

**DESCRIPTION**

<b>Source</b>	<i>E. coli</i> -derived Ser31-Lys183 Accession # Q9D6Z6
<b>N-terminal Sequence Analysis</b>	Ser31
<b>Predicted Molecular Mass</b>	17.4 kDa

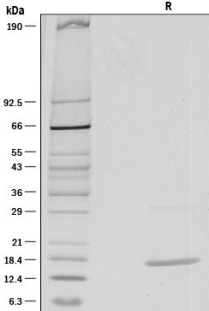
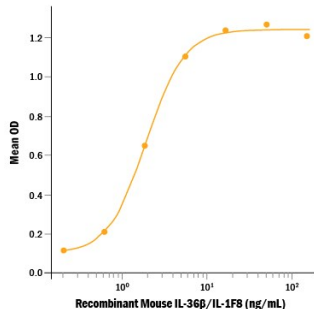
**SPECIFICATIONS**

<b>SDS-PAGE</b>	16 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) J. Biol. Chem. 279:13677. The ED <sub>50</sub> for this effect is typically 1-6 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 under reducing conditions and visualized by silver stain.
<b>Formulation</b>	Lyophilized from a 0.2 $\mu$ m filtered solution in MES, NaCl, TCEP, EDTA, CHAPS and PEG 8000 with Trehalose. See Certificate of Analysis for details.

**PREPARATION AND STORAGE**

<b>Reconstitution</b>	Reconstitute at 100 $\mu$ g/mL in 10 mM Tris-HCl, pH 8.0.
<b>Shipping</b>	The product is shipped at ambient temperature. 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>SDS-PAGE</b></p>  <p>1 <math>\mu</math>g/lane of Recombinant Mouse IL-36<math>\beta</math>/IL-1F8 (aa 31-183) was resolved with SDS-PAGE under reducing (R) conditions and visualized by silver staining, showing a single band at 16 kDa.</p>	<p><b>Bioactivity</b></p>  <p>Recombinant Mouse IL-36<math>\beta</math>/IL-1F8 (aa 31-183) (Catalog # 7060-ML/CF) induces IL-6 secretion in the NIH-3T3 mouse embryonic fibroblast cell line. The ED<sub>50</sub> for this effect is typically 1-6 ng/mL.</p>
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**BACKGROUND**

Mouse interleukin-36 beta [IL-36 $\beta$ ; previously IL-1F8, FIL-1 $\eta$  (eta) and IL-1H2] is a member of the IL-1 family of proteins that includes IL-1 $\beta$ , IL-1 $\alpha$ , IL-1ra, IL-18, IL-36Ra/IL-1F5, IL-36 $\alpha$ /IL-1F6, IL-37/IL-1F7, IL-36 $\gamma$ /IL-1F9 and IL-1F10 (1 - 6). All family members show a 12  $\beta$ -stranded  $\beta$ -trefoil configuration, share up to 50% amino acid (aa) sequence identity, and are believed to have arisen from a common ancestral gene (3, 4). Although two alternatively spliced transcript variants for human IL-36 $\beta$ /IL-1F8 have been described, to date, only one mouse IL-36 $\beta$ /IL-1F8 isoform is known (3). Mouse IL-36 $\beta$ /IL-1F8 is synthesized as a 183 amino acid (aa) protein that contains no signal sequence, no prosegment and no potential N-linked glycosylation site(s) (1, 2). Mouse IL-36 $\beta$ /IL-1F8 shares 61% and 74% aa identity with human IL-36 $\beta$  isoform 2 and rat IL-36 $\beta$ , respectively. IL-36 $\beta$  is agonistic, stimulating release of inflammatory mediators such as IL-6 and IL-8, and cytotoxic peptides such as beta-defensins 2 and 3 that aid in defense against microbial pathogens (7-10). The receptor for IL-36 proteins is IL-1 Rrp2, with IL-1 RAcP as a coreceptor (7, 9). Antagonism of IL-36 proteins by IL-36Ra, which also binds IL-1 Rrp2, has been shown by some investigators (5, 6). Skin keratinocytes express highest levels of IL-36 proteins and their receptors, followed by epithelia in the esophagus, trachea and bronchae (7 - 9). IL-36 $\beta$  expression is increased in psoriatic skin and may play a role in pathogenesis of psoriasis (7, 8). IL-36 $\beta$  is also expressed in resting and activated monocytes and B cells, synovial fibroblasts, neurons and glia, and is detectable in plasma and body fluids (1, 7, 9, 11). IL-36 $\beta$ , along with IL-36 $\alpha$  and IL-36 $\gamma$ , is up-regulated by IL-1 $\alpha$  and TNF- $\alpha$  in keratinocytes, and has been shown to activate NF- $\kappa$ B and MAPK signaling pathways in an IL-1 Rrp2-dependent manner (7-9). Full-length recombinant IL-36 proteins appear less active than their endogenous counterparts, but trimming of the N-termini enhances their activity (9, 12).

**References:**

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