SANTA CRUZ BIOTECHNOLOGY, INC.

TGFβ1/2/3 (H-112): sc-7892

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

Transforming growth factor betas (TGFβs) were originally discovered due to their ability to promote anchorage-independent growth of rat NRK fibroblasts in the presence of TGFα. It is now realized that TGFβs mediate many cell-cell interactions that occur during embryonic development. Three TGFβs have been identified in mammals. TGFβ1, TGFβ2 and TGFβ3 are each synthesized as precursor proteins that are very similar in that each is cleaved to yield a 112 amino acid polypeptide that remains associated with the latent portion of the molecules. Biologically active TGFβ requires dimerization of the monomers (usually homodimers) and release of the latent peptide portion. Overall, the mature region of the TGFβ3 protein has approximately 80% identity to the mature region of both TGFβ1 and TGFβ2. However, the NH_2 terminals or precursor regions of their molecules share only 27% sequence identity.

SOURCE

TGFβ1/2/3 (H-112) is a rabbit polyclonal antibody raised against amino acids 301-412 of TGFβ1 of human origin.

PRODUCT

Each vial contains 200 µg IgG in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

APPLICATIONS

TGFβ1/2/3 (H-112) is recommended for detection of precursor and mature TGFβ1, TGFβ2 and TGFβ3 of mouse, rat, human and *Xenopus laevis* 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).

TGFβ1/2/3 (H-112) is also recommended for detection of precursor and mature TGFβ1, TGFβ2 and TGFβ3 in additional species, including equine, canine, bovine, porcine and avian.

Suitable for use as control antibody for TGFβ1/2/3 siRNA (h): sc-44146, TGFβ1/2/3 siRNA (m): sc-44147, TGFβ1/2/3 shRNA Plasmid (h): sc-44146-SH, TGFβ1/2/3 shRNA Plasmid (m): sc-44147-SH, TGFβ1/2/3 shRNA (h) Lentiviral Particles: sc-44146-V and TGFβ1/2/3 shRNA (m) Lentiviral Particles: sc-44147-V.

Molecular weight of TGF 1/2/3 monomer: 13 kDa.

Molecular weight of TGF 1/2/3 dimer: 25 kDa.

STORAGE

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

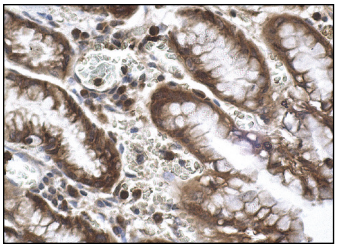
PROTOCOLS

See our web site at www.scbt.com or our catalog for detailed protocols and support products.

RESEARCH USE

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

DATA



TGFβ1/2/3 (H-112): sc-7892. Immunoperoxidase staining of formalin fixed, paraffin-embedded human stomach tissue showing cytoplasmic staining of glandular cells.

SELECT PRODUCT CITATIONS

1. Pu, L., et al. 2002. Dual G_1 and G_2 phase inhibition by a novel, selective Cdc25 inhibitor 6-chloro-7-(2-morpholin-4-ylethylamino)-quinoline-5,8-dione. J. Biol. Chem. 277: 46877-46885.
2. Carlson, M.E., et al. 2008. Imbalance between pSmad3 and Notch induces CDK inhibitors in old muscle stem cells. Nature 454: 528-532.
3. Ghannad, F., et al. 2008. Absence of αvβ6 Integrin is linked to initiation and progression of periodontal disease. Am. J. Pathol. 172: 1271-1286.
4. Jadhav, A., et al. 2009. Heme arginate suppresses cardiac lesions and hypertrophy in deoxycorticosterone acetate-salt hypertension. Exp. Biol. Med. 234: 764-778.
5. Li, R., et al. 2009. Expression of IL-1α, IL-6, TGF-β, FasL and ZNF265 during sertoli cell infection by ureaplasma urealyticum. Cell. Mol. Immunol. 6: 215-221.
6. Jadhav, A., et al. 2009. Hemin therapy attenuates kidney injury in deoxycorticosterone acetate-salt hypertensive rats. Am. J. Physiol. Renal Physiol. 296: F521-F534.
7. Sadallah, S., et al. 2011. Microparticles (ectosomes) shed by stored human platelets downregulate macrophages and modify the development of dendritic cells. J. Immunol. 186: 6543-6552.
8. Nakatsuka, A., et al. 2012. RXR antagonism induces G_0 /G_1 cell cycle arrest and ameliorates obesity by up-regulating the p53-$p21^{Cip1}$ pathway in adipocytes. J. Pathol. 226: 784-795.