SANTA CRUZ BIOTECHNOLOGY, INC.

p-Smad2 (Ser 467): sc-101801

BACKGROUND

Smad proteins, the mammalian homologs of the *Drosophila* mothers against decapentaplegic (Mad), have been implicated as downstream effectors of TGFβ/BMP signaling. Smad1 (also designated Madr1 or JV4-1) and Smad5 are effectors of BMP-2 and BMP-4 function, while Smad2 (also designated Madr2 or JV18-1) and Smad3 are involved in TGFβ and activin-mediated growth modulation. Smad4 (also designated DPC4) has been shown to mediate all of the above activities through interaction with various Smad family members. Smad6 and Smad7 regulate the response to activin/TGFβ signaling by interfering with TGFβ-mediated phosphorylation of other Smad proteins. Mouse, rat and human Smad2 are subject to phosphorylation by TGFβ receptors, and Thr 220 is one of the sites that is targeted for phosphorylation.

REFERENCES

1. Liu, F., et al. 1996. A human Mad protein acting as a BMP-regulated transcriptional activator. Nature 381: 620-623.
2. Hoodless, P.A., et al. 1996. Madr1, a Mad-related protein that functions in BMP-2 signaling pathways. Cell 85: 489-500.

CHROMOSOMAL LOCATION

Genetic locus: SMAD2 (human) mapping to 18q21.1; Smad2 (mouse) mapping to 18 E3.

SOURCE

p-Smad2 (Ser 467) is a rabbit polyclonal antibody raised against a short amino acid sequence containing Ser 467 phosphorylated Smad2 of human origin.

PRODUCT

Each vial contains 100 µg IgG in 1.0 ml PBS with < 0.1% sodium azide and 0.1% gelatin.

STORAGE

Store at 4° C, **DO NOT FREEZE**. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.

APPLICATIONS

p-Smad2 (Ser 467) is recommended for detection of Ser 467 phosphorylated Smad2 of mouse, rat and human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000) and immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)].

Suitable for use as control antibody for Smad2 siRNA (h): sc-38374, Smad2 siRNA (m): sc-38375, Smad2 siRNA (r): sc-77325, Smad2 shRNA Plasmid (h): sc-38374-SH, Smad2 shRNA Plasmid (m): sc-38375-SH, Smad2 shRNA Plasmid (r): sc-77325-SH, Smad2 shRNA (h) Lentiviral Particles: sc-38374-V, Smad2 shRNA (m) Lentiviral Particles: sc-38375-V and Smad2 shRNA (r) Lentiviral Particles: sc-77325-V.

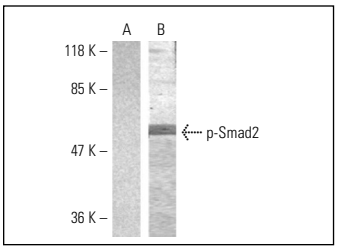
Molecular Weight of p-Smad2: 52 kDa.

Positive Controls: PMA-treated 293 whole cell lysate.

RECOMMENDED SECONDARY REAGENTS

To ensure optimal results, the following support (secondary) reagents are recommended: 1) Western blotting: use goat anti-rabbit IgG-HRP: sc-2004 (dilution range: 1:2000-1:100,000) or Cruz Marker™ compatible goat anti-rabbit IgG-HRP: sc-2030 (dilution range: 1:2000-1:5000), Cruz Marker™ Molecular Weight Standards: sc-2035, TBS Blotto B Blocking Reagent: sc-2335 (use 50 mM NaF, sc-24988, as diluent), Western Blotting Luminol Reagent: sc-2048 and Lambda Phosphatase: sc-200312A. 2) Immunoprecipitation: use Protein A/G PLUS-Agarose: sc-2003 (0.5 ml agarose/2.0 ml).

DATA



p-Smad2 (Ser 467): sc-101801. Western blot analysis of phosphorylated Smad2 expression in untreated (**A**) and PMA-treated (**B**) 293 whole cell lysates.

SELECT PRODUCT CITATIONS

1. Chen, T.C., et al. 2013. Moxifloxacin modifies corneal fibroblast-to-myofibroblast differentiation. Br. J. Pharmacol. 168: 1341-1354.
2. Shyu, K.G., et al. 2013. Mechanical stretch via transforming growth factor-β1 activates microRNA208a to regulate endoglin expression in cultured rat cardiac myoblasts. Eur. J. Heart Fail. 15: 36-45.
3. Mytilinaiou, M., et al. 2013. Syndecan-2 is a key regulator of transforming growth factor β 2/Smad2-mediated adhesion in fibrosarcoma cells. IUBMB Life 65: 134-143.
4. Yu, L., et al. 2013. Exosomes with membrane-associated TGF-β1 from gene-modified dendritic cells inhibit murine EAE independently of MHC restriction. Eur. J. Immunol. 15: 36-45.
5. Yang, F., et al. 2015. Activated cytotoxic lymphocytes promote tumor progression by increasing the ability of 3LL tumor cells to mediate MDSC chemoattraction via Fas signaling. Cell. Mol. Immunol. 12: 66-76.
6. Liu, C., et al. 2015. Xia-yu-xue decoction (XYXD) reduces carbon tetrachloride (CCl4)-induced liver fibrosis through inhibition hepatic stellate cell activation by targeting NFκB and TGF-β1 signaling pathways. BMC Complement. Altern. Med. 15: 201.
7. Ding, H., et al. 2015. Antifibrotic properties of receptor for advanced glycation end products in idiopathic pulmonary fibrosis. Pulm. Pharmacol. Ther. 35: 34-41.

RESEARCH USE

For research use only, not for use in diagnostic procedures.