

TNF-R2 (H-202): sc-7862

BACKGROUND

Tumor necrosis factor (TNF) is a pleiotropic cytokine whose function is mediated through two distinct cell surface receptors. These receptors, designated TNF-R1 and TNF-R2, are expressed on most cell types. The majority of TNF functions are primarily mediated through TNF-R1, while signaling through TNF-R2 occurs less extensively and is confined to cells of the immune system. Both of these proteins belong to the growing TNF and nerve growth factor (NGF) receptor superfamily, which includes FAS, CD30, CD27 and CD40. The members of this superfamily are type I membrane proteins that share sequence homology confined to the extracellular region. TNF-R1 shares a motif termed the "death domain" with FAS and three structurally unrelated signaling proteins, TRADD, FADD and RIP. This death domain is required for transduction of the apoptotic signal.

REFERENCES

1. Smith, C.A., et al. 1994. The TNF receptor superfamily of cellular and viral proteins: activation, costimulation, and death. *Cell* 76: 959-962.
2. Cleveland, J.L., et al. 1995. Contenders in FAS-L/TNF death signaling. *Cell* 81: 479-482.

CHROMOSOMAL LOCATION

Genetic locus: TNFRSF1B (human) mapping to 1p36.22; Tnfrsf1b (mouse) mapping to 4 E1.

SOURCE

TNF-R2 (H-202) is a rabbit polyclonal antibody raised against amino acids 260-461 of TNF-R2 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

TNF-R2 (H-202) is recommended for detection of TNF-R2 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) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

Suitable for use as control antibody for TNF-R2 siRNA (h): sc-36689, TNF-R2 siRNA (m): sc-36690, TNF-R2 shRNA Plasmid (h): sc-36689-SH, TNF-R2 shRNA Plasmid (m): sc-36690-SH, TNF-R2 shRNA (h) Lentiviral Particles: sc-36689-V and TNF-R2 shRNA (m) Lentiviral Particles: sc-36690-V.

Molecular Weight of TNF-R2: 75 kDa.

Positive Controls: Jurkat whole cell lysate: sc-2204, SK-BR-3 cell lysate: sc-2218 or MCF7 whole cell lysate: sc-2206.

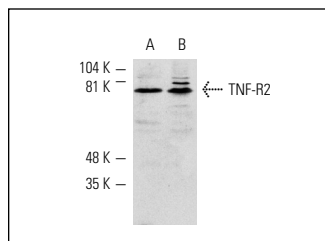
RESEARCH USE

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

STORAGE

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

DATA



TNF-R2 (H-202): sc-7862. Western blot analysis of TNF-R2 p75 expression in MCF7 (A) and SK-BR-3 (B) whole cell lysates.

SELECT PRODUCT CITATIONS

1. Qin, J.Z., et al. 2001. Role of NFκB activity in apoptotic response of keratinocytes mediated by interferon-γ, tumor necrosis factor α, and tumor necrosis factor-related apoptosis-inducing ligand. *J. Invest. Dermatol.* 117: 898-907.
2. Pentikäinen, V., et al. 2001. TNFα down-regulates the FAS ligand and inhibits germ cell apoptosis in the human testis. *J. Clin. Endocrinol. Metab.* 86: 4480-4488.
3. Vaitaitis, G.M. and Wagner, D.H. 2010. CD40 glycoforms and TNF-receptors 1 and 2 in the formation of CD40 receptors in autoimmunity. *Mol. Immunol.* 47: 2303-2313.
4. Thoh, M., et al. 2010. Azadirachtin interacts with the tumor necrosis factor (TNF) binding domain of its receptors and inhibits TNF-induced biological responses. *J. Biol. Chem.* 285: 5888-5895.
5. Yin, H., et al. 2011. Ginsenoside-Ry1 enhances angiogenesis and ameliorates ventricular remodeling in a rat model of myocardial infarction. *J. Mol. Med.* 89: 363-375.
6. Andrade, P., et al. 2011. Tumor necrosis factor-α levels correlate with post-operative pain severity in lumbar disc hernia patients: opposite clinical effects between tumor necrosis factor receptor 1 and 2. *Pain* 152: 2645-2652.
7. Andrade, P., et al. 2012. The thalidomide analgesic effect is associated with differential TNF-α receptor expression in the dorsal horn of the spinal cord as studied in a rat model of neuropathic pain. *Brain Res.* 1450: 24-32.

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