Recombinant Mouse IFNAR1/IFNAR Protein (His Tag)

Catalog No. PKSM040664

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

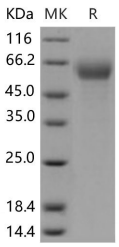
Description

Synonyms	CD118;Ifar;Ifnar;Ifrc;Infar
Species	Mouse
Expression Host	HEK293 Cells
Sequence	Met 1-Thr 429
Accession	NP_034638.2
Calculated Molecular Weight	47.0 kDa
Observed molecular weight	55-60 kDa
Tag	C-His
Bioactivity	Measured by its ability to inhibit mIFNB1-mediated protection of L929 mouse fibroblast cells infected with vesicular stomatitis virus (VSV) to viral lysis. The ED50 for this effect is typically 0.2-1μg/mL.

Properties

Purity	> 96 % 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℃. Reconstituted protein solution can be stored at 4-8℃ for 2-7 days. Aliquots of reconstituted samples are stable at < -20℃ 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



> 96 % 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 interaction between JAK kinases and the STAT transcription factors.

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