

DESCRIPTION

Source	<i>E. coli</i> -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. <i>et al.</i> (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 μ g 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 μ m filtered solution in MOPS, NaCl, TCEP and EDTA with BSA as a carrier protein. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 100 μ g/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-36α/IL-1F6 (aa 8-160) (Catalog # 7059-ML) 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-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.</p>
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BACKGROUND

IL-36 α , previously called IL-1F6 and FIL1 ϵ (family of IL-1 member epsilon), is a member of the IL-1 family which includes IL-1 β , IL-1 α , IL-1ra, IL-18, and novel family members IL-36 Ra (IL-1F5), IL-36 β (IL-1F8), IL-36 γ (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-36 α 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 α is less active than endogenous IL-36 α , but trimming of the N-termini enhances its activity (6). Mouse IL-36 α (aa 8-160) shares 83% aa sequence identity with rat IL-36 α , 54-60% with human, rabbit, equine and bovine IL-36 α , and 27-57% aa sequence identity with other novel IL-1 family members. IL-36 α 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 α 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 α , β and γ all activate NF- κ 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 α and other family members are overexpressed in psoriatic skin lesions, and transgenic overexpression of IL-36 α in skin keratinocytes produces epidermal hyperplasia (6-9). IL-36 α 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 α is induced by inflammation in adipose tissue-resident alternately activated (M2) macrophages, and reduces adipocyte differentiation (11).

References:

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