Recombinant Human IFN-γ (HEK293-expressed)

Catalog Number: 10067-IF

DESCRIPTION

Source	Human embryonic kidney cell, HEK293-derived human IFN-gamma protein Met1-Gln166 Accession # NP_000610.2
N-terminal Sequence Analysis	Gln24 inferred from enzymatic pyroglutamate treatment revealing Asp25.
Structure / Form	Noncovalent homodimer
Predicted Molecular Mass	16.8 kDa

SPECIFICATIONS

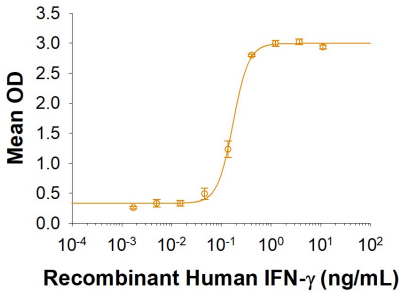
SDS-PAGE	19-25 kDa, reducing conditions
Activity	Measured in anti-viral assays using HeLa human cervical epithelial carcinoma cells infected with encephalomyocarditis (EMC) virus. Meager, A. (1987) in Lymphokines and Interferons, a Practical Approach. Clemens, M.J. *et al.* (eds): IRL Press. 129. The ED_{50} for this effect is 0.06-0.36 ng/mL.
Endotoxin Level	<0.10 EU per 1 µg of the protein by the LAL method.
Purity	>95%, by SDS-PAGE visualized with Silver Staining and quantitative densitometry by Coomassie® Blue Staining.
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 250 µg/mL in PBS.
Shipping	The product is shipped with polar packs. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	**Use a manual defrost freezer and avoid repeated freeze-thaw cycles.** • 12 months from date of receipt, -20 to -70 °C as supplied. • 1 month, 2 to 8 °C under sterile conditions after reconstitution. • 3 months, ≤ -20 °C under sterile conditions after reconstitution.

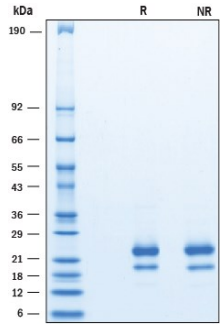
DATA

Bioactivity



Recombinant Human IFN-γ (Catalog # 10067-IF) demonstrates anti-viral activity in HeLa human cervical epithelial carcinoma cells infected with encephalomyocarditis (EMC) virus. The ED_{50} for this effect is 0.06-0.36 ng/mL

SDS-PAGE



2 µg/lane of Recombinant Human IFN-γ (HEK293-expressed) was resolved with SDS-PAGE under reducing (R) and non-reducing (NR) conditions and visualized by Coomassie® Blue staining, showing bands at 19-25 kDa.

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BACKGROUND

Interferon-gamma (IFN-gamma), also known as type II or immune interferon, exerts a wide range of immunoregulatory activities and is considered to be the prototype proinflammatory cytokine (1, 2). Mature human IFN-gamma exists as a non-covalently linked homodimer of 20-25 kDa variably glycosylated subunits (3). Glycosylation of IFN-gamma at sites Asn25 and Asn97 is critical for protease resistance (4, 5). The mature protein shares 36% and 35% amino acid (aa) sequence identity with mouse and rat IFN-gamma, respectively. IFN-gamma dimers bind to IFN-gamma RI (alpha subunits) which then interact with IFN-gamma RII (beta subunits) to form the functional receptor complex of two alpha and two beta subunits. Inclusion of IFN-gamma RII increases the binding affinity for ligand and the efficiency of signal transduction (6, 7). IFN-gamma is produced by a variety of immune cells under inflammatory conditions, notably by T cells and NK cells (8). It plays a key role in host defense by promoting the development and activation of Th1 cells, chemoattraction and activation of monocytes and macrophages, up-regulation of antigen presentation molecules, and immunoglobulin class switching in B cells. It also exhibits antiviral, antiproliferative, and apoptotic effects (8, 9). In addition, IFN-gamma functions as an anti-inflammatory mediator by promoting the development of regulatory T cells and inhibiting Th17 cell differentiation (10, 11). The pleiotropic effects of IFN-gamma contribute to the development of multiple aspects of atherosclerosis (9).

References:

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