

Recombinant Mouse IFNGR2 Protein (His Tag)

Catalog Number: PKSM040555

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

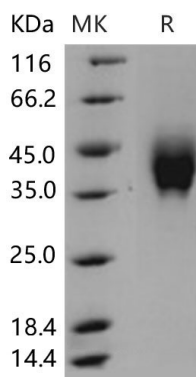
Description

Species	Mouse
Source	HEK293 Cells-derived Mouse IFNGR2 protein Met 1-Val 243, with an C-terminal His
Mol_Mass	26.7 kDa
Accession	NP_032364.1
Bio-activity	Not validated for activity

Properties

Purity	> 97 % 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% Tween 80 are added as protectants before lyophilization.
Reconstitution	Please refer to the specific buffer information in the printed manual. Please refer to the printed manual for detailed information.

Data



> 97 % as determined by reducing SDS-PAGE.

Background

For Research Use Only

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Interferon gamma receptor beta chain (IFNgammaR2), also known as IFNGR2, belongs to the type II cytokine receptor family, whose deficiency is a cause of autosomal recessive mendelian susceptibility to mycobacterial disease (MSMD), also known as familial disseminated atypical mycobacterial infection. This accessory factor is an integral part of the IFN-gamma signal transduction pathway and is likely to interact with GAF, JAK1, and/or JAK2. IFNGR2 is a component of the IFNgamma receptor complex along with the IFNgammaR alpha chain (IFNGR1), and is a new Bax suppressor. The C-terminal fragment (cytoplasmic domain) of IFNgammaR2 is expressed in human cancer cell lines of megakaryocytic cancer (DAMI), breast cancer (MDA-MD-468), and prostate cancer (PC3 cells). The Th1 cytokine IFNgamma, acting through its heterodimeric receptors, IFNgammaR1 and IFNgammaR2, in the induction/proliferation of Th1 cells, might suppress the Th2 responses that may underlie atopic asthma. IFNGR2 has always been seen as a key mechanism for shielding T lymphocytes from the antiproliferative effects of the IFNgamma-signal transducer and activator of transcription 1 (STAT1) pathway.

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