

# Smad3 (Phospho-Ser213) Antibody

Catalog # AP68150

## Product Information

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<b>Application</b>	WB, E, IHC-P
<b>Primary Accession</b>	<a href="#">P84022</a>
<b>Reactivity</b>	Human, Mouse, Rat
<b>Host</b>	Rabbit
<b>Clonality</b>	Polyclonal
<b>Calculated MW</b>	48081

## 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)
<b>Dilution</b>	WB~~WB 1:500-2000, ELISA 1:10000-20000 E~~N/A IHC-P~~N/A
<b>Format</b>	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.09% (W/V) sodium azide.
<b>Storage Conditions</b>	-20°C

## Protein Information

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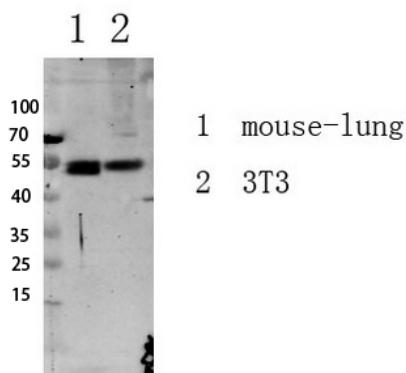
<b>Name</b>	SMAD3 ( <a href="#">HGNC:6769</a> )
<b>Synonyms</b>	MADH3
<b>Function</b>	Receptor-regulated SMAD (R-SMAD) that is an intracellular signal transducer and transcriptional modulator activated by TGF-beta (transforming growth factor) and activin type 1 receptor kinases. Binds the TRE element 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

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.

## Images



Western blot analysis of various lysate, antibody was diluted at 1000. Secondary antibody was diluted at 1:20000

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