

# TNF-R1 (hBA-162): sc-4566

## 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 coined 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., Farrah, T. and Goodwin, R.G. 1994. The TNF receptor superfamily of cellular and viral proteins: activation, costimulation, and death. *Cell* 76: 959-962.
2. Nagata, S. and Golstein, P. 1995. The FAS death factor. *Science* 267: 1449-1456.
3. Cleveland, J.L. and Ihle, J.N. 1995. Contenders in FAS-L/TNF death signaling. *Cell* 81: 479-482.
4. Hsu, H., Xiong, J. and Goeddel, D.V. 1995. The TNF receptor 1-associated protein TRADD signals cell death and NF $\kappa$ B activation. *Cell* 81: 495-504.
5. Chinnaiyan, A.M., O'Rourke, K., Tewari, M. and Dixit, V.M. 1995. FADD, a novel death domain-containing protein, interacts with the death domain of FAS and initiates apoptosis. *Cell* 81: 505-512.
6. Stanger, B.Z., Leder, P., Lee, T.H., Kim, E. and Seed, B. 1995. RIP: a novel protein containing a death domain that interacts with FAS/APO-1 (CD95) in yeast and causes cell death. *Cell* 81: 513-523.
7. Boldin, M.P., Mett, I.L., Varfolomeev, E.E., Chumakov, I., Shemer-Avni, Y., Camonis, J.H. and Wallach, D. 1995. Self-association of the "death domains" of the p55 tumor necrosis factor (TNF) receptor and FAS/APO-1 prompts signaling for TNF and FAS/APO-1 effects. *J. Biol. Chem.* 270: 387-391.
8. Hofmann, K. and Tschopp, J. 1995. The death domain motif found in FAS/APO-1 and TNF receptor is present in proteins involved in apoptosis and axonal guidance. *FEBS Lett.* 371: 321-323.
9. Baker, S.J. and Reddy, E.P. 1996. Transducers of life and death: TNF receptor superfamily and associated proteins. *Oncogene* 12: 1-9.

## CHROMOSOMAL LOCATION

Genetic locus: TNFRSF1A (human) mapping to 12p13.2; Tnfrsf1a (mouse) mapping to 6 F3.

## SOURCE

TNF-R1 (hBA-162) is produced in *E. coli* as 18.3 kDa biologically active, tagged fusion protein corresponding to 162 amino acids comprising the extracellular domain of TNF-R1 of human origin.

## PRODUCT

TNF-R1 (hBA-162) is purified from bacterial lysates (>98%); supplied as 50  $\mu$ g purified protein.

## Biological Activity

TNF-R1 (hBA-162) is biologically active as determined by inhibitory effect of the TNF $\alpha$  mediated cytotoxicity in murine L929 cells.

Expected ED<sub>50</sub>: 0.05  $\mu$ g/ml.

## RECONSTITUTION

In order to avoid freeze/thaw damaging of the active protein, dilute protein when first used to desired working concentration. Either a sterile filtered standard buffer (such as 50mM TRIS or 1X PBS) or water can be used for the dilution. Store any thawed aliquot in refrigeration at 2° C to 8° C for up to four weeks, and any frozen aliquot at -20° C to -80° C for up to one year. It is recommended that frozen aliquots be given an amount of standard cryopreservative (such as Ethylene Glycol or Glycerol 5-20% v/v), and refrigerated samples be given an amount of carrier protein (such as heat inactivated FBS or BSA to 0.1% v/v) or non-ionic detergent (such as Triton X-100 or Tween 20 to 0.005% v/v), to aid stability during storage.

## SELECT PRODUCT CITATIONS

1. Qin, Y., Cheng, C., Wang, H., Shao, X., Gao, Y. and Shen, A. 2008. TNF- $\alpha$  as an autocrine mediator and its role in the activation of Schwann cells. *Neurochem. Res.* 33: 1077-1084.
2. Liu, S., Wu, F., Wu, Z., Li, Y., Zhang, S. and Yu, N. 2019. IL-17A synergistically enhances TLR3-mediated IL-36 $\gamma$  production by keratinocytes: a potential role in injury-amplified psoriatic inflammation. *Exp. Dermatol.* 28: 233-239.

## STORAGE

Store desiccated at -20° C. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.

## RESEARCH USE

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

## PROTOCOLS

See our web site at [www.scbt.com](http://www.scbt.com) for detailed protocols and support products.