Recombinant Mouse Interferon γ/IFNG Protein (E.coli)

Catalog Number: PKSM041063

Note: *Centrifuge before opening to ensure complete recovery of vial contents.*

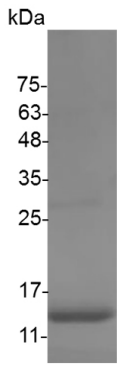
Description

Species	Mouse
Source	E.coli-derived Mouse Interferon γ/IFNG protein His23-Cys155, with an C-terminal His
Calculated MW	16.5 kDa
Observed MW	16 kDa
Accession	P01580
Bio-activity	Measure by its ability to anti-viral assay in L-929 cells infected with encephalomyocarditis (EMC) virus.The ED_{50} for this effect is <0.5 ng/mL.The specific activity of recombinant mouse IFN gamma is approximately >2x 10^6 IU/mg.

Properties

Purity	> 98 % as determined by reducing SDS-PAGE.
Endotoxin	< 0.01 EU per μg of the protein as determined by the LAL method.
Storage	Generally, lyophilized proteins are stable for up to 12 months when stored at -20 to -80 ℃. Reconstituted protein solution can be stored at 4-8℃ for 2-7 days. Aliquots of reconstituted samples are stable at < -20℃ for 3 months.
Shipping	This product is provided as lyophilized powder which is shipped with ice packs.
Formulation	Lyophilized from sterile PBS, pH 7.4. Normally 5% - 8% trehalose, mannitol and 0.01% Tween 80 are added as protectants before lyophilization. Please refer to the specific buffer information in the printed manual.
Reconstitution	Please refer to the printed manual for detailed information.

Data



> 98 % as determined by reducing SDS-PAGE.

Background

Mouse Ifng is a secreted protein which belongs to the type I I (or gamma) interferon family. IFNG is produced by lymphocytes and activated by specific antigens or mitogens. In addition to having antiviral activity, IFNG also has important immunoregulatory functions. It is a potent activator of macrophages and has antiproliferative effects on transformed cells. It can potentiate the antiviral and antitumor effects of the type I interferons. Genetic variation in IFNG is associated with the risk of aplastic anemia (AA) which is a rare disease in which the reduction of the circulating blood cells results from damage to the stem cell pool in bone marrow. In most patients, the stem cell lesion is caused by an autoimmune attack. T-lymphocytes, activated by an endogenous or exogenous, and most often unknown antigenic stimulus, secrete cytokines, including IFN-gamma, which would in turn be able to suppress hematopoiesis.

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