

# TNFRSF21 Polyclonal Antibody

catalog number: AN006810L

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

## Description

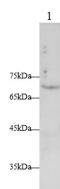
<b>Reactivity</b>	Human
<b>Immunogen</b>	Recombinant Human TNFRSF21 protein expressed by Mammalian
<b>Host</b>	Rabbit
<b>Isotype</b>	IgG
<b>Purification</b>	Antigen Affinity Purification
<b>Conjugation</b>	Unconjugated
<b>buffer</b>	PBS with 0.05% proclin 300, 1% protective protein and 50% glycerol,pH7.4

## Applications

## Recommended Dilution

<b>WB</b>	1:500-1:1000
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## Data



Western blot with Anti TNFRSF21 Polyclonal antibody at dilution of 1:1000. Lane 1: 293 cell lysate.

**Observed-MV:70 kDa**

**Calculated-MV:72 kDa**

## Preparation & Storage

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

## Background

Death Receptor 6 (DR6), also known as TNFRSF21 and CD358, is a type I transmembrane protein in the TNF receptor superfamily. Human DR6 consists of a 308 amino acid (aa) extracellular domain (ECD) with four cysteine-rich motifs, a 21 aa transmembrane segment, and a 285 aa palmitoylated cytoplasmic region that contains one death domain. Within the ECD, human and mouse DR6 share 82% aa sequence identity. DR6 is expressed as an approximately 110 kDa molecule that carries extensive N-linked and O-linked glycosylation in its extracellular region. Among hematopoietic cells, DR6 is expressed on monocytes, resting CD4<sup>+</sup> T cells, and pro-, pre-, and naïve B cells. DR6 knockout mice exhibit a Th2-biased immune response characterized by exaggerated Th2 and B cell responsiveness in combination with reduced Th1 cell responsiveness and inflammatory leukocyte infiltration. DR6 knockout mice are resistant to induced airway inflammation and experimental autoimmune encephalitis but more susceptible to severe graft versus host disease. DR6 is also expressed on developing neurons where it can bind a shed 35 kDa N-terminal fragment of APP or a fragment of APLP2. This APP fragment is generated following deprivation of neurotrophic factors, and its binding to DR6 triggers DR6-mediated axonal pruning. DR6 is constitutively expressed on some prostate cancer cells and can be induced by TNF- $\alpha$  on others.

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