

## Product Data Sheet

## Animal-Free RANK L/TNFSF11 Protein, Mouse (His)

Cat. No.:	HY-P700226AF
Synonyms:	soluble Receptor Activator of NF-kB Ligand; TNFSF11; TRANCE (TNF-Related Activation-induced Cytokine); OPGL; ODF (Osteoclast Differentiation Factor); CD254; sRNAK Ligand
Species:	Mouse
Source:	E. coli
Accession:	O35235 (P143-D316)
Gene ID:	21943
Molecular Weight:	Approximately 20.35 kDa

PROPERTIES	
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AA Sequence	MPAMMEGSWLDVAQRGKPEAQPFAHLTINAASIPSGSHKVTLSSWYHDRGWAKISNMTLSNGKLRVNQDGFYYLYANICFRHHETSGSVPTDYLQLMVYVVKTSIKIPSSHNLMKGGSTKNWSGNSEFHFYSINVGGFFKLRAGEEISIQVSNPSLLDPDQDATYFGAFKVQDID
<b>Biological Activity</b>	Measure by its ability to induce osteoclast differentiation in RAW264.7 cells. The ED <sub>50</sub> for this effect is <2 ng/mL.
Appearance	Lyophilized powder.
Formulation	Lyophilized from a solution containing 1X PBS, pH 8.0.
Endotoxin Level	<0.1 EU per 1 $\mu$ g of the protein by the LAL method.
Reconsititution	It is not recommended to reconstitute to a concentration less than 100 $\mu\text{g}/\text{mL}$ in ddH_2O.
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). I recommended to freeze aliquots at -20°C or -80°C for extended storage.
Shipping	Room temperature in continental US; may vary elsewhere.

Background RANKL (TNFSF11) belongs to TNF family. RANKL is a type II transmembrane protein and is a receptor activator of NF-κB	DESCRIPTION	
(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, RANK	Background	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 <sup>[1]</sup> . RANKL is also expressed by T cells and increases proliferation and survival of dendritic cells <sup>[2]</sup> . In mice, RANKL/RANK signaling attenuates

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<sup>[1][3]</sup>. RANKL is critical for osteoclasts maturation, bone modeling, and bone remodeling, as well as the development of lymph nodes (LNs)<sup>[1]</sup>.

## 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.

Caution: Product has not been fully validated for medical applications. For research use only.

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