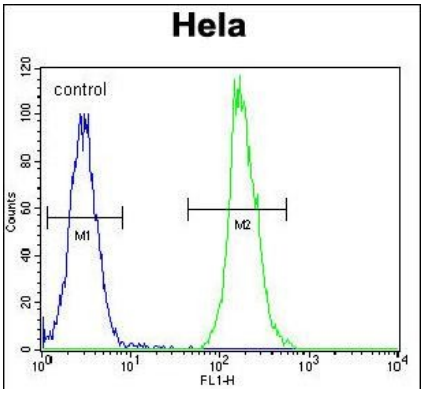




SMAD3 antibody (Center D201)(Cat.#AP6267a) western blot analysis in mouse brain tissue lysates (35ug/lane).This demonstrates the SMAD3 antibody detected the SMAD3 protein (arrow).



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 filamentsl 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'.