

DESCRIPTION

Source *E. coli*-derived
Val2-Asp155
Accession # Q9UBH0

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-8 secretion in A431 human epithelial carcinoma cells. The ED₅₀ for this effect is 0.2-1 μ g/mL in the presence of 10 ng/mL of recombinant human IL-36 β .

Endotoxin Level <0.10 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 PBS with BSA as a carrier protein. See Certificate of Analysis for details.

PREPARATION AND STORAGE

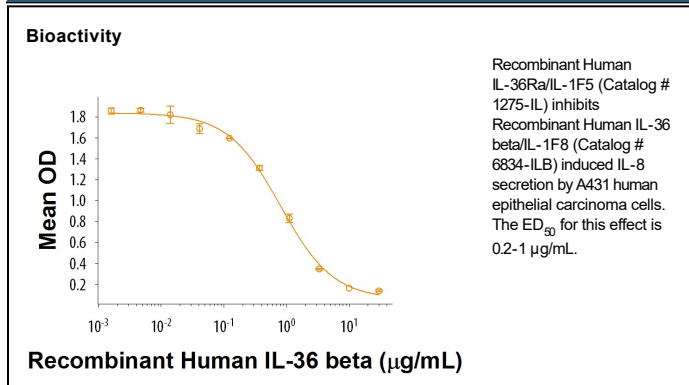
Reconstitution Reconstitute at 250 μ g/mL in sterile PBS.

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.

DATA



BACKGROUND

Human interleukin-36 receptor antagonist [IL-36Ra; previously IL-1F5 and also named FIL-1δ (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-1β, IL-1α, IL-1ra, IL-18 and IL-1F5-F10 (7). All family members show a 12 β-strand, β-trefoil configuration, and all family members are believed to have arisen from a common ancestral gene that underwent multiple duplications (7). The human IL-36Ra/IL-1F5 gene is in closest proximity to the gene for IL-1ra and is likely a relatively recent duplication of the IL-1ra gene (2, 3). IL-36Ra/IL-1F5 is synthesized as a 155 amino acid (aa) protein that contains no signal sequence, no prosegment and no potential N-linked glycosylation site(s) (2 - 5). Nevertheless, it appears to be secreted as a 17 kDa monomer (5). There is an alternate start site that potentially gives rise to an alternate splice form (5). The translated product, however, has a premature stop codon, resulting in a truncated 16 aa peptide. Human to mouse, full length IL-1F5 has 90% aa identity. Within the family, IL-36Ra/IL-1F5 is 50% aa identical to IL-1ra, and 32%, 31%, 35%, 37%, 32% and 42% aa identical to IL-1β, IL-36α/IL-1F6, IL-37/IL-1F7, IL-36β/IL-1F8, IL-36γ/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 also unclear. It was initially reported to be an antagonist of IL-36γ/IL-1F9 activity (4, 6). This would be consistent with its hypothesized relationship to IL-1ra. Studies, however, find IL-36Ra/IL-1F5 antagonist activity difficult to demonstrate (9).

References:

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