Recombinant Mouse IL-36α/IL-1F6 (aa 8-160)

Catalog Number: 7059-ML/CF

DESCRIPTION

Source	*E. coli*-derived Arg8-His160 Accession # Q9JLA2
N-terminal Sequence Analysis	Arg8
Predicted Molecular Mass	17 kDa

SPECIFICATIONS

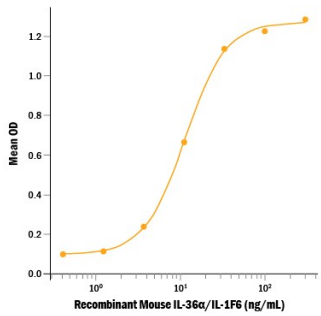
SDS-PAGE	16.5 kDa, reducing conditions
Activity	Measured by its ability to induce IL-6 secretion by NIH-3T3 mouse embryonic fibroblast cells. Towne, J.E. *et al.* (2004) J. Biol. Chem. **279**:13677. The ED_{50} for this effect is typically 3-18 ng/mL.
Endotoxin Level	<0.01 EU per 1 µg of the protein 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 MOPS, NaCl, TCEP and EDTA with Trehalose. 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.

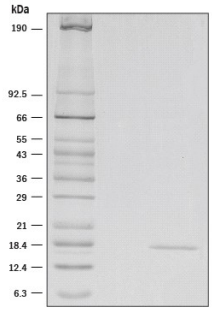
DATA

Bioactivity



Recombinant Mouse IL-36α/IL-1F6 (aa 8-160) (Catalog # 7059-ML/CF) induces IL-6 secretion in the NIH-3T3 mouse embryonic fibroblast cell line. The ED_{50} for this effect is 3-18 ng/mL.

SDS-PAGE



1 µg/lane of Recombinant Mouse IL-36α/IL-1F6 (aa 8-160) was resolved with SDS-PAGE under reducing (R) conditions and visualized by silver staining, showing a single band at 17 kDa.

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BACKGROUND

IL-36α, previously called IL-1F6 and FIL1ε (family of IL-1 member epsilon), is a member of the IL-1 family which includes IL-1β, IL-1α, IL-1ra, IL-18, and novel family members IL-36 Ra (IL-1F5), IL-36β (IL-1F8), IL-36γ (IL-1F9), IL-37 (IL-1F7) and IL-1F10 (14). All family members show a 12 β-strand, β-trefoil configuration, and are believed to have arisen from a common ancestral gene (1, 2). IL-36α is an 18-22 kDa, 160 amino acid (aa) intracellular and secreted protein that contains no signal sequence, no prosegment and no potential N-linked glycosylation sites (1-3). It can be externalized non-specifically in response to LPS and ATP-induced activation of the P2X7 receptor (5). Full-length recombinant IL-36α is less active than endogenous IL-36α, but trimming of the N-termini enhances its activity (6). Mouse IL-36α (aa 8-160) shares 83% aa sequence identity with rat IL-36α, 54-60% with human, rabbit, equine and bovine IL-36α, and 27-57% aa sequence identity with other novel IL-1 family members. IL-36α is mainly found in skin and lymphoid tissues, but also in fetal brain, trachea, stomach and intestine (1, 3, 7). It is expressed by monocytes, B and T cells (1, 2). The receptor for IL-36α is a combination of IL-1 Rrp2 (also called IL-1 RL2 or IL-1 R6), mainly found in epithelia and keratinocytes, and the widely expressed IL-1 RAcP (3, 6, 7). IL-36α, β and γ all activate NF-κB and MAPK pathways in an IL-1 Rrp2 dependent manner, and induce production of inflammatory cytokines and chemokines such as CXCL8/IL-8 (7). IL-36α and other family members are overexpressed in psoriatic skin lesions, and transgenic overexpression of IL-36α in skin keratinocytes produces epidermal hyperplasia (6-9). IL-36α is present in kidney tubule epithelia; it is highly overexpressed in tubulointerstitial lesions in mouse models of chronic glomerulonephritis, lupus nephritis and diabetic nephritis (10). IL-36α is induced by inflammation in adipose tissue-resident alternately activated (M2) macrophages, and reduces adipocyte differentiation (11).

References:

1. Smith, D.E. *et al.* (2000) J. Biol. Chem. **275**:1169.
2. Dunn, E. *et al.* (2001) Trends Immunol. **22**:533.
3. Barksby, H.E. *et al.* (2007) Clin. Exp. Immunol. **149**:217.
4. Dinarello, C. *et al.* (2010) Nat. Immunol. **11**:973.
5. Martin, U. *et al.* (2009) J. Immunol. **183**:4021.
6. Blumberg, H. *et al.* (2007) J. Exp. Med. **204**:2603.
7. Towne, J.E. *et al.* (2004) J. Biol. Chem. **279**:13677.
8. Blumberg, H. *et al.* (2010) J. Immunol. **185**:4354.
9. Johnston, A. *et al.* (2011) J. Immunol. **186**:2613.
10. Ichii, O. *et al.* (2010) Lab. Invest. **90**:459.
11. van Asseldonk, E.J.P. *et al.* (2010) Obesity **18**:2234.

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