

# Phospho-SMAD3(S213) Antibody

Affinity Purified Rabbit Polyclonal Antibody (Pab) Catalog # AP3250a

## **Product Information**

Application	WB, IHC-P, E
Primary Accession	<u>P84022</u>
Other Accession	<u>P84025, P84024, Q8BUN5, P84023</u>
Reactivity	Human
Predicted	Mouse, Rat, Pig, Chicken
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Clone Names	RB29526
Calculated MW	48081

### **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 phosphopeptide corresponding to amino acid residues surrounding S213 of human SMAD3.
Dilution	WB~~1:1000 IHC-P~~1:100~500 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	Phospho-SMAD3(S213) Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

#### **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: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:19218245, ECO:0000269   PubMed:19289081, ECO:0000269   PubMed:21145499}

## Background

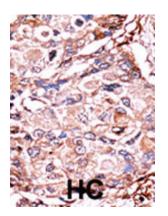
SMAD3, a receptor regulated SMAD (R-SMAD) is a transcriptional modulator activated by TGF-beta (transforming growth factor) and activin type 1 receptor kinase. SMAD3 is estimated to account for at least 80% of all TGF-beta-mediated response. Activated type I receptor phosphorylates receptor-activated SMADS (RSMADS) at their c-terminal two extreme serines in the SSXS motif. The phosphorylated R-SMAD translocate into nucleus, where they regulate transcription of target genes. 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

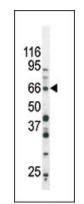
Imoto, S., et al., FEBS Lett. 579(13):2853-2862 (2005). Dubrovska, A., et al., Oncogene 24(14):2289-2297 (2005). Furumatsu, T., et al., J. Biol. Chem. 280(9):8343-8350 (2005). Kobayashi, T., et al., Biochem. Biophys. Res. Commun. 327(2):393-398 (2005). Kamaraju, A.K., et al., J. Biol. Chem. 280(2):1024-1036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

#### Images

Formalin-fixed and paraffin-embedded human cancer tissue reacted with the primary antibody, which was peroxidase-conjugated to the secondary antibody, followed by AEC staining. This data demonstrates the use



of this antibody for immunohistochemistry; clinical relevance has not been evaluated. BC = breast carcinoma; HC = hepatocarcinoma.



The anti-Phospho-SMAD3-S213 Pab (Cat. #AP3250a) is used in Western blot to detect Phospho-SMAD3-S213 in Ramos tissue lysate

# Citations

- Tripartite motif protein 52 (TRIM52) promoted fibrosis in LX-2 cells through PPM1A-mediated Smad2/3 pathway.
  Asiaticoside hinders the invasive growth of keloid fibroblasts through inhibition of the GDF-9/MAPK/Smad pathway.

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