Recombinant Human IL-13 Rα2 Fc Chimera

Catalog Number: 7147-IR

DESCRIPTION

Source

Chinese Hamster Ovary cell line, CHO-derived

Human IL-13 R alpha 2 (Met1-Leu342) Accession # NP_000631	IEGRMD	Human IgG_1 (Pro100-Lys330)

N-terminus C-terminus

N-terminal Sequence Analysis: Cys22

Structure / Form: Disulfide-linked homodimer

Predicted Molecular Mass: 64 kDa (monomer)

SPECIFICATIONS

SDS-PAGE: 75-95 kDa, reducing conditions

Activity: Measured by its ability to inhibit IL-13-dependent proliferation of TF-1 human erythroleukemic cells. Kitamura, T. *et al.* (1989) J. Cell Physiol. **140**:323.
The ED_{50} for this effect is 0.1-0.5 µg/mL in the presence of 8 ng/mL recombinant human IL-13.

Endotoxin Level: <0.10 EU per 1 µg 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 µm filtered solution in PBS. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution: Reconstitute at 100 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.

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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