CD267 Rabbit pAb

CatalogNo: YT5632

Key Features

Host Species	Reactivity	Applications
• Rabbit	• Human,Mouse	• WB,ELISA

MW	Isotype
• 32kD (Observed)	• IgG

Recommended Dilution Ratios

WB 1:500-1:2000
ELISA 1:10000
Not yet tested in other applications.

Storage

Storage*	-15°C to -25°C/1 year(Do not lower than -25°C)
Formulation	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.

Basic Information

Clonality	Polyclonal

Immunogen Information

Immunogen	The antiserum was produced against synthesized peptide derived from the Internal region of human TNFRSF13B. AA range:81-130
Specificity	CD267 Polyclonal Antibody detects endogenous levels of CD267 protein.

Target Information

Gene name TNFRSF13B

Protein Name Tumor necrosis factor receptor superfamily member 13B

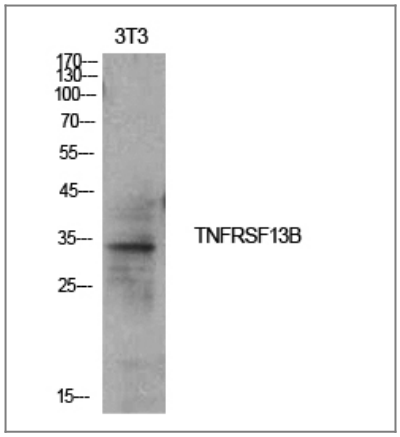
Organism	Gene ID	UniProt ID
Human	23495;	O14836;
Mouse	57916;	Q9ET35;

Cellular Localization Membrane; Single-pass type III membrane protein.

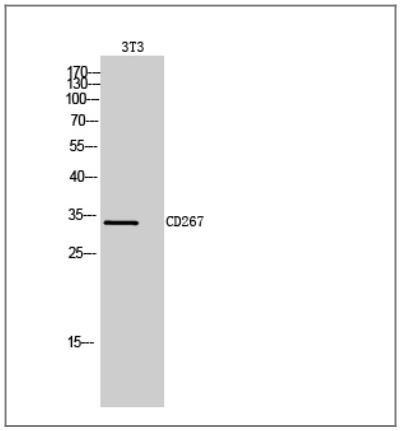
Tissue specificity Highly expressed in spleen, thymus, small intestine and peripheral blood leukocytes. Expressed in resting B-cells and activated T-cells, but not in resting T-cells.

Function Disease:Defects in TNFRSF13B are a cause of common variable immunodeficiency (CVID) [MIM:240500]. CVID is characterized by a deficiency in all immunoglobulin (Ig) isotypes. Individuals with CVID suffer from recurrent sinopulmonary and gastrointestinal infections and have an increased incidence of autoimmune disorders and of lymphoid and non-lymphoid malignancies. There is evidence for a global isotype switching defect in some individuals with CVID. But CVID is a complex and heterogeneous disease in which defects in B-cell survival, number of circulating CD27+ memory B-cells (including IgM+CD27+ B-cells), B-cell activation after antigen receptor cross-linking, T-cell signaling and cytokine expression have been observed.,Disease:Defects in TNFRSF13B are a cause of immunoglobulin A deficiency 2 (IGAD2) [MIM:609529]. Selective deficiency of immunoglobulin A (IGAD) is the most common form of primary immunodeficiency, with an incidence of approximately 1 in 600 individuals in the western world. Individuals with symptomatic IGAD often have deficiency of IgG subclasses or decreased antibody response to carbohydrate antigens such as pneumococcal polysaccharide vaccine. Individuals with IGAD also suffer from recurrent sinopulmonary and gastrointestinal infections and have an increased incidence of autoimmune disorders and of lymphoid and non-lymphoid malignancies. In vitro studies have suggested that some individuals with IGAD have impaired isotype class switching to IgA and others may have a post-switch defect. IGAD and CVID have been known to coexist in families. Some individuals initially present with IGAD1 and then develop CVID. These observations suggest that some cases of IGAD and CVID may have a common etiology.,Function:Receptor for TNFSF13/APRIL and TNFSF13B/TALL1/BAFF/BLYS that binds both ligands with similar high affinity. Mediates calcineurin-dependent activation of NF-AT, as well as activation of NF-kappa-B and AP-1. Involved in the stimulation of B- and T-cell function and the regulation of humoral immunity.,online information:TNFRSF13B mutation db,similarity:Contains 2 TNFR-Cys repeats.,subunit:Binds TRAF2, TRAF5 and TRAF6. Binds the NH2-terminal domain of CAMLG with its C-terminus.,tissue specificity:Highly expressed in spleen, thymus, small intestine and peripheral blood leukocytes. Expressed in resting B-cells and activated T-cells, but not in resting T-cells.,

Validation Data



Western Blot analysis of NIH-3T3 cells using CD267 Polyclonal Antibody. Secondary antibody(catalog#:RS0002) was diluted at 1:20000



Western Blot analysis of 3T3 cells using CD267 Polyclonal Antibody. Secondary antibody(catalog#:RS0002) was diluted at 1:20000

Contact information

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Please scan the QR code to access additional product information:
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