

Human IFN-α 2b Recombinant Protein



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MW (kDa): UniProt ID: **Entrez-Gene Id:** #P01563 3440 19.4

Background

20 µg

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.

The human IFN- α 2 subvariants 2a and 2b differ by one or two amino acids at positions 23 and/or 34 of the mature protein (4). Pegylated forms of IFN-α 2 and recombinant IFN-α 2 protein are used clinically as standard treatments for anti-viral and anti-tumor therapies (5). IFN- α 2a and 2b proteins have also been shown to have implications in treating hepatitis C (6).

Endotoxin

Endotoxin levels are less than or equal to 1 EU / 1 μ g hIFN- α 2b.

Purity

A greater than or equal to 95% purity was determined by SDS-PAGE.

Source / Purification

Recombinant human IFN-α 2b was expressed in *E. coli* and is supplied in a lyophilized form.

Bioactivity

The bioactivity of recombinant hIFN-α 2b was determined in a viral CPE assay using EMC virus on A549 cells. The ED₅₀ of each lot is greater than or equal to 2.0×10^8 units/mg.

Background References

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- 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. von Gabain, A. et al. (1990) Eur J Biochem 190, 257-61.
- 5. CA Cancer J Clin (1988) 38, 280-90.
- 6. Castelruiz, Y. et al. (1999) Hepatology 29, 1900-4.

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