

Recombinant Protein Technical Manual Recombinant Human IL18RAP/IL1R7 Protein (His Tag)(Active) RPES0892

Product Data:

Product SKU: RPES0892

Size: 20µg

Species: Human

Expression host: HEK293 Cells

Uniprot: NP_003844.1

Protein Information:	
Molecular Mass:	40 kDa
AP Molecular Mass:	50-55 kDa
Tag:	C-His
Bio-activity:	Measured by its ability to bind biotinylated Cynomolgus IL18 in a functional ELISA.
Purity:	> 97 % as determined by reducing SDS-PAGE.
Endotoxin:	< 1.0 EU per μg 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:	Functional ELISA
Synonyms:	ACPL;CD218b;CDw218b;IL8R-beta;IL8RAcP;IL8Rbeta;ILR-7;ILR7;ILRAcPL;IL18RB

Sequence: Met 1-Gly 357

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 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 (IL8) to its receptor complex. IL18RAP together with IL18R1 mediates IL8-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 aetiology 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.