

ARG42807 anti-Smad 3 phospho (Ser423 / Ser425) antibody

Package: 100 μl Store at: -20°C

Summary

Product Description	Rabbit Polyclonal antibody recognizes Smad 3 phospho (Ser423 / Ser425)
Tested Reactivity	Hu, Ms
Tested Application	ICC/IF, IHC-P, WB
Host	Rabbit
Clonality	Polyclonal
Isotype	IgG
Target Name	Smad 3
Species	Human
Immunogen	Phosphospecific peptide around Ser423 / Ser425 of Human Smad 3.
Conjugation	Un-conjugated
Alternate Names	JV15-2; SMAD 3; MADH3; Mothers against decapentaplegic homolog 3; LDS3; Smad3; HsT17436; Mothers against DPP homolog 3; hSMAD3; Mad3; HSPC193; hMAD-3; SMAD family member 3; LDS1C; MAD homolog 3

Application Instructions

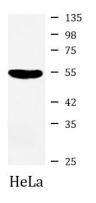
Application table	Application	Dilution	
	ICC/IF	1:50	
	IHC-P	1:50	
	WB	1:1000	
Application Note		* The dilutions indicate recommended starting dilutions and the optimal dilutions or concentrations should be determined by the scientist.	
Positive Control	HeLa		
Observed Size	~ 55 kDa		

Properties

Form	Liquid
Purification	Affinity purified.
Buffer	50 mM Tris-Glycine (pH 7.4), 150 mM NaCl, 0.01% Sodium azide, 40% Glycerol and 0.05% BSA.
Preservative	0.01% Sodium azide
Stabilizer	40% Glycerol and 0.05% BSA
Concentration	Batch dependent

Bioinformation

Gene Symbol	SMAD3	
Gene Full Name	SMAD family member 3	
Background	The SMAD family of proteins are a group of intracellular signal transducer proteins similar to the gene products of the Drosophila gene 'mothers against decapentaplegic' (Mad) and the C. elegans gene Sma. The SMAD3 protein functions in the transforming growth factor-beta signaling pathway, and transmits signals from the cell surface to the nucleus, regulating gene activity and cell proliferation. It also functions as a tumor suppressor. Mutations in this gene are associated with aneurysms-osteoarthritis syndrome and Loeys-Dietz Syndrome 3. [provided by RefSeq, Nov 2019]	
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. [UniProt]	
Calculated Mw	48 kDa	
РТМ	Phosphorylated on serine and threonine residues. Enhanced phosphorylation in the linker region on Thr-179, Ser-204 and Ser-208 on EGF and TGF-beta treatment. Ser-208 is the main site of MAPK- mediated phosphorylation. CDK-mediated phosphorylation occurs in a cell-cycle dependent manner and inhibits both the transcriptional activity and antiproliferative functions of SMAD3. This phosphorylation is inhibited by flavopiridol. Maximum phosphorylation at the G(1)/S junction. Also phosphorylated on serine residues in the C-terminal SXS motif by TGFBR1 and ACVR1. TGFBR1-mediated phosphorylation at these C-terminal sites is required for interaction with SMAD4, nuclear location and transactivational activity, and appears to be a prerequisite for the TGF-beta mediated phosphorylation in the linker region. Dephosphorylated in the C-terminal SXS motif by PPM1A. This dephosphorylation disrupts the interaction with SMAD4, promotes nuclear export and terminates TGF-beta-mediated signaling. Phosphorylation at Ser-418 by CSNK1G2/CK1 promotes ligand- dependent ubiquitination and subsequent proteasome degradation, thus inhibiting SMAD3-mediated TGF-beta responses. Phosphorylated by PDPK1.	
	Acetylation in the nucleus by EP300 in the MH2 domain regulates positively its transcriptional activity and is enhanced by TGF-beta.	
	Poly-ADP-ribosylated by PARP1 and PARP2. ADP-ribosylation negatively regulates SMAD3 transcriptional responses during the course of TGF-beta signaling.	
	Ubiquitinated. Monoubiquitinated, leading to prevent DNA-binding (PubMed:21947082). Deubiquitination by USP15 alleviates inhibition and promotes activation of TGF-beta target genes (PubMed:21947082). Ubiquitinated by RNF111, leading to its degradation: only SMAD3 proteins that are 'in use' are targeted by RNF111, RNF111 playing a key role in activating SMAD3 and regulating its turnover (By similarity). Undergoes STUB1-mediated ubiquitination and degradation (PubMed:24613385). [UniProt]	
Cellular Localization	Cytoplasm. Nucleus. Note=Cytoplasmic and nuclear in the absence of TGF-beta. On TGF-beta stimulation, migrates to the nucleus when complexed with SMAD4. Through the action of the phosphatase PPM1A, released from the SMAD2/4 complex, and exported out of the nucleus by interaction with RANBP1. Co-localizes with LEMD3 at the nucleus inner membrane. MAPK-mediated phosphorylation appears to have no effect on nuclear import. PDPK1 prevents its nuclear translocation in response to TGF-beta. [UniProt]	



ARG42807 anti-Smad 3 phospho (Ser423 / Ser425) antibody WB image

Western blot: HeLa cell lysate stained with ARG42807 anti-Smad 3 phospho (Ser423 / Ser425) antibody at 1:1000 dilution.