

DESCRIPTION

Source *E. coli*-derived
Asn36-Ser192
Accession # P70380

N-terminal Sequence Analysis Asn36 & Leu40

Predicted Molecular Mass 18 kDa

SPECIFICATIONS

SDS-PAGE 17 kDa, reducing conditions

Activity Measured by its ability to induce mouse IFN- γ secretion by activated mouse T cells. The ED₅₀ for this effect is typically 0.06-0.36 ng/mL.

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

Purity >90%, by SDS-PAGE visualized with Silver Staining and quantitative densitometry by Coomassie® Blue Staining.

Formulation Lyophilized from a 0.2 μ m filtered solution in PBS, EDTA and DTT with trehalose. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution Reconstitute at 100 μ g/mL in PBS.

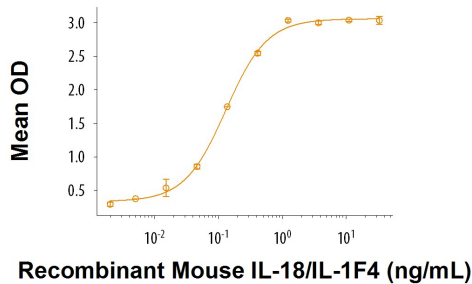
Shipping The product is shipped with polar packs. Upon receipt, store it immediately at the temperature recommended below.

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 to -70 °C under sterile conditions after reconstitution.

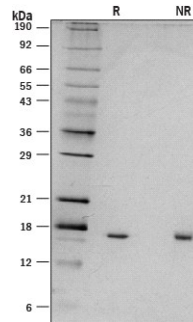
DATA

Bioactivity



Recombinant mouse IL-18/IL-1F4 (Catalog # 9139-IL/CF) induces IFN γ secretion by activated mouse T cells. The ED₅₀ for this effect is typically 0.06-0.36 ng/mL.

SDS-PAGE



1 μ g/lane of Recombinant Mouse IL-18 was resolved with SDS-PAGE under reducing (R) and non-reducing (NR) conditions and visualized by silver staining, showing a single band at 18 kDa.

BACKGROUND

Interleukin-18 (IL-18) is a proinflammatory cytokine in the IL-1 family that exerts distinct immune effects depending on the local cytokine environment. It is expressed as a 24 kDa precursor by endothelial and epithelial cells, keratinocytes, $\gamma\delta$ T cells, and phagocytes. The precursor is activated intracellularly by Caspase-1 mediated proteolysis to release the 17 kDa mature cytokine. The precursor can also be released by necrotic cells for extracellular cleavage by multiple proteases. IL-18 activation is induced by infection or tissue damage and contributes to disease pathology in chronic inflammation (1-3). IL-18 binds to the widely expressed IL-18 R α which recruits IL-18 R β to form the signaling receptor complex (4, 5). Its bioactivity is negatively regulated by interactions with IL-18 binding proteins and virally encoded IL-18BP homologs (6). In the presence of IL-12 or IL-15, IL-18 enhances anti-viral Th1 immune responses by inducing IFN- γ production and the cytolytic activity of CD8⁺ T cells and NK cells (7, 8). In the absence of IL-12 or IL-15, however, IL-18 promotes production of the Th2 cytokines IL-4 and IL-13 by CD4⁺ T cells and basophils (9, 10). In the presence of IL-1 β or IL-23, IL-18 induces the antigen-independent production of IL-17 by $\gamma\delta$ T cells and CD4⁺ T cells (11). IL-18 also promotes myeloid dendritic cell maturation and triggers neutrophil respiratory burst (12, 13). In cancer, IL-18 exhibits diverse activities including enhancing anti-tumor immunity, inhibiting or promoting angiogenesis, and promoting tumor cell metastasis (14). Mature mouse IL-18 shares 63% and 91% amino acid sequence identity with mouse and rat IL-18, respectively (15).

References:

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