

DESCRIPTION

Source *E. coli*-derived mouse IL-36Ra/IL-1F5 protein
Val2-Asp156
Accession # Q9QYY1

N-terminal Sequence Analysis Val2

Predicted Molecular Mass 17 kDa

SPECIFICATIONS

Activity Measured by its ability to inhibit IL-36 α , IL-36 β or IL-36 γ -induced IL-6 secretion by NIH-3T3 mouse embryonic fibroblast cells. The ED₅₀ for this effect is 0.8-4 μ g/mL in the presence of 15 ng/mL of Recombinant Mouse IL-36 β /IL-1F8 (aa 31-183) (Catalog # 7060-ML).

Measured by its binding ability in a functional ELISA. Immobilized Recombinant Mouse IL-36Ra/IL-1F5 at 1 μ g/mL (100 μ L/well) can bind Recombinant Human IL-1 Rrp2/IL-1 R6 Fc Chimera (Catalog # 872-RP) with a linear range of 0.15-5 μ g/mL.

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

Purity >97%, by SDS-PAGE under reducing conditions and visualized by silver stain.

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

PREPARATION AND STORAGE

Reconstitution Reconstitute at 250 μ g/mL in sterile 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

Mouse interleukin-36 receptor antagonist (IL-36Ra; previously IL-1F5; also named FIL-1 δ [delta], IL-1HY1, IL-1H3, and IL-1L1) is a member of the IL-1 family of proteins (1-6). IL-1 family members include IL-1 β , IL-1 α , IL-1ra, IL-18 and IL-1F5 through F10 (6, 7). All family members show a 12 β -strand, β -trefoil configuration, and are believed to have arisen from a common ancestral gene that underwent multiple duplications (8). The mouse IL-36Ra/IL-1F5 gene maps to a region on mouse chromosome 2 that contains all other IL-1 family members (except IL-18), supporting an evolutionary relationship with the IL-1 family (1, 9). It is particularly close to the gene for IL-1ra and is likely a relatively recent duplication of that gene. IL-36Ra/IL-1F5 is synthesized as a 156 amino acid (aa) protein that contains no signal sequence, no prosegment and no potential N-linked glycosylation site(s) (2, 5, 8). Nevertheless, it appears to be secreted as a 17 kDa monomer. In humans, there is an alternate start site that potentially gives rise to an alternate splice form (5). This translated product has a premature stop codon, resulting in a truncated 16 aa peptide. Mouse to human, full length IL-36Ra/IL-1F5 has 90% aa identity. Within the family, IL-36Ra/IL-1F5 is 48%, 30%, 35%, 35%, 35%, 37% and 43% aa identical to IL-1ra, IL-1 β , IL-36 α /IL-1F6, IL-37/IL-1F7, IL-36 β /IL-1F8, IL-36 γ /IL-1F9 and IL-1F10, respectively. Cells reported to express IL-36Ra/IL-1F5 include monocytes, B cells, dendritic cells/Langerhans cells, keratinocytes, and gastric fundus Parietal and Chief cells (1, 8). The receptor for IL-36Ra/IL-1F5 has not been positively identified. Indirect evidence suggests it is IL-1 Rrp2 and/or IL-1 RAcP (9). In either case, activity association with receptor binding is unclear. It was initially reported to be an antagonist of IL-36 γ activity (4, 7). This would be consistent with its hypothesized relationship to IL-1ra.

References:

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