

DESCRIPTION

Source *E. coli*-derived mouse IL-33 protein
Ser109-Ile266
Accession # Q8BVZ5

N-terminal Sequence Analysis Ser109

Predicted Molecular Mass 18 kDa

SPECIFICATIONS

SDS-PAGE 20 kDa, reducing conditions

Activity Measured in a cell proliferation assay using D10.G4.1 mouse helper T cells.
The ED₅₀ for this effect is 0.0125-0.05 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 PBS, EDTA and DTT with BSA as a carrier protein. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution Reconstitute at 100 µg/mL in sterile 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 °C under sterile conditions after reconstitution.

BACKGROUND

IL-33, also known as NF-HEV and DVS 27, is a 30 kDa proinflammatory protein that may also regulate gene transcription (1-3). DVS 27 was identified as a gene that is up-regulated in vasospastic cerebral arteries (1). NF-HEV was described as a nuclear factor that is preferentially expressed in the endothelial cells of high endothelial venules relative to endothelial cells from other tissues (2). IL-33 was identified based on sequence and structural homology with IL-1 family cytokines (3). DVS 27, NF-HEV, and IL-33 share 100% amino acid sequence identity. IL-33 is constitutively expressed in smooth muscle and airway epithelia. It is up-regulated in arterial smooth muscle, dermal fibroblasts, and keratinocytes following IL-1α or IL-1β stimulation (1, 3). Similar to IL-1, IL-33 can be cleaved *in vitro* by caspase-1, generating an N-terminal fragment that is slightly shorter than the C-terminal fragment (3, 4). The N-terminal portion of full length IL-33 contains a predicted bipartite nuclear localization sequence and a homeodomain-like helix-turn-helix DNA binding domain. By immunofluorescence, full length IL-33 localizes to the nucleus in HUVECs and transfectants (2). The C-terminal fragment, corresponding to mature IL-33, binds and triggers signaling through mast cell IL-1 R4/ST2L, a longtime orphan receptor involved in the augmentation of Th2 cell responses (3, 5-7). A ternary signaling complex is formed by the subsequent association of IL-33 and ST2L with IL-1 RAcP (8). Stimulation of Th2 polarized lymphocytes with mature IL-33 *in vitro* induces IL-5 and IL-13 secretion (3). *In vivo* administration of mature IL-33 promotes increased production of IL-5, IL-13, IgE, and IgA, as well as splenomegaly and inflammatory infiltration of mucosal tissues (3). Full length and mature mouse IL-33 share approximately 55% and 90% aa sequence identity with human and rat IL-33, respectively. Mouse IL-33 shares less than 25% aa sequence identity with other IL-1 family proteins.

References:

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2. Baekkevold, E.S. *et al.* (2003) *Am. J. Pathol.* **163**:69.
3. Schmitz, J. *et al.* (2005) *Immunity* **23**:479.
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5. Xu, D. *et al.* (1998) *J. Exp. Med.* **187**:787.
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7. Dinarello, C.A. (2005) *Immunity* **23**:461.
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