

**SMURF2 Antibody (C-term) Blocking Peptide**  
**Synthetic peptide**  
**Catalog # BP2105b****Specification**

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**SMURF2 Antibody (C-term) Blocking Peptide - Product Information**Primary Accession [Q9HAU4](#)**SMURF2 Antibody (C-term) Blocking Peptide - Additional Information**

Gene ID 64750

**Other Names**

E3 ubiquitin-protein ligase SMURF2, hSMURF2, 632-, SMAD ubiquitination regulatory factor 2, SMAD-specific E3 ubiquitin-protein ligase 2, SMURF2

**Target/Specificity**

The synthetic peptide sequence used to generate the antibody [AP2105b](/product/products/AP2105b) was selected from the C-term region of human SMURF2. 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.

**SMURF2 Antibody (C-term) Blocking Peptide - Protein Information**Name SMURF2 ([HGNC:16809](#))**Function**

E3 ubiquitin-protein ligase which accepts ubiquitin from an E2 ubiquitin-conjugating enzyme in the form of a thioester and then directly transfers the ubiquitin to targeted substrates (PubMed: [11016919](http://www.uniprot.org/citations/11016919)). Interacts with SMAD7 to trigger SMAD7-mediated transforming growth factor beta/TGF-beta receptor ubiquitin-dependent degradation, thereby down-regulating TGF-beta signaling (PubMed: [11163210](http://www.uniprot.org/citations/11163210), PubMed: [12717440](http://www.uniprot.org/citations/12717440), PubMed: [21791611](http://www.uniprot.org/citations/21791611)). In addition, interaction with SMAD7 activates autocatalytic degradation, which is prevented by interaction with AIMP1 (PubMed: [18448069](http://www.uniprot.org/citations/18448069)). Also forms a stable complex with TGF-beta receptor-mediated

phosphorylated SMAD1, SMAD2 and SMAD3, and targets SMAD1 and SMAD2 for ubiquitination and proteasome-mediated degradation (PubMed: [11016919](http://www.uniprot.org/citations/11016919), PubMed: [11158580](http://www.uniprot.org/citations/11158580), PubMed: [11389444](http://www.uniprot.org/citations/11389444)). SMAD2 may recruit substrates, such as SNON, for ubiquitin-dependent degradation (PubMed: [11389444](http://www.uniprot.org/citations/11389444)). Negatively regulates TGFB1-induced epithelial-mesenchymal transition and myofibroblast differentiation (PubMed: [30696809](http://www.uniprot.org/citations/30696809)).

**Cellular Location**

Nucleus. Cytoplasm. Cell membrane. Membrane raft. Note=Cytoplasmic in the presence of SMAD7. Colocalizes with CAV1, SMAD7 and TGF-beta receptor in membrane rafts

**Tissue Location**

Widely expressed.

**SMURF2 Antibody (C-term) Blocking Peptide - Protocols**

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

- [Blocking Peptides](#)

**SMURF2 Antibody (C-term) Blocking Peptide - Images****SMURF2 Antibody (C-term) Blocking Peptide - Background**

SMURF2 is an E3 ubiquitin-protein ligase which accepts ubiquitin from an E2 ubiquitin-conjugating enzyme in the form of a thioester and then directly transfers the ubiquitin to targeted substrates. This protein interacts with SMAD1, SMAD2 and SMAD7 in order to trigger their ubiquitination and proteasome-dependent degradation. It enhances the inhibitory activity of SMAD7 and reduces the transcriptional activity of SMAD2. Coexpression of SMURF2 with SMAD1 results in considerable decrease in steady-state level of SMAD1 protein and a smaller decrease of SMAD2 level.

**SMURF2 Antibody (C-term) Blocking Peptide - References**

Tajima, Y., et al., J. Biol. Chem. 278(12):10716-10721 (2003). Suzuki, C., et al., J. Biol. Chem. 277(42):39919-39925 (2002). Ebisawa, T., et al., J. Biol. Chem. 276(16):12477-12480 (2001). Zhu, H., et al., Nature 400(6745):687-693 (1999). Lambris, J., et al., J. Immunol. Methods 27(1):55-59 (1979).