

DESCRIPTION

Source *E. coli*-derived
Cys2-Lys131
Accession # AAS80146

N-terminal Sequence Analysis Cys2

Predicted Molecular Mass 14.9 kDa

SPECIFICATIONS

SDS-PAGE 18.8 kDa, reducing conditions

Activity Measured by its ability to induce TNF-α secretion by RAW 264.7 mouse monocyte/macrophage cells under serum free conditions in the presence of muramyl dipeptide (MDP) and Polymyxin B. Netea, M.G. *et al.* (2005) *Proc. Nat. Acad. Sci.* **102**:16309. The ED₅₀ for this effect is typically 2–12 µg/mL.

Endotoxin Level <0.10 EU per 1 µg of the protein by the LAL method.

Purity >85%, by SDS-PAGE under reducing conditions and visualized by silver stain.

Formulation Lyophilized from a 0.2 µm filtered solution in PBS and DTT. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution Reconstitute at 200 µg/mL in PBS.

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.**

- 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.

BACKGROUND

Interleukin 32 (IL-32) is an N-glycosylated cytokine that is up-regulated by inflammatory stimulation in monocytes, NK cells, epithelial cells, and pancreatic myofibroblasts (1 - 5). It cooperates with these stimuli to promote the expression of other proinflammatory molecules such as TNF-α, IL-6, IL-1β, IL-1α, and CXCL8/IL-8 (5 - 7). The longest of several IL-32 splicing variants is the 20 - 25 kDa γ isoform which is also known as natural killer cell transcript 4 (NK4) (8, 9). The α isoform (IL-32α) lacks a portion of the putative signal peptide as well as 57 aa from the C-terminal region. IL-32α is less potent than IL-32β, γ, or δ at inducing the expression of proinflammatory molecules in peripheral blood mononuclear cells (PBMC) (8, 10). Neutrophil-derived Proteinase 3 (PR3) cleaves IL-32α between Thr57 and Val58, a cleavage site that is retained in other IL-32 isoforms (11). The N-terminal fragment of PR3-cleaved IL-32α shows increased potency at inducing CXCL2/MIP-2 and CXCL8 expression in PBMC relative to uncleaved IL-32α (11, 12). IL-32 is highly expressed by colonic epithelial cells in inflammatory bowel disease and Crohn's disease, rheumatoid arthritis synovium, and ductal epithelial cells in chronic pancreatitis and pancreatic cancer (5, 13 - 15). IL-32 inhibits HIV-1 replication *in vitro*, and it is elevated in the serum of HIV-1 patients (16, 17).

References:

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