SANTA CRUZ BIOTECHNOLOGY, INC.

p-Smad2/3 (Ser 423/425): sc-11769

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

Smad proteins, the mammalian homologs of the *Drosophila* mothers against dpp (Mad), mediate intracellular signaling of the TGFβ superfamily. TGFβ type I receptor mediates phosphorylation of Smad2 (also designated Madr2 or JV18-1) at the C-terminal Ser 465 and Ser 467 residues, and phosphorylation of Smad3 at the C-terminal Ser 423 and Ser 425 residues in an obligate order. Upon phosphorylation and activation by the active TGFβ receptor complex, Smad2 and Smad3 homo-oligomerize or hetero-oligomerize with Smad4, and translocate into the nucleus. There they interact with various cellular partners, bind DNA, regulate transcription and cross-talk with other signaling pathways. Activin A also induces these cellular responses by phosphorylating Smad2, but it is a less potent activator than TGFβ. TGFβ-induced phosphorylation of Smad3 promotes its interaction with coactivators CBP and p300 and is essential for the TGFβ-mediated inhibition of cellular proliferation.

CHROMOSOMAL LOCATION

Genetic locus: SMAD3 (human) mapping to 15q22.33, SMAD2 (human) mapping to 18q21.1; Smad3 (mouse) mapping to 9 C, Smad2 (mouse) mapping to 18 E3.

SOURCE

p-Smad2/3 (Ser 423/425) is available as either goat (sc-11769) or rabbit (sc-11769-R) polyclonal affinity purified antibody raised against a short amino acid sequence containing Ser 423 and Ser 425 phosphorylated Smad3 of human origin.

PRODUCT

Each vial contains either 200 µg (sc-11769) or 100 µg (sc-11769-R) IgG in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

Blocking peptide available for competition studies, sc-11769 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

Available as TransCruz reagent for Gel Supershift and ChIP applications, sc-11769 X, 200 µg/0.1 ml.

APPLICATIONS

p-Smad2/3 (Ser 423/425) is recommended for detection of Ser 423 and Ser 425 dually phosphorylated Smad3 and correspondingly phosphorylated Smad2 of mouse, rat and human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)], immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500), immunohistochemistry (including paraffin-embedded sections) (starting dilution 1:50, dilution range 1:50-1:500) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

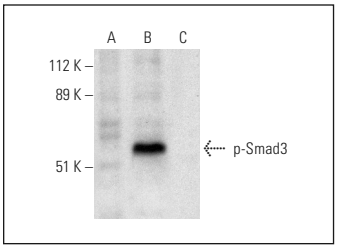
Suitable for use as control antibody for Smad2/3 siRNA (h): sc-37238, Smad2/3 siRNA (m): sc-37239, Smad2/3 shRNA Plasmid (h): sc-37238-SH, Smad2/3 shRNA Plasmid (m): sc-37239-SH, Smad2/3 shRNA (h) Lentiviral Particles: sc-37238-V and Smad2/3 shRNA (m) Lentiviral Particles: sc-37239-V.

Molecular Weight of p-Smad2/3: 55-60 kDa.

STORAGE

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

DATA



Western blot analysis of Smad3 phosphorylation in non-transfected: sc-117752 (**A**), untreated human Smad3 transfected: sc-116400 (**B**) and lambda protein phosphatase (sc-200312A) treated human Smad3 transfected: sc-116400 (**C**) 293T whole cell lysates. Antibody tested is p-Smad2/3 (Ser 423/425)-R: sc-11769-R (**A**,**B**,**C**).

SELECT PRODUCT CITATIONS

1. Bogdanovich, S., et al. 2002. Functional improvement of dystrophic muscle by myostatin blockade. Nature 420: 418-421.
2. Langley, B., et al. 2002. Myostatin inhibits myoblast differentiation by down-regulating MyoD expression. J. Biol. Chem. 277: 49831-49840.
3. Tamosiuniene, R., et al. 2011. Regulatory T cells limit vascular endothelial injury and prevent pulmonary hypertension. Circ. Res. 109: 867-879.
4. Li, Z.I., et al. 2011. C-reactive protein promotes acute renal inflammation and fibrosis in unilateral ureteral obstructive nephropathy in mice. Lab. Invest. 91: 837-851.
5. Meng, X.M., et al. 2011. Disruption of Smad4 impairs TGF-β/Smad3 and Smad7 transcriptional regulation during renal inflammation and fibrosis *in vivo* and *in vitro*. Kidney Int. 81: 266-279.
6. Liu, F., et al. 2011. C-reactive protein promotes diabetic kidney disease in a mouse model of type 1 diabetes. Diabetologia 54: 2713-2723.
7. Ceballos, M.P., et al. 2011. Interferon-α2b and transforming growth factor-β1 treatments on HCC cell lines: are Wnt/β-catenin pathway and Smads signaling connected in hepatocellular carcinoma? Biochem. Pharmacol. 82: 1682-1691.
8. Cozzoli, A., et al. 2011. Enalapril treatment discloses an early role of angiotensin II in inflammation- and oxidative stress-related muscle damage in dystrophic mdx mice. Pharmacol. Res. 64: 482-492.
9. Handra-Luca, A., et al. 2012. Tumoral epithelial and stromal expression of SMAD proteins in pancreatic ductal adenocarcinomas. J. Hepatobiliary Pancreat. Sci. 20: 294-302.

RESEARCH USE

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