

Recombinant Human IFN-beta

Accession #	P01574
Alternate Names	Fibroblast interferon; IFB; IFBIFNB; IFF; IFNB; IFNB1; IFNbeta; IFN-beta; interferon beta
Source	Human embryonic kidney cell, HEK293-derived human IFN-beta protein
Protein sequence	Met22-Asn187
M.Wt	20.0 kDa
Appearance	Solution protein.
Stability & Storage	Avoid repeated freeze-thaw cycles. It is recommended that the protein be aliquoted for optimal storage. 12 months from date of receipt, -20 to -70 °C as supplied.
Concentration	0. 2 mg/mL
Formulation colors	Dissolved in sterile PBS buffer.
Reconstitution	We recommend that this vial be briefly centrifuged prior to opening to bring the contents to the bottom. This solution can be diluted into other aqueous buffers.
Biological Activity	Measured in anti-viral assays using HeLa human cervical epithelial carcinoma cells infected with encephalomyocarditis (EMC) virus. The EC50 for this effect is 1-10 ng/mL
Shipping Condition	Shipping with dry ice.
Handling	Centrifuge the vial prior to opening.
Usage	For Research Use Only! Not to be used in humans.
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Quality Control

Purity	> 95%, determined by SDS-PAGE.
Endotoxin	< 0.010 EU per 1 ug of the protein by the LAL method.

Description

Interferon beta (IFN-beta), also known as fibroblast IFN, is a secreted, approximately 22 kDa member of the type I interferon family of molecules ^[1]. Mature human IFN-beta shares 47% and 46% amino acid sequence identity with the mouse and rat proteins, respectively. Fibroblasts are the major producers of IFN-beta, but it can also be produced by dendritic cells, macrophages, and endothelial cells in response to pathogen exposure ^[2]. It is trans -criptionally regulated by TRAF3, IRF3, IRF7, and NF-kappa B ^[3]. Following secretion, IFN-beta signals through the heterodimeric IFN-alpha / beta Receptor and activates the JAK/STAT signaling pathway ^[4-7]. IFN-beta -deficient mice show increased susceptibility to experimental autoimmune encephalomyelitis (EAE), a disease model of human multiple sclerosis (MS) ^[8]. Furthermore, IFN-beta has been shown to suppress the Th17 cell response in both MS and EAE and has commonly been used as a treatment for MS ^[9-13].

Reference

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