SANTA CRUZ BIOTECHNOLOGY, INC.

RANKL (N-19): sc-7628

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

Members of the tumor necrosis factor (TNF) receptor superfamily interact with signaling molecules of the TNF receptor-associated factor (TRAF) family to activate the NFκB and JNK pathways. RANK (receptor activator of NFκB) is a member of the TNFR family identified on dendritic cells. This type I membrane receptor is expressed in a broad range of tissues. The C-terminus of RANK is required for RANK to bind TRAF2, 5 and 6, and it is also necessary for stimulating NFκB activation. The ligand for this receptor, RANKL (also designated TRANCE, OPGL or ODF), is a type II transmembrane protein expressed primarily in lymphoid tissues and T cell lines. RANKL appears to be an important regulator of T cells and osteoclasts.

CHROMOSOMAL LOCATION

Genetic locus: TNFSF11 (human) mapping to 13q14.11; Tnfsf11 (mouse) mapping to 14 D3.

SOURCE

RANKL (N-19) is an affinity purified goat polyclonal antibody raised against a peptide mapping at the N-terminus of RANKL of mouse origin.

PRODUCT

Each vial contains 100 µg IgG in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

Blocking peptide available for competition studies, sc-7628 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

APPLICATIONS

RANKL (N-19) is recommended for detection of RANKL 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 RANKL siRNA (h): sc-29464, RANKL siRNA (m): sc-37270, RANKL shRNA Plasmid (h): sc-29464-SH, RANKL shRNA Plasmid (m): sc-37270-SH, RANKL shRNA (h) Lentiviral Particles: sc-29464-V and RANKL shRNA (m) Lentiviral Particles: sc-37270-V.

Molecular Weight of RANKL full length/membrane bound: 35-40 kDa.

Molecular Weight of soluble RANKL: 20-30 kDa.

Positive Controls: BYDP whole cell lysate: sc-364368.

RESEARCH USE

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

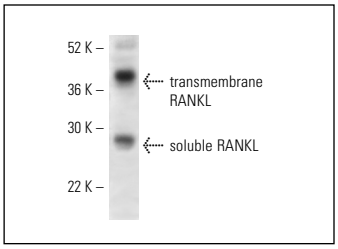
PROTOCOLS

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

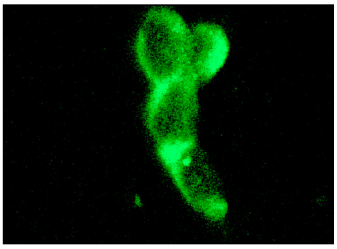
STORAGE

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

DATA



RANKL (N-19): sc-7628. Western blot analysis of RANKL expression in BYDP whole cell lysate.



RANKL (N-19): sc-7628. Immunofluorescence staining of methanol-fixed LNCaP cells showing membrane staining.

SELECT PRODUCT CITATIONS

1. Lubberts, E., et al. 2000. IL-4 gene therapy for collagen arthritis suppresses synovial IL-17 and osteoprotegerin ligand and prevents bone erosion. J. Clin. Invest. 105: 1697-1710.
2. Wehrhan, F., et al. 2010. Expression of Msx-1 is suppressed in bisphosphonate associated osteonecrosis related jaw tissue-etiopathology considerations respecting jaw developmental biology-related unique features. J. Transl. Med. 8: 96.
3. Kayamori, K., et al. 2010. Roles of interleukin-6 and parathyroid hormone-related peptide in osteoclast formation associated with oral cancers: significance of interleukin-6 synthesized by stromal cells in response to cancer cells. Am. J. Pathol. 176: 968-980.
4. Elias, L.S., et al. 2010. Markers of bone remodeling in neoplastic and bone-related lesions. Oral Surg. Oral Med. Oral Pathol. Oral Radiol. Endod. 110: 624-631.
5. Jiao, K., et al. 2011. Subchondral bone loss following orthodontically induced cartilage degradation in the mandibular condyles of rats. Bone 48: 362-371.
6. Garcia, V.G., et al. 2011. Treatment of experimental periodontal disease with antimicrobial photodynamic therapy in nicotine-modified rats. J. Clin. Periodontol. 38: 1106-1114.
7. Claudino, M., et al. 2012. Spontaneous periodontitis development in diabetic rats involves an unrestricted expression of inflammatory cytokines and tissue destructive factors in the absence of major changes in commensal oral microbiota. Exp. Diabetes Res. 2012: 356841.