

RANKL/TNFSF11 Protein, Mouse (HEK293, Fc)

Cat. No.:	HY-P73388
Synonyms:	Tumor necrosis factor ligand superfamily member 11; RANKL; CD254; ODF; OPGL; TNFSF11; TRANCE
Species:	Mouse
Source:	HEK293
Accession:	AAC40113.1 (R72-D316)
Gene ID:	21943
Molecular Weight:	Approximately 58.46 kDa

PROPERTIES

AA Sequence	<pre> RAQMDPNRIS EDSTHCFYRI LRLHENAGLQ DSTLESEDTL PDCSRRMKQA FQGAVQKELQ HIVGPQRFSG APAMMEGSWL DVAQRGKPEA QPFAHLTINA ASIPSGSHKV T LSSWYHDRG WAKISNMTLS NGKLRVNQDG FYYLYANICF RHHETSGSVP TDY LQLMVYV VKTSIKIPSS HNL MKGGSTK NWSGNSEFHF YSINVGGFFK LRAGEEISIQ VSNPSLLDPD QDATYFGAFK VQDID </pre>
Biological Activity	<p>1. Measured by its ability to induce osteoclast differentiation of RAW 264.7 mouse leukemia cells of monocyte macrophage. The ED₅₀ for this effect is 1.137 ng/mL, corresponding to a specific activity is 8.795×10⁵ units/mg.</p> <p>2. Immobilized mouse Fc-TNFSF11 at 10 µg/mL (100 µl/well) can bind biotinylated human TNFRSF11B-His, The EC₅₀ of biotinylated human TNFRSF11B-His is 0.07-0.17 µg/mL.</p>
Appearance	Lyophilized powder.
Formulation	Lyophilized from a 0.2 µm filtered solution of PBS, pH 7.4 (Normally 5% - 8% trehalose, mannitol and 0.01% Tween 80 are added as protectants before lyophilization.) or 20 mM PB, 150 mM NaCl, pH 7.4.
Endotoxin Level	<1 EU/µg, determined by LAL method.
Reconstitution	It is not recommended to reconstitute to a concentration less than 100 µg/mL in ddH ₂ O.
Storage & Stability	Stored at -20°C for 2 years. After reconstitution, it is stable at 4°C for 1 week or -20°C for longer (with carrier protein). It is recommended to freeze aliquots at -20°C or -80°C for extended storage.
Shipping	Room temperature in continental US; may vary elsewhere.

DESCRIPTION

Background

RANKL (TNFSF11) belongs to TNF family. RANKL is a type II transmembrane protein and is a receptor activator of NF- κ B (RANK) ligand. RANKL is an activator of RANK. RANKL binds to RANK and induces the differentiation of monocyte/macrophage-lineage cells into osteoclasts and leads to osteoclast precursor maturation. In bone tissue, RANKL is expressed by osteoblasts, osteocytes and immune cells, especially in osteoblasts and osteocytes^[1]. RANKL is also expressed by T cells and increases proliferation and survival of dendritic cells^[2]. In mice, RANKL/RANK signaling attenuates inflammation in ischemic brains through a Toll-like receptor signaling pathway^[4].

RANKL consists of cytoplasmic domain (1-47), helical domain (48-68), and extracellular domain (69-317). The soluble chain (140-317) is released when cleaved by enzymes such as matrix metalloproteinases (MMP3 or 7) and ADAM^{[1][3]}.

RANKL is critical for osteoclasts maturation, bone modeling, and bone remodeling, as well as the development of lymph nodes (LNs)^[1].

REFERENCES

- [1]. Ono T, et al. RANKL biology: bone metabolism, the immune system, and beyond. *Inflamm Regen*. 2020 Feb 7;40:2.
- [2]. Li B, et al. Roles of the RANKL-RANK Axis in Immunity-Implications for Pathogenesis and Treatment of Bone Metastasis. *Front Immunol*. 2022 Mar 21;13:824117.
- [3]. Tobeiha M, et al. RANKL/RANK/OPG Pathway: A Mechanism Involved in Exercise-Induced Bone Remodeling. *Biomed Res Int*. 2020 Feb 19;2020:6910312.
- [4]. Shimamura M, et al. OPG/RANKL/RANK axis is a critical inflammatory signaling system in ischemic brain in mice. *Proc Natl Acad Sci U S A*. 2014 Jun 3;111(22):8191-6.
- [5]. He X, et al. Resveratrol prevents RANKL-induced osteoclast differentiation of murine osteoclast progenitor RAW 264.7 cells through inhibition of ROS production. *Biochem Biophys Res Commun*. 2010 Oct 22;401(3):356-62.

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