

DESCRIPTION

Source *E. coli*-derived
Gln27-Leu181, with an N-terminal Met
Accession # Q925S4

N-terminal Sequence Analysis Met

Predicted Molecular Mass 18.1 kDa

SPECIFICATIONS

SDS-PAGE 16 kDa, reducing conditions

Activity Measured in a cell proliferation assay using BaF3 mouse pro-B cells transfected with human IL-20 R α and human IL-20 R β . The ED₅₀ for this effect is typically 50-300 ng/mL.

Endotoxin Level <0.01 EU per 1 μ g of the protein by the LAL method.

Purity >95%, by SDS-PAGE under reducing conditions and visualized by silver stain.

Formulation Lyophilized from a 0.2 μ m filtered solution in Acetonitrile and TFA. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution Reconstitute at 100 μ g/mL in 4 mM HCl.

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.

BACKGROUND

Interleukin 24 (IL-24), also known as FISP (IL-4 induced secreted protein) in mouse, and mda-7 (melanoma differentiation associated gene-7) in humans, is a member of the IL-10 family of helical cytokines (1 - 3). The mouse IL-24 gene encodes for a 181 amino acid (aa) precursor that includes a 26 aa signal sequence and an 18 kDa (predicted), 155 aa mature segment (4). Unlike mature human IL-24, which contains three potential N-linked glycosylation sites, mouse mature IL-24 has only one. In human, both glycosylation and the presence of an intrachain disulfide bond have been found to be necessary for full activity (5). This requirement for glycosylation is unique among IL-10 family members. Presumably, the same structural modifications apply to mouse IL-24. Mouse IL-24 is a 27 kDa protein when secreted, and is active on human receptors (1, 3). Mature mouse IL-24 shares 69%, 84%, 69%, 68%, and 61% aa sequence identity with human, rat, equine, canine and bovine IL-24, respectively. A 119 aa isoform termed FISP-sp diverges at aa 77. FISP-sp has been found mainly in the endoplasmic reticulum of Th2 cells. It can dimerize with intracellular IL-24, and antagonize the pro-apoptotic effects of IL-24 (6). Under physiological conditions, cytokine-stimulated monocytes/macrophages and Th2 cells produce most secreted IL-24; other sources include B cells, keratinocytes, NK cells, and differentiating melanoma cells (1 - 3, 7 - 9). Secreted IL-24 binds and signals through complexes of IL-20 R α :IL-20 R β , or IL-22 R:IL-20 R β (10). The IL-20 R α :IL-20 R β complex is also a receptor for IL-19 and IL-20, while IL-22 R:IL-20 R β binds IL-20 (2, 10). Both receptors are widely expressed, but have not been found on hematopoietic cells (3). Even so, IL-24 induces type I proinflammatory cytokines in monocytes, and inhibits plasma cell differentiation in germinal center B cells (8, 11). The phenotype of mice transgenic for IL-24 is similar to that of IL-20 and IL-22 transgenic mice, indicating overlap in their activities, particularly in the skin (12). Secreted IL-24 is anti-angiogenic, binding receptors on endothelial cells and blocking their differentiation (13). Intratumoral injection of adenovirus coding for IL-24 causes tumor-specific apoptosis in humans, but it is not clear whether secreted IL-24 contributes to this effect (14).

References:

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