RANKL Polyclonal Antibody

catalog number: **E-AB-68352**

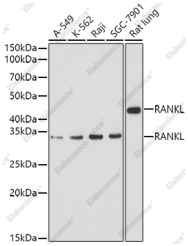
Note: *Centrifuge before opening to ensure complete recovery of vial contents.*

Description

Reactivity	Human;Mouse;Rat
Immunogen	Recombinant fusion protein of human RANKL
Host	Rabbit
Isotype	Rabbit IgG
Purification	Affinity purification
Buffer	Phosphate buffered solution, pH 7.4, containing 0.05% stabilizer and 50% glycerol.

Applications	Recommended Dilution
WB	1:500-1:2000
IF	1:50-1:200

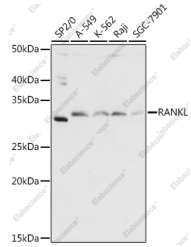
Data



Western blot analysis of extracts of various cell lines using RANKL Polyclonal Antibody at 1:1000 dilution.

Observed-MW:31 kDa/45 kDa

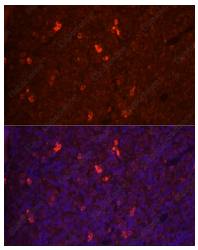
Calculated-MW:27 kDa/30 kDa/35 kDa



Western blot analysis of extracts of various cell lines using RANKL Polyclonal Antibody at 1:1000 dilution.

Observed-MW:31 kDa/45 kDa

Calculated-MW:27 kDa/30 kDa/35 kDa



Immunofluorescence analysis of rat spleen cells using RANKL Polyclonal antibody at dilution of 1:100 (40x lens). Blue: DAPI for nuclear staining.

Preparation & Storage

Storage	Store at -20°C Valid for 12 months. Avoid freeze / thaw cycles.
Shipping	The product is shipped with ice pack,upon receipt,store it immediately at the temperature recommended.

Background

This gene encodes a member of the tumor necrosis factor (TNF) cytokine family which is a ligand for osteoprotegerin and functions as a key factor for osteoclast differentiation and activation. This protein was shown to be a dentritic cell survival factor and is involved in the regulation of T cell-dependent immune response. T cell activation was reported to induce expression of this gene and lead to an increase of osteoclastogenesis and bone loss. This protein was shown to activate antiapoptotic kinase AKT/PKB through a signaling complex involving SRC kinase and tumor necrosis factor receptor-associated factor (TRAF) 6, which indicated this protein may have a role in the regulation of cell apoptosis. Targeted disruption of the related gene in mice led to severe osteopetrosis and a lack of osteoclasts. The deficient mice exhibited defects in early differentiation of T and B lymphocytes, and failed to form lobulo-alveolar mammary structures during pregnancy. Two alternatively spliced transcript variants have been found.

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