

Anti-SMAD3 (pS204) Antibody
Rabbit polyclonal antibody to SMAD3 (pS204)
Catalog # AP59615**Specification**

Anti-SMAD3 (pS204) Antibody - Product Information

Application	WB, IP, IF/IC
Primary Accession	P84022
Other Accession	Q8BUN5
Reactivity	Human, Mouse, Rat, Monkey, Pig, Chicken, SARS
Host	Rabbit
Clonality	Polyclonal
Calculated MW	48081

Anti-SMAD3 (pS204) Antibody - Additional Information**Gene ID** 4088**Other Names**

MADH3; 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

Target/Specificity

KLH-conjugated synthetic peptide encompassing a sequence within the center region of human SMAD3. The exact sequence is proprietary.

Dilution

WB~~WB (1/500 - 1/1000), IF/IC (1/100 - 1/500), IP (1/10 - 1/100)

IP~~N/A

IF/IC~~N/A

Format

Liquid in 0.42% Potassium phosphate, 0.87% Sodium chloride, pH 7.3, 30% glycerol, and 0.09% (W/V) sodium azide.

Storage

Store at -20 °C. Stable for 12 months from date of receipt

Anti-SMAD3 (pS204) Antibody - Protein Information**Name** SMAD3 ([HGNC:6769](#))**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

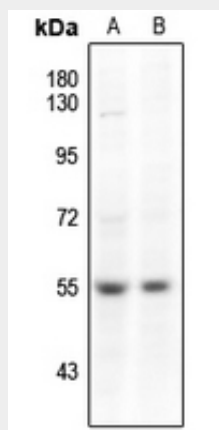
Cytoplasm. Nucleus. Note=Cytoplasmic and nuclear in the absence of TGF-beta. On TGF-beta stimulation, migrates to the nucleus when complexed with SMAD4 (PubMed:15799969, PubMed:21145499). Through the action of the phosphatase PPM1A, released from the SMAD2/SMAD4 complex, and exported out of the nucleus 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}

Anti-SMAD3 (pS204) Antibody - Protocols

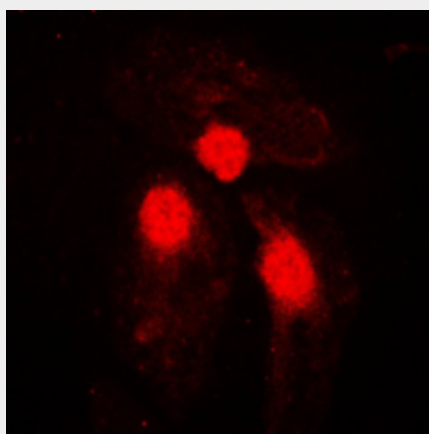
Provided below are standard protocols that you may find useful for product applications.

- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

Anti-SMAD3 (pS204) Antibody - Images



Western blot analysis of SMAD3 (pS204) expression in HCT116 (A), A549 (B) whole cell lysates.



Immunofluorescent analysis of SMAD3 (pS204) staining in HeLa cells. Formalin-fixed cells were permeabilized with 0.1% Triton X-100 in TBS for 5-10 minutes and blocked with 3% BSA-PBS for 30 minutes at room temperature. Cells were probed with the primary antibody in 3% BSA-PBS and incubated overnight at 4 °C in a humidified chamber. Cells were washed with PBST and incubated with a DyLight 594-conjugated secondary antibody (red) in PBS at room temperature in the dark.

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