Recombinant Mouse β-Defensin 3

Catalog Number: 5987-BD

DESCRIPTION

Source	*E. coli*-derived Lys23-Lys63 Accession # Q9WTL0
N-terminal Sequence Analysis	Lys23
Predicted Molecular Mass	4.6 kDa

SPECIFICATIONS

SDS-PAGE	5 kDa, reducing conditions
Activity	Measured by its anti-microbial activity against *E. coli*. Ganz, T. *et al.* (2003) Nat. Rev. Immunol. **3**:710. The ED_{50} for this effect is typically 4-20 µg/mL.
Endotoxin Level	<0.01 EU per 1 µg 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 µm filtered solution in HCl. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 500 µg/mL in 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

β-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 β-defensins which are distinguished from α-defensins by the connectivity pattern of their three intramolecular disulfide bonds (1). The 41 aa mature mouse BD3 shares 38% and 59% aa sequence identity with human and rat BD3, respectively (2, 3). It shares 14% - 46% aa sequence identity with other mouse β-defensins. BD3 is widely expressed among epithelial tissues, notably by keratinocytes and airway epithelial cells. It is up-regulated 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 and its coreceptor CXCR4 (5, 13) and with HSV glycoprotein B and its receptor heparan sulfate (14), and by forming a protective coating on the surface of influenza virus target cells (15).

References:

1. Dhople, V. *et al.* (2006) Biochim. Biophys. Acta. **1758**:1499.
2. Bals, R. *et al.* (1999) Infect. Immun. **67**:3542.
3. Schibli, D.J. *et al.* (2002) J. Biol. Chem. **277**:8279.
4. Garcia, J.-R.C. *et al.* (2001) Cell Tissue Res. **306**:257.
5. Quinones-Mateu, M.E. *et al.* (2003) AIDS **17**:F39.
6. Sorensen, O.E. *et al.* (2006) J. Clin. Invest. **116**:1878.
7. Varoga, D. *et al.* (2005) Arthritis Rheum. **52**:1736.
8. Taggart, C.C. *et al.* (2003) J. Immunol. **171**:931.
9. Joly, S. *et al.* (2004) J. Clin. Microbiol. **42**:1024.
10. Chen, X. *et al.* (2007) Eur. J. Immunol. **37**:434.
11. Kluver, E. *et al.* (2005) Biochemistry **44**:9804.
12. Wu, Z. *et al.* (2003) Proc. Natl. Acad. Sci. **100**:8880.
13. Feng, Z. *et al.* (2006) J. Immunol. **177**:782.
14. Hazrati, E. *et al.* (2006) J. Immunol. **177**:8658.
15. Leikina, E. *et al.* (2005) Nat. Immunol. **6**:995.