RANK/TNFRSF11A Protein, Rat (HEK293, Fc)

Cat. No.:	HY-P76562
Synonyms:	Tumor necrosis factor receptor superfamily member 11A; ODFR; CD265
Species:	Rat
Source:	HEK293
Accession:	F1M8Z6 (M1-P213)
Gene ID:	498206
Molecular Weight:	Approximately 61 kDa

PROPERTIES

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.
Endotoxin Level	<1 EU/μg, determined by LAL method.
Reconsititution	It is not recommended to reconstitute to a concentration less than 100 μg/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). 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

RANK (TNFRSF11A), is the receptor activator of nuclear factor-κB (NF-κB), has originally been described to play key roles in bone metabolism and the immune system. RANK belongs to tumor necrosis factor receptor superfamily, acts function during osteoclasts differentiation and activation. RANK expressed by osteoblast/stromal cells, ubiquitous expression with high levels in skeletal muscle, thymus, liver, colon, small intestine and adrenal gland. However, osteoblast typically are present in large numbers in giant cell tumors of bone (GCTBs), suggesting the affect of expressing factors in tumors that stimulate osteoclasts precursor recruitment and differentiation[1]. RANK binds RANKL, the receptor activator of NF-κB ligand, to trigger RANKL/RANK/osteoprotegerin (OPG) system to regulate bone resorption. Specifically, the RANKL/RANK signaling pathway promotes the formation of multicellular osteoclast precursors and ensures the activation and survival of multicellular osteoclasts under normal bone remodeling and various pathological conditions. OPG protects bone from excessive bone resorption by competitively binding to RANKL and hindering RANK binding to RANKL[2]. In addition to RANK's contribution to bone metabolism, the RANKL/RANK system is also involved in dendritic cell (DC)-T cell interactions. In rheumatoid synovium and lymph node pairs, the expression levels of RANK and RANKL can be used as markers to determine the interaction between dendritic cells and T cells[3]. Moreover, RANKL-RANK system is critical in the formation of mammary epithelia in lactating females and the thermoregulation of the central nervous system. As them under the tight control of the female sex hormones estradiol and progesterone, RANKL-RANK causes osteoporosis in postmenopausal

women when circulating female sex hormones decrease. Furthermore, RANKL-RANK signaling also plays a critical role in other bone pathologies, bone metastasis or hormone-driven breast cancer[4].

REFERENCES

[1]. Roux S, et al. RANK (receptor activator of nuclear factor kappa B) and RANK ligand are expressed in giant cell tumors of bone. Am J Clin Pathol. 2002 Feb;117(2):210-6.

[2]. Boyce BF, et al. Biology of RANK, RANKL, and osteoprotegerin. Arthritis Res Ther. 2007;9 Suppl 1(Suppl 1):S1.

[3]. Page G, et al. RANK and RANKL expression as markers of dendritic cell-T cell interactions in paired samples of rheumatoid synovium and lymph nodes. Arthritis Rheum. 2005 Aug;52(8):2307-12.

[4]. Nagy V, et al. The RANKL-RANK Story. Gerontology. 2015;61(6):534-42.

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