

# SMAD3 Antibody (Center N206)

Affinity Purified Rabbit Polyclonal Antibody (Pab)

Catalog # AP6267b

## Product Information

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<b>Application</b>	WB, E
<b>Primary Accession</b>	<a href="#">P84022</a>
<b>Other Accession</b>	<a href="#">P84025</a> , <a href="#">P84024</a> , <a href="#">Q8BUN5</a> , <a href="#">P84023</a>
<b>Reactivity</b>	Human, Mouse
<b>Predicted</b>	Chicken, Pig, Rat
<b>Host</b>	Rabbit
<b>Clonality</b>	Polyclonal
<b>Isotype</b>	Rabbit IgG
<b>Clone Names</b>	RB07306
<b>Calculated MW</b>	48081
<b>Antigen Region</b>	191-220

## Additional Information

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<b>Gene ID</b>	4088
<b>Other Names</b>	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
<b>Target/Specificity</b>	This SMAD3 antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 191-220 amino acids from the Central region of human SMAD3.
<b>Dilution</b>	WB~~1:1000 E~~Use at an assay dependent concentration.
<b>Format</b>	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.
<b>Storage</b>	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.
<b>Precautions</b>	SMAD3 Antibody (Center N206) is for research use only and not for use in diagnostic or therapeutic procedures.

## Protein Information

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<b>Name</b>	SMAD3
<b>Synonyms</b>	MADH3

<b>Function</b>	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.
<b>Cellular Location</b>	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

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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

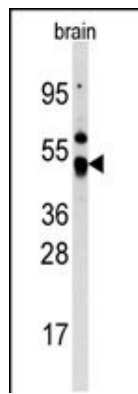
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Kobayashi, T., et al., Biochem. Biophys. Res. Commun. 327(2):393-398 (2005).  
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## Images

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Western blot analysis of SMAD3 Antibody (Center N206) polyclonal antibody(Cat.#AP6267b) in mouse brain tissue lysates (35ug/lane). SMAD3(arrow) was detected using the purified Pab.



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