

Recombinant Mouse TNFSF11/RANKL/CD254 Protein

Catalog No.: RP02134 **Recombinant** **1 Publications**

Sequence Information

Species	Gene ID	Swiss Prot
Mouse	21943	O35235-1

Tags

N-His□N-hFc

Synonyms

ODF; OPGL; RANKL; Ly109I; Trance;
TNFSF11

Product Information

Source	Purification
HEK293 cells	≥ 95 % as determined by SDS-PAGE; ≥ 95 % as determined by HPLC.

Calculated MW	Observed MW
55.63 KD	60-75 KD

Endotoxin

< 0.01 EU/μg of the protein by LAL method

Formulation

Lyophilized from a 0.22 μm filtered solution of PBS, pH 7.4.

Reconstitution

Centrifuge the vial before opening. Reconstitute to a concentration of 0.1-0.5 mg/mL in sterile distilled water. Avoid vortex or vigorously pipetting the protein. For long term storage, it is recommended to add a carrier protein or stabilizer (e.g. 0.1% BSA, 5% HSA, 10% FBS or 5% Trehalose), and aliquot the reconstituted protein solution to minimize free-thaw cycles.

Background

Tumor necrosis factor ligand superfamily member 11, also known as Receptor activator of nuclear factor kappa-B ligand, Osteoprotegerin ligand, TNFSF11, RANKL, TRANCE, OPGL and CD254, is a single-pass type II membrane protein that belongs to the tumor necrosis factor family. The receptor activator of nuclear factor-kappaB ligand (RANKL), its cognate receptor RANK, and its natural decoy receptor osteoprotegerin have been identified as the final effector molecules of osteoclastic bone resorption. RANK and RANKL are key regulators of bone remodeling and regulate T cell/dendritic cell communications, and lymph node formation. Moreover, RANKL and RANK are expressed in mammary gland epithelial cells and control the development of a lactating mammary gland during pregnancy. Genetically, RANKL and RANK are essential for the development and activation of osteoclasts and bone loss in response to virtually all triggers tested. Inhibition of RANKL function via the natural decoy receptor osteoprotegerin (OPG, TNFRSF11B) prevents bone loss in postmenopausal osteoporosis and cancer metastases. Importantly, RANKL appears to be the pathogenetic principle that causes bone and cartilage destruction in arthritis. RANK-RANKL signaling not only activates a variety of downstream signaling pathways required for osteoclast development, but crosstalk with other signaling pathways also fine-tunes bone homeostasis both in normal physiology and disease. In addition, RANKL and RANK have essential roles in lymph node formation, establishment of the thymic microenvironment, and development of a lactating mammary gland during pregnancy.

Basic Information

Description

Recombinant Mouse TNFSF11/RANKL/CD254 Protein is produced by HEK293 cells expression system. The target protein is expressed with sequence (Arg72-Asp316) of mouse TNFSF11/RANKL/CD254 (Accession #AAC40113.1) fused with a His and hFc tag at the N-terminus.

Bio-Activity

M-CSF (50 ng/mL) and RANKL (100 ng/mL) can induced differentiation of osteoclasts from primary mouse bone marrow cells for 3 days ; RANKL (100 ng/mL) can induced differentiation of osteoclasts from RAW264.7 cells for 5 days .

Storage

Store at -20°C. Store the lyophilized protein at -20°C to -80 °C up to 1 year from the date of receipt. After reconstitution, the protein solution is stable at -20°C for 3 months, at 2-8°C for up to 1 week. Avoid repeated freeze/thaw cycles.

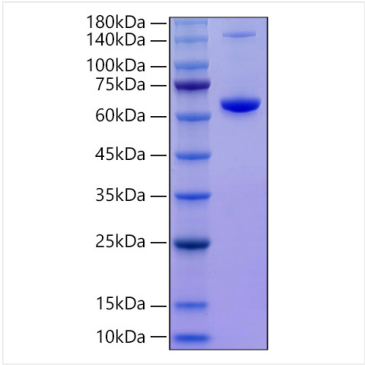
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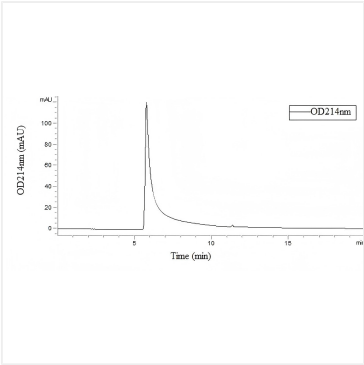
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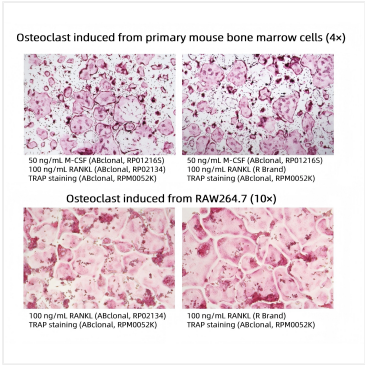
Validation Data



Recombinant Mouse TNFSF11/RANKL/CD254 Protein was determined by SDS-PAGE under reducing conditions with Coomassie Blue.



Recombinant Mouse TNFSF11/RANKL/CD254 Protein is greater than 95% as determined by SEC-HPLC.



M-CSF (RP01216S, 50 ng/mL) and RANKL (100 ng/mL) induced differentiation of osteoclasts from primary mouse bone marrow cells for 3 days (top); RANKL (100 ng/mL) induced differentiation of osteoclasts from RAW264.7 cells for 5 days (below).