

Phospho-SMAD3(S213) Antibody Blocking peptide
Synthetic peptide
Catalog # BP3250a**Specification**

Phospho-SMAD3(S213) Antibody Blocking peptide - Product InformationPrimary Accession [P84022](#)**Phospho-SMAD3(S213) Antibody Blocking peptide - 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

The synthetic peptide sequence used to generate the antibody [AP3250a](/product/products/AP3250a) was selected from the region of human Phospho-SMAD3-S213. A 10 to 100 fold molar excess to antibody is recommended. Precise conditions should be optimized for a particular assay.

Format

Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

Storage

Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

Precautions

This product is for research use only. Not for use in diagnostic or therapeutic procedures.

Phospho-SMAD3(S213) Antibody Blocking peptide - 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 Blocking peptide - Protocols

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

- [Blocking Peptides](#)

Phospho-SMAD3(S213) Antibody Blocking peptide - Images

Phospho-SMAD3(S213) Antibody Blocking peptide - 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 Blocking peptide - References

Imoto, S., et al., FEBS Lett. 579(13):2853-2862 (2005).Dubrovskaya, 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).