

## Recombinant IL18RAP/IL1R7 Monoclonal Antibody

catalog number: **AN300305P**

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

### Description

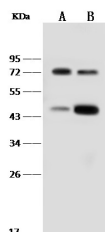
<b>Reactivity</b>	Human
<b>Immunogen</b>	Recombinant Human IL18RAP/IL1R7 Protein
<b>Host</b>	Rabbit
<b>Isotype</b>	IgG
<b>Clone</b>	B227
<b>Purification</b>	Protein A
<b>Buffer</b>	0.2 µm filtered solution in PBS

### Applications

### Recommended Dilution

<b>WB</b>	1:500-1:2000
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### Data



Western Blot with IL1R7 Monoclonal Antibody at dilution of 1:500 dilution. Lane A: RAW264.7 Whole Cell Lysate, Lane B: K562 Whole Cell Lysate, Lysates/proteins at 30 µg per lane.

**Observed-MW:75 kDa**

**Calculated-MW:68 kDa**

### Preparation & Storage

<b>Storage</b>	This antibody can be stored at 2°C-8°C for one month without detectable loss of activity. Antibody products are stable for twelve months from date of receipt when stored at -20°C to -80°C. Preservative-Free. Avoid repeated freeze-thaw cycles.
<b>Shipping</b>	Ice bag

### Background

Interleukin 18 receptor accessory protein, also known as IL18RAP and CDw218b (cluster of differentiation w218b), is an accessory subunit of the heterodimeric receptor for IL18. This protein enhances the IL18 binding activity of IL18R1 (IL1RRP), a ligand-binding subunit of the IL18 receptor. The coexpression of IL18R1 and this protein is required for the activation of NF-kappaB and MAPK8 (JNK) in response to IL18. IL18RAP is required for the high-affinity binding of interleukin 18 (IL-18) to its receptor complex. IL18RAP together with IL18R1 mediates IL-18-dependent activation of NF-kappa-B and JNK. Two putative isoforms of IL18RAP were detected and the ratios and total levels of these isoforms may contribute to the etiology of coeliac disease. IL18R1 and IL18RAP polymorphisms have been found associated with diseases such as schizophrenia, HSV1 seropositivity, and atopic asthma. Analysis of IL18R1 and IL18RAP SNPs in 5 European prospective cohorts suggests that the variability of these genes are unlikely to contribute to modulate the risk of CVD in European populations.

### For Research Use Only

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