

DESCRIPTION

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

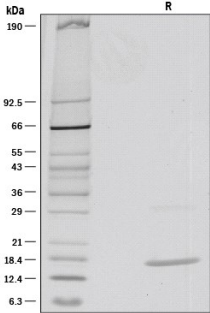
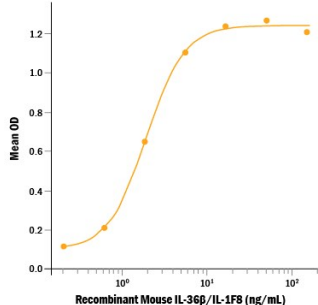
SPECIFICATIONS

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| SDS-PAGE | 16 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 1-6 ng/mL. |
| Endotoxin Level | <0.01 EU per 1 μ g 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 μ m 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

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| Reconstitution | Reconstitute at 100 μ g/mL in 10 mM Tris-HCl, pH 8.0. |
| 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

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| <p>SDS-PAGE</p>  <p>1 μg/lane of Recombinant Mouse IL-36β/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>Bioactivity</p>  <p>Recombinant Mouse IL-36β/IL-1F8 (aa 31-183) (Catalog # 7060-ML) induces IL-6 secretion in the NIH-3T3 mouse embryonic fibroblast cell line. The ED₅₀ for this effect is typically 1-6 ng/mL.</p> |
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BACKGROUND

Mouse interleukin-36 beta [IL-36 β ; previously IL-1F8, FIL-1 η (eta) and IL-1H2] is a member of the IL-1 family of proteins that includes IL-1 β , IL-1 α , IL-1ra, IL-18, IL-36Ra/IL-1F5, IL-36 α /IL-1F6, IL-37/IL-1F7, IL-36 γ /IL-1F9 and IL-1F10 (1 - 6). All family members show a 12 β -stranded β -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 β /IL-1F8 have been described, to date, only one mouse IL-36 β /IL-1F8 isoform is known (3). Mouse IL-36 β /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 β /IL-1F8 shares 61% and 74% aa identity with human IL-36 β isoform 2 and rat IL-36 β , respectively. IL-36 β 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 β expression is increased in psoriatic skin and may play a role in pathogenesis of psoriasis (7, 8). IL-36 β 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 β , along with IL-36 α and IL-36 γ , is up-regulated by IL-1 α and TNF- α in keratinocytes, and has been shown to activate NF- κ 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:

1. Smith, D.E. *et al.* (2000) *J. Biol. Chem.* **275**:1169.
2. Kumar, S. *et al.* (2000) *J. Biol. Chem.* **275**:10308.
3. Nicklin, M.J.H. *et al.* (2002) *Genomics* **79**:718.
4. Dunn, E. *et al.* (2001) *Trends Immunol.* **22**:533.
5. Dinarello, C. *et al.* (2010) *Nat. Immunol.* **11**:973.
6. Barksby, H.E. *et al.* (2007) *Clin. Exp. Immunol.* **149**:217.
7. Towne, J.E. *et al.* (2004) *J. Biol. Chem.* **279**:13677.
8. Johnston, A. *et al.* (2011) *J. Immunol.* **186**:2613.
9. Magne, D. *et al.* (2005) *Arthritis Res. Ther.* **8**:R80.
10. van Asseldonk, E.J.P. *et al.* (2010) *Obesity* **18**:2234.
11. Wang, P. *et al.* (2005) *Cytokine* **29**:245.
12. Blumberg, H. *et al.* (2010) *J. Immunol.* **185**:4354.