10320 Camino Santa Fe, Suite G
San Diego, CA 92121
Tel: 858.875.1900 Fax: 858.875.1999

Phospho-SMAD3(S213) Antibody
Affinity Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP3250a

Specification

Phospho-SMAD3(S213) Antibody - Product Information

Application	**WB, IHC-P,E**
Primary Accession	P84022
Other Accession	P84025, P84024, Q8BUN5, P84023
Reactivity	**Human**
Predicted	**Chicken, Mouse, Pig, Rat**
Host	**Rabbit**
Clonality	**Polyclonal**
Isotype	**Rabbit IgG**

Phospho-SMAD3(S213) 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

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~~1:50~100
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.

Phospho-SMAD3(S213) 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}

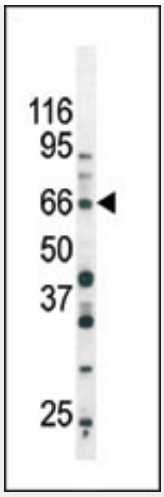
Phospho-SMAD3(S213) Antibody - Protocols

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

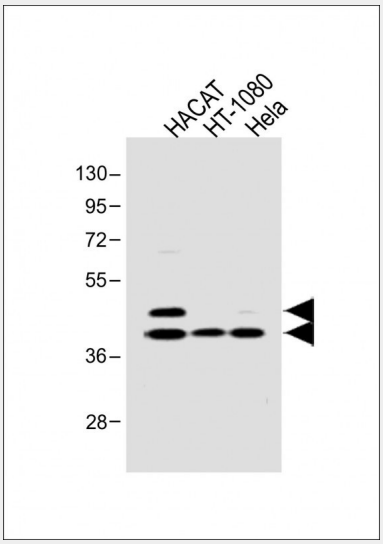
- Western Blot
- Blocking Peptides
- Dot Blot
- Immunohistochemistry
- Immunofluorescence
- Immunoprecipitation
- Flow Cytomety
- Cell Culture

Phospho-SMAD3(S213) Antibody - Images

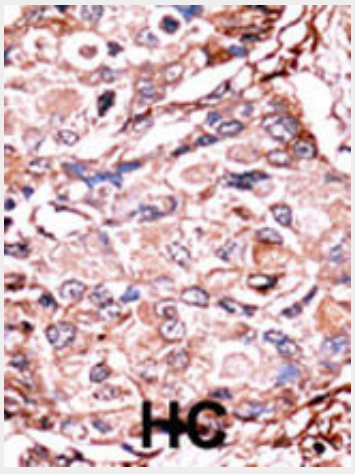
10320 Camino Santa Fe, Suite G
San Diego, CA 92121
Tel: 858.875.1900 Fax: 858.875.1999



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



All lanes : Anti-SMAD3(S213) Antibody at 1:1000 dilution Lane 1: HACAT whole cell lysate Lane 2: HT-1080 whole cell lysate Lane 3: Hela whole cell lysate Lysates/proteins at 20 µg per lane. Secondary Goat Anti-Rabbit IgG, (H+L), Peroxidase conjugated at 1/10000 dilution. Predicted band size : 48, 43 kDa Blocking/Dilution buffer: 5% NFDM/TBST.



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.

Phospho-SMAD3(S213) Antibody - 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.

Phospho-SMAD3(S213) Antibody - 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).

Phospho-SMAD3(S213) Antibody - 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.