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

SMAD1 Blocking Peptide (N-term)

Synthetic peptide
Catalog # BP20141a

Specification

SMAD1 Blocking Peptide (N-term) - Product Information

Primary Accession	Q15797
Other Accession	O54835, Q9JIW5, O15198, Q9R1V3, P97454, Q99717, Q9W7E7, Q56I99, P97588, P70340, Q9I8V2, Q1JQA2, NP_005891.1

SMAD1 Blocking Peptide (N-term) - Additional Information

Gene ID 4086

Other Names
Mothers against decapentaplegic homolog 1, MAD homolog 1, Mothers against DPP homolog 1, JV4-1, Mad-related protein 1, SMAD family member 1, SMAD 1, Smad1, hSMAD1, Transforming growth factor-beta-signaling protein 1, BSP-1, SMAD1, BSP1, MADH1, MADR1

Target/Specificity
The synthetic peptide sequence is selected from aa 14-26 of HUMAN SMAD1

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.

SMAD1 Blocking Peptide (N-term) - Protein Information

Name SMAD1

Synonyms BSP1, MADH1, MADR1

Function
Transcriptional modulator that plays a role in various cellular processes, including embryonic development, cell differentiation, and tissue homeostasis (PubMed:9335504). Upon BMP ligand binding to their receptors at the cell surface, is phosphorylated by activated type I BMP receptors (BMPRIs) and associates with SMAD4 to form an heteromeric complex which translocates into the nucleus acting as transcription factor (PubMed:33667543). In turn, the hetero-trimeric complex recognizes cis-regulatory elements containing Smad Binding Elements

(SBEs) to modulate the outcome of the signaling network (PubMed:33667543). SMAD1/OAZ1/PSMB4 complex mediates the degradation of the CREBBP/EP300 repressor SNIP1. Positively regulates BMP4-induced expression of odontogenic development regulator MSX1 following IPO7-mediated nuclear import (By similarity).

Cellular Location
Cytoplasm. Nucleus Note=Cytoplasmic in the absence of ligand. Migrates to the nucleus when complexed with SMAD4 (PubMed:15647271). Co-localizes with LEMD3 at the nucleus inner membrane (PubMed:15647271). Exported from the nucleus to the cytoplasm when dephosphorylated (By similarity) {ECO:0000250|UniProtKB:P70340, ECO:0000269|PubMed:15647271}

Tissue Location
Ubiquitous. Highest expression seen in the heart and skeletal muscle

SMAD1 Blocking Peptide (N-term) - Protocols

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

- Blocking Peptides

SMAD1 Blocking Peptide (N-term) - Images

SMAD1 Blocking Peptide (N-term) - Background

The protein encoded by this gene belongs to the SMAD, a family of proteins similar to the gene products of the Drosophila gene 'mothers against decapentaplegic' (Mad) and the C. elegans gene Sma. SMAD proteins are signal transducers and transcriptional modulators that mediate multiple signaling pathways. This protein mediates the signals of the bone morphogenetic proteins (BMPs), which are involved in a range of biological activities including cell growth, apoptosis, morphogenesis, development and immune responses. In response to BMP ligands, this protein can be phosphorylated and activated by the BMP receptor kinase. The phosphorylated form of this protein forms a complex with SMAD4, which is important for its function in the transcription regulation. This protein is a target for SMAD-specific E3 ubiquitin ligases, such as SMURF1 and SMURF2, and undergoes ubiquitination and proteasome-mediated degradation. Alternatively spliced transcript variants encoding the same protein have been observed.

SMAD1 Blocking Peptide (N-term) - References

Yang, J., et al. Circ. Res. 107(2):252-262(2010)
Smythies, L.E., et al. J. Biol. Chem. 285(25):19593-19604(2010)
Abhishek, K., et al. Biochem. Biophys. Res. Commun. 396(4):950-955(2010)
Jugessur, A., et al. PLoS ONE 5 (7), E11493 (2010) :
Ye, F., et al. J. Exp. Clin. Cancer Res. 29, 78 (2010) :