

**DESCRIPTION**

<b>Source</b>	<i>E. coli</i> -derived Arg8-His160 Accession # Q9JLA2
<b>N-terminal Sequence Analysis</b>	Arg8
<b>Predicted Molecular Mass</b>	17 kDa

**SPECIFICATIONS**

<b>SDS-PAGE</b>	16.5 kDa, reducing conditions
<b>Activity</b>	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> <b>279</b> :13677. The ED <sub>50</sub> for this effect is typically 3-18 ng/mL.
<b>Endotoxin Level</b>	<0.01 EU per 1 $\mu$ g of the protein by the LAL method.
<b>Purity</b>	>95%, by SDS-PAGE visualized with Silver Staining and quantitative densitometry by Coomassie® Blue Staining.
<b>Formulation</b>	Lyophilized from a 0.2 $\mu$ m filtered solution in MOPS, NaCl, TCEP and EDTA with BSA as a carrier protein. See Certificate of Analysis for details.

**PREPARATION AND STORAGE**

<b>Reconstitution</b>	Reconstitute at 100 $\mu$ g/mL in PBS containing at least 0.1% human or bovine serum albumin.
<b>Shipping</b>	The product is shipped with polar packs. Upon receipt, store it immediately at the temperature recommended below.
<b>Stability &amp; Storage</b>	<b>Use a manual defrost freezer and avoid repeated freeze-thaw cycles.</b> <ul style="list-style-type: none"> <li>● 12 months from date of receipt, -20 to -70 °C as supplied.</li> <li>● 1 month, 2 to 8 °C under sterile conditions after reconstitution.</li> <li>● 3 months, -20 to -70 °C under sterile conditions after reconstitution.</li> </ul>

**DATA**

<p><b>Bioactivity</b></p> <p>Recombinant Mouse IL-36<math>\alpha</math>/IL-1F6 (aa 8-160) (Catalog # 7059-ML) induces IL-6 secretion in the NIH-3T3 mouse embryonic fibroblast cell line. The ED<sub>50</sub> for this effect is 3-18 ng/mL.</p>	<p><b>SDS-PAGE</b></p> <p>1 <math>\mu</math>g/lane of Recombinant Mouse IL-36<math>\alpha</math>/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.</p>
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**BACKGROUND**

IL-36 $\alpha$ , previously called IL-1F6 and FIL1 $\epsilon$  (family of IL-1 member epsilon), is a member of the IL-1 family which includes IL-1 $\beta$ , IL-1 $\alpha$ , IL-1ra, IL-18, and novel family members IL-36 Ra (IL-1F5), IL-36 $\beta$  (IL-1F8), IL-36 $\gamma$  (IL-1F9), IL-37 (IL-1F7) and IL-1F10 (14). All family members show a 12  $\beta$ -strand,  $\beta$ -trefoil configuration, and are believed to have arisen from a common ancestral gene (1, 2). IL-36 $\alpha$  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-36 $\alpha$  is less active than endogenous IL-36 $\alpha$ , but trimming of the N-termini enhances its activity (6). Mouse IL-36 $\alpha$  (aa 8-160) shares 83% aa sequence identity with rat IL-36 $\alpha$ , 54-60% with human, rabbit, equine and bovine IL-36 $\alpha$ , and 27-57% aa sequence identity with other novel IL-1 family members. IL-36 $\alpha$  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-36 $\alpha$  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-36 $\alpha$ ,  $\beta$  and  $\gamma$  all activate NF- $\kappa$ B 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-36 $\alpha$  and other family members are overexpressed in psoriatic skin lesions, and transgenic overexpression of IL-36 $\alpha$  in skin keratinocytes produces epidermal hyperplasia (6-9). IL-36 $\alpha$  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-36 $\alpha$  is induced by inflammation in adipose tissue-resident alternately activated (M2) macrophages, and reduces adipocyte differentiation (11).

**References:**

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