Recombinant Human IL-13 R alpha 2 His-tag

Catalog Number: 11562-IR

DESCRIPTION

Source	Human embryonic kidney cell, HEK293-derived human IL-13 R alpha 2 protein Pro43-Leu342, with a C-terminal 6-His tag Accession # Q14627.1
N-terminal Sequence Analysis	Pro 43
Predicted Molecular Mass	36 kDa

SPECIFICATIONS

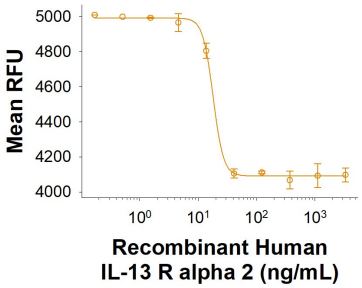
SDS-PAGE	53-58 kDa, under reducing conditions
Activity	Measured by its ability to inhibit IL-13-dependent proliferation of TF-1 human erythroleukemic cells. The ED_{50} for this effect is 4.00-60.0 ng/mL.
Endotoxin Level	<0.10 EU per 1 µg of the protein by the LAL method.
Purity	>95%, by SDS-PAGE visualized with Silver Staining and quantitative densitometry by Coomassie® Blue Staining.
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS with Trehalose. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 200 µg/mL in PBS.
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.

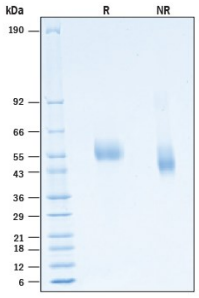
DATA

Bioactivity



Recombinant Human IL-13 R alpha 2 His-tag Protein Bioactivity. Recombinant Human IL-13 R alpha 2 His-tag Protein (Catalog # 11562-IR) inhibits Recombinant Human IL-13 (Catalog # 213-ILB) induced proliferation in the TF-1 human erythroleukemic cells. The ED_{50} for this effect is 4.00-60.0 ng/mL.

SDS-PAGE



Recombinant Human IL-13 R alpha 2 His-tag Protein SDS-PAGE. 2 µg/lane of Recombinant Human IL-13 R alpha 2 His-tag Protein (Catalog # 11562-IR) was resolved with SDS-PAGE under reducing (R) and non-reducing (NR) conditions and visualized by Coomassie® Blue staining, showing bands at 53-58 kDa, under reducing conditions.

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BACKGROUND

Interleukin-13 Receptor alpha 2 (IL-13 Rα2), also known as IL-13 binding protein, and CD213a2, is a widely expressed 55 kDa cytokine receptor that plays an important role in the Th2-polarized immune responses characteristic of a variety of pathologies, including parasitic infections and allergic asthma (1, 2). Mature human IL-13 Rα2 consists of a 317 amino acid (aa) extracellular domain with three fibronectin type-III domains, a WSxWS motif, a 20 aa transmembrane segment, and a 17 aa cytoplasmic domain (3). Within the ECD, human IL-13 Rα2 shares 64% and 62% aa sequence identity with mouse and rat IL-13 Rα2, respectively. In both mouse and human, a 40 kDa-50 kDa soluble form of IL-13 Rα2 can be generated by MMP-8 mediated shedding *in vitro* (4). Although this is assumed to occur *in vivo* in mouse, there is no evidence that shedding occurs in human (5-7). In mouse, alternative splicing also leads to sIL-13 Rα2, but again, this phenomenon apparently does not occur in human (6-7). Thus, the biological effects of human IL-13 Rα2 would appear to be mediated exclusively by membrane IL-13 Rα2 (7). The biological effects of IL-13 and IL-4 are closely related in part due to a shared receptor system. IL-13 binds to IL-13 Rα1 which then forms a signaling complex with IL-4 Rα (8, 9). IL-13 Rα2 functions as a decoy receptor by binding and internalizing IL-13 and preventing it from signaling through the IL-13 Rα1/IL-4 Rα complex (3, 10). IL-13 Rα2 can also block IL-4 induced responses by inhibiting IL-4 bound IL-13 Rα1/IL-4 Rα receptor complexes even though it does not itself bind IL-4 (11, 12). Aside from its decoy function, IL-13-activated IL-13 Rα2 directly promotes the development of tissue fibrosis by inducing the transcription of TGF-β (13). Presumably, any human soluble IL-13 Rα2, if it exists, will retain its ligand binding capability and attenuate responses to IL-13 but not to IL-4 (11, 14). The up-regulation of transmembrane during Th2-biased immune responses limits the extent of those responses (15-17).

References:

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