

DESCRIPTION

Source	<i>E. coli</i> -derived Gly13-Ser164 Accession # Q8R460
N-terminal Sequence Analysis	Gly13
Structure / Form	Monomer
Predicted Molecular Mass	17.3 kDa

SPECIFICATIONS

SDS-PAGE	17 kDa, reducing conditions
Activity	Measured by its ability to induce IL-6 secretion by NIH-3T3 mouse embryonic fibroblast cells. Towne, J.E. <i>et al.</i> (2004) <i>J. Biol. Chem.</i> 279 :13677. The ED ₅₀ for this effect is 3-18 ng/mL.
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 MES, NaCl, TCEP, EDTA, CHAPS and PEG 8000 with BSA as a carrier protein. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 100 µg/mL in PBS containing at least 0.1% human or bovine serum albumin.
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. <ul style="list-style-type: none"> ● 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

<p>Bioactivity</p> <p>Recombinant Mouse IL-36γ/IL-1F9 (aa 13-164) (Catalog # 6996-IL) induces IL-6 secretion in the NIH-3T3 mouse embryonic fibroblast cell line. The ED₅₀ for this effect is 3-18 ng/mL.</p>	<p>SDS-PAGE</p> <p>1 µg/lane of Recombinant Mouse IL-36γ/IL-1F9 (aa 13-164) was resolved with SDS-PAGE under reducing (R) conditions and visualized by silver staining, showing a single band at 17 kDa.</p>
---	---

BACKGROUND

IL-36 γ [previously called IL-1F9, IL-1 ϵ (epsilon), and IL-1H1] is a member of the IL-1 family which includes IL-1 β , IL-1 α , IL-1ra, IL-18, IL-36 Ra (IL-1F5), IL-36 α (IL-1F6), IL-36 β (IL-1F8), IL-37 (IL-1F7) and IL-1F10 (1-5). All family members show a 12 β -strand, β -trefoil configuration, and are believed to have arisen from a common ancestral gene (2, 3). Mouse IL-36 γ is an 18-22 kDa, 164 amino acid (aa) intracellular and secreted protein that contains no signal sequence, no prosegment and no potential N-linked glycosylation sites (1, 2, 4, 6, 7). Mouse IL-36 γ (aa 13-164) shares 58%, 84%, 64% and 60% aa sequence identity with human, rat, equine and bovine IL-36 γ , respectively, and 23-57% aa sequence identity with other family members. A 193 aa mouse isoform with a 29 aa N-terminal extension has been reported (8). Highest levels of IL-36 γ are produced by Langerhans cells, keratinocytes, and stomach Chief cells and parietal cells; these cells contribute to first-line defense against pathogens in the skin, lungs and digestive tract (2, 3, 6, 9). Its expression is induced by LPS treatment of monocytes, and by IL- α/β , IL-17 or TNF- α treatment of keratinocytes and bronchial epithelia (1, 6, 7, 9-11). Skin IL-36 γ expression is increased in contact hypersensitivity and psoriasis (1, 6, 11). It is elevated in inflammatory disorders of the lung (such as asthma) and viral infections. Lung IL-36 γ and other IL-36 proteins contribute to neutrophil influx (4, 7, 10). The receptor for IL-36 γ is a combination of IL-1 Rrp2, mainly found in epithelia and keratinocytes, and the widely expressed IL-1 RAcP (4, 7, 9). IL-36 α , β and γ all activate NF- κ B and MAPK pathways in an IL-1 Rrp2 dependent manner, and IL-36 γ induces production of inflammatory cytokines and chemokines such as CXCL8/IL-8 (7, 9, 10). Full-length recombinant IL-36 proteins appear less active than their endogenous counterparts, but trimming of the N-termini enhances their activity (12).

References:

1. Kumar, S. *et al.* (2000) *J. Biol. Chem.* **275**:10308.
2. Busfield, S.J. *et al.* (2000) *Genomics* **66**:213.
3. Dunn, E. *et al.* (2001) *Trends Immunol.* **22**:533.
4. Barksby, H.E. *et al.* (2007) *Clin. Exp. Immunol.* **149**:217.
5. Dinarello, C. *et al.* (2010) *Nat. Immunol.* **11**:973.
6. Debets, R. *et al.* (2001) *J. Immunol.* **167**:1440.
7. Chustz, R.T. *et al.* (2010) *Am. J. Respir. Cell Mol. Biol.* **45**:145.
8. NCBI Accession # NP_705731.2.
9. Towne, J.E. *et al.* (2004) *J. Biol. Chem.* **279**:13677.
10. Ramadas, R.A. *et al.* (2011) *Am. J. Respir. Cell Mol. Biol.* **44**:134.
11. Johnston, A. *et al.* (2011) *J. Immunol.* **186**:2613.
12. Blumberg, H. *et al.* (2010) *J. Immunol.* **185**:4354.