

DESCRIPTION

Source Human embryonic kidney cell, HEK293-derived
Ile22-Asn182
Accession # P01575

N-terminal Sequence Analysis Ile22

Structure / Form Monomer

Predicted Molecular Mass 20 kDa

SPECIFICATIONS

SDS-PAGE 30-38 kDa, reducing conditions

Activity Measured in an anti-viral assay using L-929 mouse fibroblast cells infected with encephalomyocarditis (EMC) virus. Vogel, S.N. *et al.* (1982) *Infect. Immunol.* **38**:681.
The ED₅₀ for this effect is typically 1-6 pg/mL.

The specific activity of recombinant Mouse IFN-β is approximately 1.2 x 10⁹ IU/mg, which is calibrated against Murine IFN-β WHO International Standard. The Murine IFN-β WHO International Standard (NR-3079) was obtained through the NIH Biodefense and Emerging Infections Research Resources Repository, NIAID, NIH.

Endotoxin Level <0.10 EU per 1 μg of the protein by the LAL method.

Purity >95%, by SDS-PAGE with silver staining.

Formulation Lyophilized from a 0.2 μm filtered solution in PBS and Tween® 80 with BSA as a carrier protein. See Certificate of Analysis for details.

PREPARATION AND STORAGE

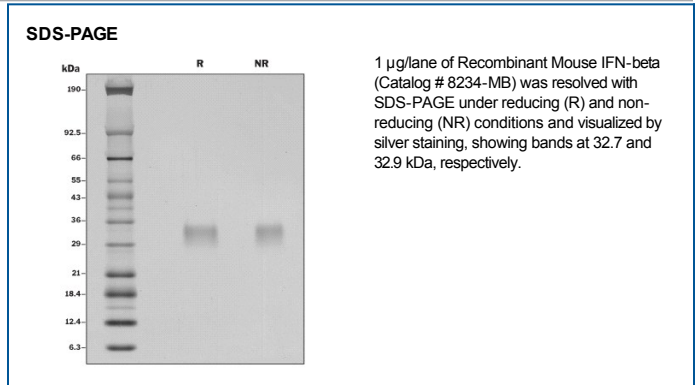
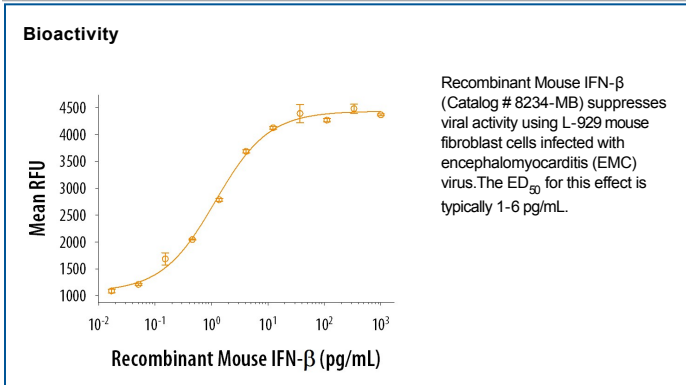
Reconstitution Reconstitute at 100 μg/mL in sterile PBS containing at least 0.1% human or bovine serum albumin.

Shipping The product is shipped at ambient temperature. 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 to -70 °C under sterile conditions after reconstitution.

DATA



BACKGROUND

Interferon beta (IFN- β), also known as fibroblast IFN, is a secreted, approximately 22 kDa member of the type I interferon family of molecules (1). Mature mouse IFN- β shares 75% and 47% amino acid sequence identity with the rat and human proteins, respectively. Fibroblasts are the major producers of IFN- β , but it can also be produced by dendritic cells, macrophages, and endothelial cells in response to pathogens (2). It is transcriptionally regulated by TRAF3, IRF3, IRF7, and NF- κ B (3, 4). IFN- β -deficient mice show increased susceptibility to experimental autoimmune encephalomyelitis (EAE), a disease model of human multiple sclerosis (MS) (5). Furthermore, IFN- β has been shown to suppress the Th17 cell response in both MS and EAE and has commonly been used as a treatment for MS (6-10). IFN- β can additionally induce the expression of the anti-inflammatory cytokine IL-10 (11).

References:

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4. Häcker, H. *et al.* (2006) *Nature* **439**:204.
5. Teige, I. *et al.* (2003) *J. Immunol.* **170**:4776.
6. Shinohara, M.L. *et al.* (2008) *Immunity* **29**:68.
7. Guo, B. *et al.* (2008) *J. Clin. Invest.* **118**:1680.
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9. Martín-Saavedra, F.M. *et al.* (2008) *Mol. Immunol.* **45**:4008.
10. Inoue, M. and M.L. Shinohara (2013) *Immunology* **139**:11.
11. Wang, H. *et al.* (2011) *J. Immunol* **186**:675.