

# Recombinant Protein Technical Manual Recombinant Rat RANK/TNFRSF11A Protein (Fc Tag)

**RPES0782** 

### **Product Data:**

**Product SKU:** RPES0782 **Size:** 100μg

Species: Rat Expression host: HEK293 Cells

**Uniprot:** NP 001258164.1

## **Protein Information:**

Molecular Mass: 47.1 kDa

AP Molecular Mass: 61 kDa

Tag: C-Fc

**Bio-activity:** 

**Purity:** > 80 % as determined by SDS-PAGE

**Endotoxin:**  $< 1.0 \text{ EU per } \mu \text{g of the protein as determined by the LAL method}$ 

**Storage:** Lyophilized proteins are stable for up to 12 months when stored at -20 to -80°C.

Reconstituted protein solution can be stored at 4-8°C for 2-7 days. Aliquots of

reconstituted samples are stable at < -20°C for 3 months.

**Shipping:** This product is provided as lyophilized powder which is shipped with ice packs.

**Formulation:** Lyophilized from sterile PBS, pH 7.4

**Reconstitution:** Please refer to the printed manual for detailed information.

Application:

Synonyms: RGD1563614

# Immunogen Information:

**Sequence:** Met1-Pro213

# Background:

TNFRSF11A is a member of the TNF-receptor superfamily. In mouse, it is also known as CD265. TNFRSF11A contains 4 TNFR-Cys repeats and is widely expressed with high levels in skeletal muscle, thymus, liver, colon, small intestine and adrenal gland. It is an essential mediator for osteoclast and lymph node development. TNFRSF11A and its ligand are important regulators of the interaction between T cells and dendritic cells. It can interact with various TRAF family proteins, through which this receptor induces the activation of NF-kappa B and MAPK8/JNK. Defects in TNFRSF11A can cause familial expansile osteolysis (FEO). FEO is a rare autosomal dominant bone disorder characterized by focal areas of increased bone remodeling. Defects in TNFRSF11A also can cause Paget disease of bone type 2 (PDB2). PDB2 is a bone-remodeling disorder with clinical similarities to FEO. Defects in TNFRSF11A are the cause of osteopetrosis autosomal recessive type 7 which characterized by abnormally dense bone, due to defective resorption of immature bone.