Revision 6

Store at -20C

#5312

Human RANKL/TRANCE/TNFSF11 (hRANKL)

10 µg

Cell Signaling TECHNOLOGY®

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MW (kDa):	UniProt ID:	Entrez-Gene Id:
30-35	#O14788	8600

Background

RANKL, also known as TRANCE or OPGL, is a member of the TNF superfamily of ligands. T cells, mammary epithelial cells, and endothelial cells can produce RANKL (1). RANKL is expressed as a type II transmembrane protein or cleaved into a soluble form by extracellular proteases, such as TACE, ADAM10, and matrix metalloproteases (1). Alternative splicing also results in the production of soluble RANKL (1). RANKL signaling is antagonized by osteoprotegerin, which functions as a soluble decoy receptor (2). RANKL plays key roles in mammary gland development and dendritic cell survival and is required for osteoclast differentiation and survival (3-6). Research studies have shown that RANKL deficiencies in both mice and humans are associated with abnormally increased bone density and defects in lymphoid organogenesis (5,6).

Endotoxin

Less than 0.01 ng endotoxin/1 µg hRANKL.

Purity

>98% as determined by SDS-PAGE of 6 µg reduced (+) and non-reduced (-) recombinant hRANKL. All lots are greater than 98% pure.

Source / Purification

Recombinant human RANKL (hRANKL) Gly63-Asp244 (Accession #NP_143026) was expressed in human 293 cells at Cell Signaling Technology.

Bioactivity

The bioactivity of hRANKL was determined by measuring the ability of hRANKL to induce TRAP activity in Raw 264.7 cells. The ED_{50} of each lot is between 1.5-5 ng/ml.

Background References

1. O'Brien, C.A. (2010) *Bone* 46, 911-9.
2. Lacey, D.L. et al. (1998) *Cell* 93, 165-76.
3. Wong, B.R. et al. (1997) *J Exp Med* 186, 2075-80.
4. Fata, J.E. et al. (2000) *Cell* 103, 41-50.
5. Kong, Y.Y. et al. (1999) *Nature* 397, 315-23.
6. Conklin, J.L. et al. (1991) *Gastroenterology* 101, 657-63.

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