Recombinant Human IFNα2 Protein(Sumo Tag)

Catalog Number: PDEH101120

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

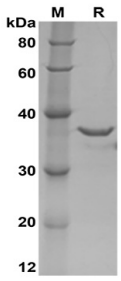
Description

Species	Human
Source	E.coli-derived Human IFNα2 protein Cys24-Glu188, with an N-teminal Sumo
Mol_Mass	31.0 kDa
Accession	P01563
Bio-activity	Not validated for activity

Properties

Purity	> 95% as determined by reducing SDS-PAGE.
Endotoxin	< 10 EU/mg 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 a 0.2 μm filtered solution in PBS with 5% Trehalose and 5% Mannitol.
Reconstitution	It is recommended that sterile water be added to the vial to prepare a stock solution of 0.5 mg/mL. Concentration is measured by UV-Vis

Data



SDS-PAGE analysis of Human IFNα2 proteins, 2μg/lane of Recombinant Human IFNα2 proteins was resolved with SDS-PAGE under reducing conditions, showing bands at 38 kDa

Background

Interferon-alpha 2 (IFN alpha-2) is one of 14 subtypes with anin the IFN-alpha class of Type I Interferons. The members of the IFN-alpha class, also known as alpha leukocyte interferons, encompass a group of distinct but closely related proteins which share approximately 80% amino acid (aa) sequence identity and have a similar globular structure composed of five alpha-helices. IFN-alpha class members signal through a common cell surface receptor complex composed of IFN-alpha R2 and IFN-alpha R1 subunits. As the first highly active IFN to be cloned and produced, IFN alpha-2 has become the prototypic IFN for academic and pharmaceutical research. The mature extracellular domain (ECD) of mouse IFN alpha-2 shares 60% and 83% aa sequence identity with an human and rat, respectively. Murine IFN-alpha 2 can eliminate cardiac viral load and protect cardiomyocytes from injury in animals infected with an coxsackievirus B3 (CVB3). IFN alpha-2 derived mutants with an reduced IFNR2 binding inhibited HIV replication and mutants with an more IFNAR1 binding potentiated antiviral activity.

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