

## DESCRIPTION

**Source** *E. coli*-derived  
Gly23-Lys67  
Accession # P81534

**N-terminal Sequence Analysis** Gly23

**Predicted Molecular Mass** 5.2 kDa

## SPECIFICATIONS

**Activity** Measured by its anti-microbial activity against *E. coli*. Ganz, T. *et al.* (2003) Nat. Rev. Immunol. **3**:710.  
The ED<sub>50</sub> for this effect is typically 7.5-30  $\mu$ g/mL.

Measured by its ability to promote internalization of cell surface CXCR4 on Jurkat human acute T cell leukemia cells. Quinones-Mateu, M.E. *et al.* (2003) AIDS **17**:F39.

As determined by flow cytometric analysis, the effect is typically a 2-3 fold reduction in fluorescence intensity of Jurkat cells stained with a CXCR4 antibody (Catalog # [FAB170P](#)) following a 2 hour pre-treatment with 10  $\mu$ g/mL human  $\beta$ -Defensin 3 compared to untreated cells.

**Optimal dilutions should be determined by each laboratory for each application.**

**Endotoxin Level** <0.01 EU per 1  $\mu$ 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  $\mu$ m filtered solution in HCl. See Certificate of Analysis for details.

## PREPARATION AND STORAGE

**Reconstitution** Reconstitute at 500  $\mu$ g/mL in sterile 4 mM HCl.

**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 to -70 °C under sterile conditions after reconstitution.

## BACKGROUND

$\beta$ -Defensin 3, also known as BD3 and DEFB-3, is a membrane-active cationic peptide that functions in inflammation and innate immune responses. There are at least 30 beta-Defensins which are distinguished from alpha-Defensins by the connectivity pattern of their three intramolecular disulfide bonds (1). The 45 amino acid (aa) mature human BD3 shares 38% and 33% aa sequence identity with mouse and rat BD3, respectively (2, 3). It shares 18%-36% aa sequence identity with other human beta-Defensins. BD3 is widely expressed among epithelial tissues, notably by keratinocytes and airway epithelial cells. It is upregulated in response to proinflammatory cytokines, microbial and viral infections, and at the edges of skin wounds (2, 4-6). BD3 induction in osteoarthritis chondrocytes promotes MMP1 and 13 production and inhibits TIMP1 and 2 expression (7). *In vivo* control of BD3 activity is accomplished in part through cleavage by cathepsins B, L, and S (8). BD3 displays strain specific microbicidal activity toward a broad spectrum of bacteria and yeast (2, 9). BD3 also induces monocyte migration, mast cell activation, and a mast cell-dependent increase in vascular permeability (4, 10). Disruption of the intramolecular disulfide bond pattern in BD3 abrogates its monocyte chemoattractant properties but not its antimicrobial properties (11, 12). BD3 inhibits viral infectivity by interacting directly with HIV-1 plus its coreceptor CXCR4 (5, 13), and with HSV glycoprotein B plus its receptor heparan sulfate (14), and by forming a protective coating on the surface of influenza virus target cells (15).

### References:

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