

Recombinant Human IFNAR1/IFNAR Protein (Fc Tag)

Catalog Number:PKSH030696



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

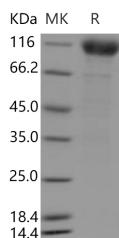
Description

Synonyms	Interferon Alpha/Beta Receptor 1;IFN-R-1;IFN-Alpha/Beta Receptor 1;Cytokine Receptor Class-II Member 1;Cytokine Receptor Family 2 Member 1;CRF2-1;Type I Interferon Receptor 1;IFNAR1;IFNAR;AVP;IFN-alpha-REC
Species	Human
Expression Host	HEK293 Cells
Sequence	Met 1-Lys 436
Accession	P17181-1
Calculated Molecular Weight	74.0 kDa
Observed molecular weight	100-120 kDa
Tag	C-hFc

Properties

Purity	> 88 % as determined by reducing SDS-PAGE.
Endotoxin	< 1.0 EU per µg of the protein as determined by the LAL method.
Storage	Generally, 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 Normally 5 % - 8 % trehalose, mannitol and 0.01% Tween80 are added as protectants before lyophilization. Please refer to the specific buffer information in the printed manual.
Reconstitution	Please refer to the printed manual for detailed information.

Data



> 88 % as determined by reducing SDS-PAGE.

Background

Interferon-alpha/beta receptor alpha chain (IFNAR1) is a type I membrane protein that forms one of the two chains of a receptor for interferons alpha and beta. Binding and activation of the receptor stimulates Janus protein kinases; which in turn phosphorylate several proteins; including STAT1 and STAT2. The encoded protein also functions as an antiviral factor. Tyk2 slows down IFNAR1 degradation and that this is due; at least in part; to inhibition of IFNAR1 endocytosis. Mutant versions of IFNAR1; in which Tyr466 is changed to phenylalanine; can act in a dominant negative manner to inhibit phosphorylation of STAT2. These observations are consistent with a model in which IFNAR1 mediates the

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interaction between JAK kinases and the STAT transcription factors.

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