SANTA CRUZ BIOTECHNOLOGY, INC.

Fn14 (ITEM-4): sc-56250

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

Fn14, the TWEAK receptor, is a recently identified member of the TNF receptor superfamily and is expressed on smooth muscle cells and endothelial cells. It is a weak inducer of apoptosis and promotes angiogenesis. Fn14 is a type 1 membrane protein. It associates with TRAF1 and TRAF2, and may modulate cellular adhesion to matrix proteins. Fn14 is highly expressed in heart, placenta and kidney, and moderately expressed in lung, skeletal muscle and pancreas. It is the smallest member of the TNF receptor (TNFR) superfamily described to date, and signals via recruitment of several different TNFR-associated factors.

CHROMOSOMAL LOCATION

Genetic locus: TNFRSF12A (human) mapping to 16p13.3; Tnfrsf12a (mouse) mapping to 17 A3.3.

SOURCE

Fn14 (ITEM-4) is a mouse monoclonal antibody raised against Fn14 of human origin.

PRODUCT

Each vial contains 200 µg IgG_{2b} kappa light chain in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

Fn14 (ITEM-4) is available conjugated to agarose (sc-56250 AC), 500 µg/ 0.25 ml agarose in 1 ml, for IP; to HRP (sc-56250 HRP), 200 µg/ml, for WB, IHC(P) and ELISA; to either phycoerythrin (sc-56250 PE), fluorescein (sc-56250 FITC), Alexa Fluor® 488 (sc-56250 AF488), Alexa Fluor® 546 (sc-56250 AF546), Alexa Fluor® 594 (sc-56250 AF594) or Alexa Fluor® 647 (sc-56250 AF647), 200 µg/ml, for WB (RGB), IF, IHC(P) and FCM; and to either Alexa Fluor® 680 (sc-56250 AF680) or Alexa Fluor® 790 (sc-56250 AF790), 200 µg/ml, for Near-Infrared (NIR) WB, IF and FCM.

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APPLICATIONS

Fn14 (ITEM-4) is recommended for detection of Fn14 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 flow cytometry (1 µg per 1 x 10^6 cells).

Suitable for use as control antibody for Fn14 siRNA (h): sc-43764, Fn14 siRNA (m): sc-145209, Fn14 shRNA Plasmid (h): sc-43764-SH, Fn14 shRNA Plasmid (m): sc-145209-SH, Fn14 shRNA (h) Lentiviral Particles: sc-43764-V and Fn14 shRNA (m) Lentiviral Particles: sc-145209-V.

Molecular Weight of Fn14: 14 kDa.

Positive Controls: Fn14 (h2): 293T Lysate: sc-174426 or HISM cell lysate: sc-2229.

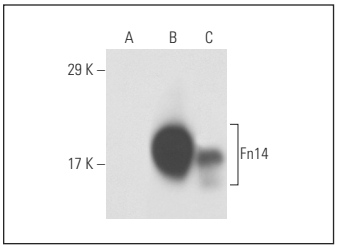
STORAGE

Store at 4° C, **DO NOT FREEZE**. 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.

DATA



Fn14 (ITEM-4): sc-56250. Western blot analysis of Fn14 expression in non-transfected 293T: sc-117752 (**A**), human Fn14 transfected 293T: sc-174426 (**B**) and HISM (**C**) whole cell lysates.

SELECT PRODUCT CITATIONS

1. Alexaki, V.I., et al. 2009. Adipocytes as immune cells: differential expression of TWEAK, BAFF, and APRIL and their receptors (Fn14, BAFF-R, TACI, and BCMA) at different stages of normal and pathological adipose tissue development. J. Immunol. 183: 5948-5956.
2. Pelekanou, V., et al. 2011. Detection of the TNFSF members BAFF, APRIL, TWEAK and their receptors in normal kidney and renal cell carcinomas. Anal. Cell. Pathol. 34: 49-60.
3. Sabour Alaoui, S., et al. 2012. TWEAK affects keratinocyte G_2/M growth arrest and induces apoptosis through the translocation of the AIF protein to the nucleus. PLoS ONE 7: e33609.
4. Dore-Duffy, P. 2014. Pericytes and adaptive angioplasticity: the role of tumor necrosis factor-like weak inducer of apoptosis (TWEAK). Methods Mol. Biol. 1135: 35-52.
5. Doerner, J.L., et al. 2015. TWEAK/Fn14 signaling involvement in the pathogenesis of cutaneous disease in the MRL/lpr model of spontaneous lupus. J. Invest. Dermatol. 135: 1986-1995.
6. Winer, H., et al. 2018. Autophagy differentially regulates TNF receptor Fn14 by distinct mammalian Atg8 proteins. Nat. Commun. 9: 3744.
7. Yadav, A., et al. 2021. Magnoflorine prevent the skeletal muscle atrophy via Akt/mTOR/FoxO signal pathway and increase slow-MyHC production in streptozotocin-induced diabetic rats. J. Ethnopharmacol. 267: 113510.
8. Ni, Y., et al. 2021. Interruption of neutrophil extracellular traps formation dictates host defense and tubular HOXA5 stability to augment efficacy of anti-Fn14 therapy against septic AKI. Theranostics 11: 9431-9451.
9. Bonan, N.F., et al. 2022. Anti-Fn14-conjugated prussian blue nanoparticles as a targeted photothermal therapy agent for glioblastoma. Nanomaterials 12: 2645.

PROTOCOLS

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