Revision 1

Human Interferon-γ (IFN-γ)

#3158 Store at +4C

100 µg

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MW (kDa):	UniProt ID:	Entrez-Gene Id:
16.9	#P01579	3458

Background

Interferons (IFNs) appear both locally and systematically early after viral infection and participate in limiting the spread of infection. They also affect cell differentiation, growth, surface antigen expression, and immunoregulation (1). There are three naturally occurring interferons: α, β, and γ. IFN-α is derived from lymphoblastic tissue and has a number of therapeutic applications in the treatment of various human cancers and diseases of viral origin. Recombinant IFN-α from both natural and synthetic genes binds to a common cell surface receptor and induces antiviral activity in a variety of cell lines. When binding to discrete cell surface receptors on target cells, IFN-α induces rapid changes in Jak/Stat phosphorylation, which initiates the Jak/Stat signaling pathway (2). IFN-α signaling also involves production of DAG without an increased intracellular free calcium concentration and the subsequent activation of calcium-independent isoforms of PKC (β and ε) (3). All IFN-α signaling pathways lead to final alterations of gene expression, which mediate their pleiotropic biologic activities.

IFN-γ, also known as type II interferon, is produced mainly in activated T lymphocytes and natural killer cells (4) and has broad effects on various cells of the immune system. Synthesis of IFN-γ is induced by many signaling proteins including IL-2, FGF, and EGF.

Purity

>95%

Source / Purification

Recombinant human IFN-γ expressed in *E. coli*

Bioactivity

Specific activity as determined by a viral resistance assay was less than 0.05 ng/ml.

Background References

1. Stiehm, E.R. et al. (1982) *Ann Intern Med* 96, 80-93.
2. Pellegrini, S. et al. (1989) *Mol Cell Biol* 9, 4605-12.
3. Pfeffer, L.M. and Colamonici, O.R. (1991) *Pharmacol Ther* 52, 149-57.
4. Young, H.A. and Hardy, K.J. (1995) *J. Leukoc. Biol.* 58, 373-381.

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Limited Uses